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UNDERWATER BLAST INJURY - A REVIEW OF THE LITERATURE

by

Nelson M. Wolf, LT MC USNR

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SUMMARY PAGE

THE PROBLEM

To review the literature on underwater blast injury, the mechanism of this type of injury, the pathology, and to present a discussion of the clinical considerations involved.

FINDINGS

A study was completed which correlated all of the aspects of blast injury, the physics of blast, the mechanism of injury, and the clinical aspects of the potential situation.

APPLICATIONS

The information presented in this study is of interest to submarine medical officers and all medical personnel involved with Navy diving operations, or any salvage or military operations where explosives may be employed, or any situation where hostile forces may employ explosives to thwart operations of underwater swimmers.

ADMINISTRATIVE INFORMATION

The material contained in this report was prepared by the author for partial fulfillment of the requirements for qualification as a Submarine Medical Officer. It was subsequently chosen for publication as a Submarine Medical Research Laboratory Report No. 646, in order to make the material available to students in the School of Submarine Medicine, in the Technical Library at NSMRL, and to submarine medical officers in the field.

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ABSTRACT

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Underwater blast injury is reviewed for the period 1916 to the present date (1970). The physics of the blast, the mechanism of injury, the pathology, and clinical considerations are discussed. A discussion and criticism is presented of the various formulae for damage range. Much of the material is supported with references to both animal and human data.

ABSTRACT

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UNDERWATER BLAST INJURY - A REVIEW OF THE LITERATURE

INTRODUCTION

Underwater blast injury due to mines or depth charges was first described, in the medical literature in 1916, by medical officers of the Royal Navy. The entity had the symptoms of hemoptysis, bloody diarrhea, and hematuria.⁵

During the period after World War I, little work was done to further elucidate the nature of this problem; and, it was not until the second World War that an impetus was given for further study. During that war, it was common for much of a ship's complement to be in the water (due to air, surface, or submarine attack) at the time of underwater explosions.⁵ In 1945, Wakely²⁶ reported that the mortality, from such injury, might approach 80 per cent.

Since World War II, the blast problem has changed from one affecting many individuals, to one that has the potential of affecting many. Its interest to medical officers, involved with diving, is obvious. Divers use explosives in salvage and military operations. In addition, hostile forces have used explosives, in combat, against underwater swimmers.

Although various papers, during and since the Second World War, have discussed this entity, no paper has really correlated all of the aspects - physics, mechanism of injury, and clinical points. The purpose of this paper will be to give an overall discussion of the so-called underwater blast injury.

PHYSICS OF THE EXPLOSIVE WAVE

In order to understand the mechanism of injury, the nature of the underwater explosion must be explained.

The detonation of an explosive leads to the formation of a gas bubble. This bubble is spherical in shape and may have an initial temperature of 3000°C and a pressure equivalent to 50,000 atmospheres.¹² This peak pressure is reached within a very short period of time, on the order of 10^{-5} seconds after detonation. The bubble tends to expand, compressing the water in its immediate vicinity. This sets up the pressure pulse. The bubble continues to expand beyond the point of equilibrium due to the inertia of the water. As a result, the bubble now collapses and with this collapse (again beyond equilibrium) another or subsidiary pulse is generated. 1 With the re-expansion of the bubble, a third pulse is generated - the bubble pulse. The process of expansion and collapse continues until energy is dissipated; the result is an oscillating system. 1 (see Fig. 1).

The pressure waves move through the water at varying velocities. Near the point of the detonation, the velocity is vary high and is related to the pressure. ²⁶ At some point about 20 charge diameters distant from the detonation, the velocity of the pressure waves reaches that of sound in water -5000 ft/sec. From this point on, the pressure waves follow the laws of sound in water. ²⁶ The energy of the waves



Fig. 1. Diagram explaining the pressure waves produced by an underwater detonation, dr = direct wave; sr = surface reflected wave; br = bottom reflected wave; ip = initial pulse; sp = subsidiary pulse; bp = bubble pulse; Pinax = peak pressure; t = time interval; Pt = pressure at the time interval; t. (from Anderson¹)

varies inversely with the square of the distance and they are reflected and absorbed in a similar fashion as sound waves.

The pressure and subsequent pulses, passing through the water rapidly reaches a p-max or maximum pressure. The pressure then falls as an exponential function with a time constant T, i.e. $-pT = \frac{1}{e}p-max$.¹

The energy of the system can be divided as follows: 25% - initial pressure wave, 25% - bubble and subsidiary waves, and 50% - heat, turbulence, etc. ²¹ It can be seen that, in an explosion, most of the energy available for damage is in the initial pressure pulse.

In addition to the pulses coming from the explosion directly, there are various other waves that may occur. If the pulse contacts the bottom, it may be reflected or absorbed. The proportion of each will depend on the nature of the bottom: a hard bottom has little absorption and much reflection. Again, as in the case of a sound wave, the angle of incidence will be equal to the angle of reflection. The reflected wave, coming from the bottom will tend to add to the initial wave. 1 (see Fig. 1). If the bottom is very distant from the point of detonation this effect will be negligible.

At the surface, a number of things occur that tend to modify the pulse. If the pressure of the pulse exceeds 500 lb/in^2 at the surface ¹⁶, the water will shred, producing the dome. This is a small part of the pulse, as a rule; the remainder is reflected with an angle of reflection equal to the angle of incidence. ²¹ This pulse, coming from the surface, tends to subtract from the initial pulse. (see Fig. 1).

Disturbances can be seen at the surface other than the dome phenomenon; these follow the dome in time. The second occurrence is the 'slick'; this is a rapidly expanding ring of darkened water due to the pressure wave advance. ¹² The plume is the last manifestation of the explosion seen at the surface, and results from the gaseous products of the explosion reaching and breaking the surface of the water. This last disturbance may be quite spectacular but does little damage.

The magnitude of the surface phenomena varies with the size of the charge and the depth of detonation. There is a depth beyond which a given charge will produce no aberrations at the surface. The 'slick' tends to be retained to a greater depth, being only dependent on the presence of the pulse wave and not on its ability to shred the water. 12

Thermal layers also tend to reflect the explosive waves. A nearby bank, shore, or large ship may also modify the pressure wave.

The size of the charge, depth of detonation, and distance from the target all have an influence on the available damaging capability of the pressure wave at a given point.

In summary, it can be seen that the pressure wave is basically a pulse moving through the water at the velocity of sound, dissipating its energy directly with the square of the distance traveled, and is influenced by many factors. Essentially, the major changes in the pressure wave occur when the wave comes in contact with the interface between two media of different densities - i. e. water-air, water-bottom.

MECHANISM OF INJURY

An explosion in the water differs from that in air, and this results in a difference in the mechanism of injury. The strength of the wave in water is maintained to a greater distance than in air.²¹ For example, a peak pressure 10 ft. from a 1 lb. air burst is 7 psi; in water, this becomes 1600 psi.⁷ The shape of the shock envelope in air is influenced greatly by the geometry of the charge container, i.e. - a cylindrical container gives a different shock wave configuration from that of a spherical container.²⁷ Due to the low compressibility of the water, the blast is quickly forced into a spherical shape.

Much of the injury in an air explosion is secondary to the fragmentation of the charge container and bits of gravel sucked into the explosive wave. ²¹ In a water blast, these particles are retarded by the increased viscosity of the medium.

Finally, much of the air pressure wave is reflected at the body surface, since this represents an interface between media of different densities. 1

For practical purposes, blast injury in the water depends on the pressure wave; in air, shrapnel may also play a large role.

The pressure wave has been discussed in the above section. It can be described as having a p-max at any given point through which it advances. It also has duration, energy, and an impulse. The impulse is the time integral of the pressure wave -I = pdt, where I = impulse, p = pressure, and dt = change in time. ¹ Which of these functions causes the biological damage has been the subject of much debate. It is first necessary to describe how the injury occurs, before entering the discussion.

As a wave passes through a medium of equal density the individual molecules are displaced very little, except at areas of compression and rarefaction. In the previous section, the wave only displaced the water molecules extensively at the surface (water-air interface). Since most of the body tissues are of water density, damage would also be expected to occur at similar air-water interfaces. These are found in the lung, air filled viscera in the abdomen, sinus cavities and the middle ear. With the exception of the sinus areas, which are given some protection by the rigid bony frame, these areas are most often affected. The actual mechanism may be described as similar to the shredding in the dome phenomenon. This occurs, states one author, ¹⁶ if the pulse delivers to the tissues a pressure greater than 500 lb/in.²

Using the thoracic cavity as an example, Benzinger ³ showed that the damage is not due to pressure changes transmitted via the upper airways, but is the result of the transmission of the wave directly through the thoracic wall. Body tissues in the thorax are of different densities; this results in a large pressure gradient as the wave passes through this area. The tissues are accelerated at different rates; literally, they are torn apart.

The injury mechanism, therefore, is due to the pressure wave which accelerates various tissues at different rates and thereby causes shredding. The exact function of the wave which does this has not been uniformly implicated. The importance of this becomes obvious in that the range of lethality, injury, etc., will vary with the function that is causing the injury. These ranges, in turn, are important if it becomes necessary to keep divers in the water during an underwater blast, or if the blasts are being used to deter swimmers from some target.

In a paper Ly Clemedson and Criborn, 10 the effect of various parts of the body to various functions of the pressure wave were studied. It was found that the damaging function - duration, impulse, or peak pressure - varied with the tissues involved. The maximum pressure is the most important factor if the natural period of the tissue is short, compared to the shock wave's duration. If the natural period is long, compared to the duration of the shock wave, then the injury that is done varies with the impulse. Whether a part of the body has a long or short period will depend on the density, elasticity, and physical properties of che part's component tissues. Each area of the body has a mixture of long and short period tissues; however, the leg and ribs are primarily short period areas, the abdomen and the costal interspaces are long, and the thoracic wall is intermediate.

A tissue must be chosen as the most sensitive to blast injury, in order to derive a range equation. During the Second World War and immediately after, it was felt that air explosions affected the lungs and water explosions affected the abdominal hol'ow viscera. It has been shown by many workers that the effect on the lungs is similar, whether the blast took place in air or water. 3, 8, 11, 16, 24 They differ only in that the pressure will be maintained to a greater distance in water than in air. Much of the original confusion as to difference in injury may be due to the selection of patients

during the war. Those with severe respiratory injury may have died prior to medical care. 10

There are a few range formulae that are being used today; none take in all of the factors. The U. S. Navy Diving Manual (Mar 1970)²⁵ uses the peak pressure as the damaging variable. It assumes that a pressure greater than 500 lb/in^2 will cause some injury and that one greater than 2000 lb/in² will

cause death.
$$(p = \frac{13,000\sqrt[3]{w}}{d})$$

[p = pressure (psi), w = wgt of chg (lb), d = dist (ft)]

Hartman ¹⁸ used peak pressure to determine an LD_{50} range (R_L) and arrived at the following: $R_L = 44w^{1/3}$, R_L = ft and w = lb.

Another author assumes that the energy of the wave is the crucial variable.² His formula is $R_L = 7w^{1/2}$.

None of the examples cited take into account any variable, save the size of the charge. For example, they all ignore the secondary waves coming from the surface and the bottom, that are known to greatly modify the pressure wave. In addition, some of the above examples use the peak pressure as the prime determinant, when the impulse (if any single determinant) function should be used. It can be recalled that the lung is probably the crucial organ.

Christian⁷ attempts to predict the lethality curves for explosions of a given magnitude, depth, and type of wave. She assumes that the thorax is the target organ and that the impulse is the function of importance. Basically, she states that an explosion will probably inflict the stipulated mortality level if it delivers an impulse equal to I_{O} in a time no greater than t_0 . It is assumed that the duration of the pressure wave is much less than the natural period of the thorax (about 2 milliseconds). This relation is assured by the small charges, therefore a short duration, and the relatively shallow depth of detonation. The shallowness leads to a negative surface reflected wave that further limits the duration of the pressure wave.

Although Christian's curves work well for the situational model that she puts forth, it is of very limited usefulness due to the strict criteria necessary to derive her formulae.

Assuming that the impulse is the true function causing biological damage, another interesting point about underwater blast injury becomes apparent. It is known that an explosion is much more likely to produce injury as the diver's depth is increased; the probable explanation is that the impulse increases with depth (the peak pressure does not). There is a depth where the inverse square distance law tends to over-ride this and beyond this the impulse decreases.⁷

It has been stated above that the most likely organs to sustain an injury from an underwater blast are those that contain gas. This is only true when these organs are immersed in the water. If the lower extremity, only, is immersed, a pressure gradient will be set up from the areas below to those

above the water's surface. The impulse and the peak pressure will be greatest at the deeper portions and least at those areas closest to the surface. This is due to the negative surface reflected wave's reaching the deeper areas after a longer time and therefore having less of a dampening effect on the wave in this area. In addition to the impulse gradient, a pressure gradient is set up, since very little of the wave's energy crosses the water-air interface. As a result of the above, the soft tissues are pushed to the surface and shredded.²⁶ (see Fig. 2).



Fig 2

Fig. 2. Action of underwater explosion on dependent leg on the surface. (from Wakely ²⁶),

CLINICAL ASPECTS

Following the discussion of the physics of underwater blast and potential tissue damage, it is now appropriate to discuss, in detail, the symptoms, signs and management of actual damage produced by the explosion. The animal data will be described first and then the human data will be presented.

The methodology of most animal studies involved placing the animal in a partially submerged position, with either the head, or the head and the chest out of the water. They were placed at various distances from the explosion. The charge was then detonated and the clinical symptoms were noted. After a short time, all of the animals were sacrificed and a postmortem examination was performed. During the explosion, an attempt was made to monitor the pressure and the impulse.^{5, 6, 15, 16}

Most of the animals showed some paralysis immediately after the injury, and there was no sign of external violence.⁵ One author, ¹⁵ using small animals, reported extremity twitching and opisthotonos in the preterminal cases.

Greaves¹⁶ divided his experimental results into three groups: a) lethal, b) sublethal, and c) minimal. The lethal group was not immediately rendered unconscious but struggled in the water. Cyanosis was apparent and a bloody liquid was seen coming from the nose and the mouth. Death followed in about one minute. The sublethal group continued to swim but remained quiet and appeared ill upon removal from the water. A bloody froth was occasionally seen about the mouth. Some of this group also died. Those that remained regained normal activity and showed no ill effects. The minimally injured animals remained quiet for a short time and then demonstrated no ill effects.

The pathological examination revealed injury to the lung, abdominal gas-filled viscera, central nervous system, and solid viscera. It must be emphasized that the respiratory and the abdominal organs were the hardest hit.

According to Cameron⁵ the respiratory findings included: a) pulmonary hemorrhage, usually in the basilar areas and occasionally resembling lobar congestion or an embolic event, b) bronchial and tracheal hemorrhage, c) acute vesicular and interstitial emphysema, and d) pneumothorax and hemothorax. Although intestinal injury was described, this was not as constant as the respiratory injury. It consisted of subserous and submucosal hemorrhage and perforation of the gasfilled viscera. The urinary and gall bladder were spared. It was also noted that there were great species differences.

In a somewhat later report,⁶ it was shown that the lesions varied with the extent of the submergence. If the chest and the abdomen were both submerged, then the lung lesions were more severe, with a greater percentage of the animals having blood in the large airways. If the abdomen alone was immersed, then there were incidents of bleeding via the rectum, more mesenteric hemorrhage and less pulmonary damage.

One author, ¹⁶ by injecting air into the peritoneal cavity prior to the detonation, produced injury to the liver, spleen, and other solid viscera. In addition, by tying off three loops of bowel (collapsing one, filling another with saline, and filling the third with air), he found that only the air-containing loop sustained injury.

With the data presenting that, primarily the damage is produced in the lung and the air-filled viscera, it is now possible to postulate a mechanism of death. In order to do this, it is necessary to divide the animals into two groups: early death - that occurring within a few minutes to one hour after the injury and late death that occurring after one hour. Early death probably was due to the pulmonary lesions. This is supported by the data of Clemedson, ⁹ He measured the respiratory function in animals subjected to blast injury. In the slightly injured animals, the respiratory minute volume increased but returned to normal, the O_2 consumption decreased but gradually returned to the normal, the arterial O2 saturation declined rapidly but was corrected by pure oxygen (indicating the absence of a major arteriovenous shunt) and a respiratory alkalosis appeared - the CO_2 was easily eliminated. In contrast, the severely injured animals tended to have a high respiratory minute volume that continued to death, a low O_2 consumption, a low O2 arterial saturation - corrected by pure oxygen and a respiratory acidosis due to the retention of CO_2 . Essentially the slightly injured animals were able to maintain an adequate elimination of CO_2 , where the severely injured animals did not have an adequate perfusion and ventilation of enough lung tissue to support life. It is interesting to note that there was no correlation between the degree of O_2 saturation and the extent of the pulmonary hemorrhage. This is similar to that seen in lobar pneumonia, where a reflex may decrease the blood flow to a large area of consolidation and underventilation. This tends to correct the blood gas picture.

There are also a few workers that believe the early death is due to central

nervous system lesions. 3, 15, 27 It was shown that early death may not correlate well with respiratory injury.⁵ Young reported petechial hemorrhages and edema in the vital areas of the brain following blast injury.²⁷ He felt that these may be caused by a rapid increase in the venous pressure, secondary to the compression of the chest and abdominal venous reservoirs by the pressure wave. When this increase is transmitted to the brain, the small blood vessels may rupture and produce the injury. Another author ¹⁵ postulated that rupture of the alveoli, by the pressure wave, may result in the release of air emboli. He demonstrated air in the cerebrovascular system, if the animal was upright during the blast.

Late death could result from the complications of the respiratory, abdominal and neurological injuries.^{5, 6, 16} These could include bronchopneumonia, peritonitis and coma with all of their sequelae. Most of the animals that may have died in the late period were sacrificed. A definite statement cannot be made on the mechanism of their death.

The human work in this area consists of collections of case reports. As a result, the magnitude of the explosion and the position of the victim relative to the charge is unknown. It is not known with certainty, for example, if both the abdomen and the chest were submerged at the time of the explosion. The case reports all came from experiences encountered in the Second World War and involved men in the water, secondary to an attack on their ships. 6, i4, 19, 20, 22, 23 An illustrative example of the subjective feeling is taken from a paper by Cameron: 6

"... After approximately 20 minutes in the water and whilst swimming around collecting the men into groups, there was a sudden explosion which I judged to be about 100 to 150 yards away. This I thought to be a bomb, although no sound of aircraft was heard. It is hard to describe actually, the effect it had on me, in medical terms, but it was just like a band being placed around my waist and being quickly tightened, together with a collapsed feeling as is felt after a sudden diarrhea evacuation. I decided that if it was a bomb, more would follow, so attempted to turn over on my back, but found that everything had gone numb from my waist down and I could not raise my legs. This condition lasted for about one hour, then a tingling sensation like that of 'pins and needles' set in until the movements of my legs became more or less normal again. I could not climb aboard the destroyer that picked me up so was hauled up on a rope's end and helped down to the wardroom, but, thinking of the lads still left in the sea I went back on deck and did what I could to assust. The excitement probably took my thoughts off my own self, for I cannot remember feeling any further effects until after landing and going to bed at the hotel, when severe griping pains started, particularly on passing my motions. The bowels were open at least 7 or 8 times a day and it was then that I first saw that blood of a normal colour was being passed. This continued for a week, but the slack motions and pain continued for a month before becoming less frequent....."

The patient in this case was wearing a life jacket; this afforded some protection to the chest area. The author believed that the initial tight feeling and the transient neurological symptoms were due to spinal concuscion. Perhaps, this is a human example of the mechanism postulated by Young.²⁷

Other symptoms include hemoptysis, vomiting - especially if visceral perforation has occurred, and testicular pain. This latter was reported by Breden⁴ and probably represents a hemorrhage beneath the tunica albuginea. An alternate explanation was given by Hamlin¹⁷who believed that this represented neurological compression,

According to a review of 80 nonoperated cases by Cameron, 6 abdominal pain was the most common symptom and lasted from a few days to a few months after the injury. Melena was present in 82% of his cases and persisted up to four months. Hemoptysis occurred in 20% and hematemesis in 14%. No abnormalities were found on the upper gastrointestinal nor the barium enema radiological studies. Sigmoidoscopy rarely revealed areas of petchial hemorrhages in the rectum and the sigmoid colon.

It must be recalled that many of the cases were not seen medically since they died in the water. The cause of death in these cases can only be postulated by referring to the animal data.

Of 20 cases that came to surgery,⁶ there were 10 deaths. The most common lesions were retroperitoneal and subserosal hemorrhage; these occurred in all of the cases. Perforations of the caecum were present in 9 cases, in the ileum in 7 cases and were multiple in 4 cases. These appeared within one to two days of the injury. Late perforations were reported in two cases, one appearing on the 8th and one appearing on the 10th day post injury. These were probably due to infection of an area of hemorrhage in the bowel wall with subsequent rupture. Bronchopneumonia complicated many of the cases.

The experience of the other authors is similar with retroperitoneal hemor-

rhage and perforation of the large bowel and the ileum representing the most common lesions in the abdomen. Pugh²² described two unusual cases of underwater blast injury to the abdomen. One was a perforation of the jejunum two feet below the ligament of Treitz. This case was later complicated by a volvulus. The second case was that of a subphrenic abscess in a non-operated victim of the blast.

The vulnerability of the low abdominal hollow viscera to blast injury probably is secondary to their containing relatively large amounts of gas and having relatively non-muscular walls.

From the radiologist's point of view, ¹³ the chest X-ray revealed most commonly a linear zone of increased density that was situated in the costophrenic angles and tended to parallel the postero-lateral ribs. Wedged shaped densities, confined to the lower lobes, were also seen. The densities in the costophrenic areas were described as being fainter and less sharply defined than either atelectasis or bronchopneumonia. The abdominal film may reveal gaseous distention of the small bowel and the colon that is consistent with a paralytic ileus. Free gas may be seen in the peritoneal cavity in the case of a perforated viscus. Occasionally, small bubbles are noted outside of the gastrointestinal tract; these are also seen in perforation and may represent dissection of the bowel wall by the gas.¹³ Abnormal soft tissue shadows were seen unilaterally and may be collections of blood, fluid or perhaps an exudate. Barium studies occasionally showed abnormal mucosal patterns such as coarse jejunal markings,

'puddling' in the jejunum and the ileum, and segmentation. (There were no reports of a malabsorption syndrome in the works that were reviewed.)

Neurologically, ¹⁷ many of the patients complained of a sharp pain in the chest, the abdomen, the testes, and down the legs. This and the common paralytic ileus may be due, at least in part, to a force that is applied to the peripheral and the autonomic nerves. Headache, depressed reflexes, and sensorial changes were also reported. These latter are usually transient. Hamlin ¹⁷ believed that they may be due to rapid shifts in the vascular reservoirs. Rare subdural hematomas may also be seen. ¹⁷

The management of the underwater blast injured patient is similar to that for one who is suspected of having total body trauma. The patient should be admitted for observation for the development of complications to the injuries described above. The patient may be asymptomatic immediately after the injury and may show no external signs of injury, ecchymoses, lacerations, etc. Probably all patients with a bonafide history of blast injury to the abdomen should be placed on intravenous fluids and kept NPO, initially. The appropriate studies should be obtained to ascertain if lung or abdominal injury has occurred. These may include a complete blood count and differential, chest X-ray, and plain film and upright of the abdomen.

The major decision involves the need for surgical intervention in the abdomen. The signs of peritonitis rebound, rigidity, tenderness, and decreased bowel sounds may not require intervention. These may all be secondary to small hemorrhagic areas scattered throughout the peritoneal cavity. ¹⁹ Bleeding from the rectum also may not be an indication for operation; since it may be due to small hemorrhages in the mucosal lining of the lower gastro-intestinal tract.

Naso-gastric suction may be indicated, especially, if a paralytic ileus exists.

The physical findings of consolidation, pneumothorax, or pleural effusion may be elicited. These must be treated appropriately depending primarily on the size of the area involved and its effect on the respiratory functions. Blood gases may also be indicated as a means of following these functions. Antibiotics may be needed on an individual basis.

Finally, if after an appropriate period of time, the patient still shows the signs of peritonitis and his condition is not improving, surgery may be necessary. A perforated viscus may be present in spite of other studies to the contrary.

CONCLUSION

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The subject of underwater injury has been reviewed from both the physical and medical aspects.

There is one area of almost total lack of knowledge; this involves the prediction of the injury produced by a given explosion at a given range. It is now obvious that this varies not only with the size of the charge but also with the type and contour of the bottom, distance to the diver, depth of detonation, depth of the diver, the presence of nearby large ships or banks and thermal layers between the diver and the charge. The present range formulae can be criticized since they do not allow for these variables. In addition, they are based on animal experiments and, as was mentioned in a previous section, there is a species difference in susceptibility to underwater blast. The formulae are also derived from explosions where the animals are at or near the surface; this data may not be appropriate if the diver is much under the water. It should be kept in mind that the use of these formulae, in a real situation, may lead to a false sense of security and injury to the swimmers.

With the kncwledge of the type and mechanism of injury, it is possible to advise a course of action in the event of an underwater explosion. The most important and probably the only sure way to avoid injury is to be out of the water at the time of the blast. The blast wave will not pass much of its energy through the surface of the water. If the above is not possible then one should try to get at least his chest and abdomen out of the water; this may be done by pulling oneself onto a piece of debris or floating on one's back. A final means involves interposing an air containing appliance between the chest and abdominal areas and the blast wave, i.e. - a life vest. This tends to reflect some of the wave at its water-air interface and absorb some of the remainder.

Since there have been no recent reviews of human blast injury since the war, (1945), it is difficult to predict the mortality. It is probably safe to assume that those whom the theories indicate would have died in the water would still die. However, it is felt that once having reached medical aid, an individual's prognosis would be markedly improved over the figures given in those reports coming out of the Second World War. This is directly related to better surgical techniques, the availability of antibiotics, and better pulmonary care.

SUMMARY

Underwater blast injury has been shown to be due to the effects of the pressure wave as it travels through the tissues of different densities. The amount of injury probably correlates most with the impulse function of the wave. This may be affected by many factors.

The most constant area of injury appears to be the lung, although the abdominal air-filled viscera are frequently injured. Occasionally, central nervous system lesions are produced, probably secondary to blood shifts or air emboli. The prognosis depends on the extent of the initial injury and the time that elapses before adequate medical care is obtained.

At present, the only sure way to avoid injury due to an underwater blast is to be out of the water at the time of the charge. No formula is now available that can predict accurately the damaging range of a given explosion. A few protective measures are listed that might aid survival in the event of an underwater blast.

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