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TEMPORARY CAVITY EFFECTS IN BLOOD VESSEL INJURY
BY HIGH VELOCITY MISSILES

JOSEPH J. AMATO, MAJ, MC, NORMAN M. RICH, LTC, MC, NOEL S. LAWSON,
MAJ, MC, RONALD P. GRUBER, CPT, MC, AND LAWRENCE J. BILLY, CPT, MC
BIOMEDICAL DEPARTMENT, BIOPHYSICS LABORATORY
RESEARCH LABORATORIES, EDGEWOOD ARSENAL, MARYLAND 21010

The wounding potential of high velocity missiles is greatly accentuated by the explosive effects of the temporary cavity. The missile transfers energy to the tissues within microseconds after impact. Tissue along the wound tract expands laterally forming a cavity with internal pressures as high as 100 atmospheres (1,500 pounds per square inch).⁽¹⁾ Description of the extent and nature of destruction to the various tissues by this phenomenon remains incomplete, and the effect upon blood vessels has been controversial.

Low velocity missiles have been known to push the blood vessels aside as the missile traverses the tissue. An early account of these injuries was described in 1862 by George Macleod in War in the Crimea.⁽²⁾ Huguier in 1848 first observed the bursting effect of high velocity missiles on soft tissues.⁽³⁾ He attributed this effect to the dispersion of water particles. This hydrodynamic theory was first experimentally tested by Kocher in 1876⁽⁴⁾ and confirmed by Sir Victor Horsely in 1894.⁽⁵⁾ Destruction of tissues appeared to correlate with the amount of fluid within the tissues. In 1898, Woodruff applied a concept of marine engineering to wound ballistics. The term cavitation was used to describe the vacuum immediately following a solid object moving rapidly through a gas or fluid.⁽⁶⁾ He predicted the formation of a cavity following a high velocity projectile and very accurately illustrated its movements. Stevenson in 1910⁽⁷⁾ and LaGarde in 1916⁽⁸⁾ accepted the cavitation theory which was also described as the theory of accelerated particles. Lesions to the blood vessels were described as one of the chief characteristics of the high velocity bullet. They stated that the arteries are neatly sheared as though it were done with a cutting instrument leaving the edges without irregular lacerations. Observations of the effect of the temporary cavity upon vessels were first made by Wilson in 1921.⁽⁹⁾ He described slit-like lesions in areas distant to the segment actually touched by the bullets. Similar lesions occurred in near misses.

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Photographic documentation of the formation of the temporary cavity was first obtained by Black and associates.⁽¹⁰⁾ Contrary to Wilson's observations, they stated that tissues of a highly elastic nature such as arteries were able to escape both anatomical and functional injury. Experiments by Harvey et al at Princeton in 1946 verified this opinion and demonstrated for the first time that the artery when not directly injured by the missile was pushed aside by the temporary cavity.⁽¹¹⁾ They also concluded that the blood vessel escaped gross injury.

In the recent Korean Conflict, Jahnke and Seeley⁽¹²⁾ described microscopic changes within the blood vessel wall adjacent to the grossly injured segment. Failures of repair and increased morbidity were ascribed to these changes. They arbitrarily advocated resection of one centimeter of artery beyond the area of gross damage for more successful surgical repair of the vessels. The need for further investigation was stressed.

Experiments were designed in our laboratory to study the pathophysiology of ballistic injury to the blood vessels. The mechanism of wounding in arteries has been previously reported.^(13,14) This communication is concerned with the effects of the temporary cavity including wounds produced by the M-16 bullet. Sequential events which occur in the mechanism of injury by both direct and indirect hits are documented by angiography and high speed photography. To our knowledge we have presented the first thorough documentation of the effect of the temporary cavity upon arteries.^(13,14) Observations of microscopic mechanical injury are also discussed.

METHOD

In the initial experiment, the femoral arteries of thirty anesthetized dogs were exposed and injured with missiles of specific velocities. A 0.25 inch sphere weighing 16 grains was accurately calibrated to be fired at 1,000, 2,000 and 3,000 feet per second.

X rays of the extremity were taken at intervals following injection of 90 percent hypaque in some vessels. The Fexitron flash X ray system which is capable of producing pulses of one-tenth microsecond duration was used. Instrumentation was designed to inject the dye, fire at the vessels, and obtain X rays at varying intervals.

High speed photography of the mechanism of injury was taken at 3,750 to 4,500 pictures per second utilizing a Hycam Camera. This camera is a 16 mm high speed rotating prism camera used especially for photography of short-lived events.

To study the microscopic mechanical changes immediately post wounding, segments of the injured vessel approximately 20 millimeters on either side of disruption were excised. These were prepared in the normal manner with Hematoxylin and eosin stain. The Verhoeff-VanGieson method for specific elastic and connective tissue staining was used.

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A second experiment was designed to visualize more adequately the vessel in an isolated environment. Segments of the femoral arteries measuring six to nine centimeters in length were removed from fourteen dogs. These vessels were filled with safranin dye or hypaque, weighted on one end and suspended within a 20 percent gelatin solution which was used to simulate normal muscle tissue density. These vessels were sighted, injured and photographed as described above.

Because of the small caliber of these arteries, a third series of experiments were performed utilizing twenty segments of calves vessels. Both width and thickness of these arterial walls approximated the size of the human femoral vessels. Injury was produced in the first ten segments with 0.25 inch spheres so that we could solely obtain the effects of the temporary cavity at specific varying velocities. The M-16 rifle bullet was used in the remaining ten segments to document other factors effecting the behavior of the military bullet. The M-16 rifle is currently used in Vietnam and utilizes a 55 grain bullet fired at a muzzle velocity of 3,250 feet per second.

RESULTS

Angiograms of the femoral artery in situ are shown at micro-seconds after impact (Fig. 1). The 0.25 sphere struck the artery at a velocity of 3,000 feet per second. The vessel was neatly transected by the sphere without moving the remaining artery. However, within five to ten microseconds after impact, the proximal and distal segments of the injured artery were compressed against the cavity wall (Fig. 2). High speed photography demonstrated the suspended vessel in gelatin which was struck by a sphere at 3,000 feet per second (Fig. 3) with the subsequent formation of the temporary cavity. Again, it is seen that the vessel was cut by the missile but violently hurled sideways and compressed laterally (Fig. 4,5,6).

The M-16 injury was similar to that of the sphere. The calves' vessels were stretched by the undulation of the temporary cavity in an indirect hit (Fig. 9,10). Stretching and swaying of the torn ends were documented when the vessel was struck directly by the 55 grain bullet at a striking velocity of 3,250 feet per second (Fig. 11,12).

The contrasting effects of the low velocity missile which struck the gelatin block at 1,000 feet per second were seen with minimal formation of a temporary cavity (Fig. 7) and stretching forward of the blood vessel until it was severed (Fig. 8).

Microscopic mechanical changes in these blood vessels correlate well with previous descriptions of changes along the blood vessel wall (9,12,20). The endothelium was frequently lost. Small and large breaks were seen in the internal elastic membrane. There were microthrombi formation within the media with disruption and less commonly herniation of the media through the external elastic membrane of the adventia.

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DISCUSSION

The wounding potential of high velocity missiles is a complex subject which is beyond the scope of this paper. However, several important factors, must be included in discussing ballistic behavior of the bullet in relation to wounding capacity. These are: A. The energy transmitted to the tissues by the missile. B. The direction of the transmitted energy released as determined by the shape and motion of the missile. C. The density of the tissues through which it travels.

A. The kinetic energy theory of wounding capacity indicates that the energy expended is directly proportional to the mass and to the square of the velocity ($KE :: M \times \frac{V^2}{2g}$). Thus, doubling of the velocity quadruples the kinetic energy of the missile. As the missile comes in contact with the artery, it bisects the wall sharply as demonstrated in Figures 1 and 3, but energy is transferred to the tissues. The arterial ends are accelerated away from the wound track radially within the formation of the temporary cavity (Fig. 2-6). The development and decay of the cavity occurs within 20 microseconds after impact causing the segments to undulate and be thrashed about within the cavity until it comes to rest (Fig. 12). Cavitation is primarily due to the energy released. However, both its size and shape are modified by the configuration of the missile and striking angle at moment of impact. To avoid the above factors a steel sphere was utilized in the majority of our experiments as was suggested by Woodruff in 1898.⁽⁶⁾

B. The direction of the energy released to the tissues is governed by shape of the missile and its motion in flight. A brief description of the bullet's motion would include yawing, tumbling, precession and nutation. When a bullet deviates from the straight line of flight, in its longitudinal axis, it is described as yawing. Tumbling is simply described as the action of forward rotation around the center of mass. As the bullet yaws and/or tumbles, it is also spinning laterally around its center of mass. This is termed precession. A fourth motion of the bullet in flight and perhaps the most difficult to visualize is nutation. Hopkinson appropriately describes nutation as the rotational movement of the bullet in small circles progressing forward in a rosette pattern.⁽¹⁵⁾ The varying severity of wounds and amount of tissue destruction by identical missiles can be explained in most part by the angle of the bullet to the tissue at moment of impact.

The density of tissues is a third factor in the explanation of wounding effects on tissue. As mentioned earlier, Kocher in 1876⁽⁴⁾ demonstrated that tissues which contained large quantities of water were most severely damaged. Daniel in 1944 demonstrated that the severity of high velocity wounds increased with the specific gravity of the tissue struck.⁽¹⁶⁾ DeMuth stated in his review that bone injuries are most severe while lung tissues are least affected.⁽¹⁷⁾ Injury to the blood filled arteries can be extensive. This perhaps, is only modified by the elastic nature of the artery and its ability to absorb the energy transmitted by the missile. When

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large amounts of energy exceed the tolerable elastic limits of the cellular structures within the arterial wall, microscopic injury can occur. When the vessel is indirectly struck by the missile, the amount of energy is proportional not only to the velocity of the missile, but to the proximity of the vessel to the center of the temporary cavity, and to size of the blood vessel. This mechanism of injury is not clearly understood by a majority of surgeons faced with arterial repair.

The extent of debridement of the injured vessel has been controversial. Wilson stated that the slit-like lesions within the intima may cause thrombosis, fatal secondary hemorrhage or aneurysmal dilatation at a later date.⁽⁹⁾ The treatment for arterial injury at that time was simple ligation. In the past the advice of resection of one centimeter of arterial wall beyond the area of gross injury by Jahnke and Seeley⁽¹²⁾ has been followed by a majority of military surgeons. However, others have advocated the removal of one to two centimeter segments in high velocity wounds.⁽¹⁸⁾ Contrary to the above, one author has shown the extent of microscopic injury to be minimal and debridement of only 3 millimeters of tissue was deemed necessary.⁽¹⁹⁾

Recently, Rich, Manion and Hughes reviewed a series of clinical cases from the Vietnam Conflict and stated that there was no correlation between the microscopic damages which were present and the success of repair.⁽²⁰⁾ Adequate debridement with good surgical judgement was advocated. In our series, microscopic examination of the segments removed post injury have demonstrated as in humans, various changes within the arterial wall. The significance of the changes mentioned earlier remain unanswered at present in our experimental model.

An important aspect of this study is to realize that the low velocity gunshot wound is a different entity. The mechanism of injury is seen as a local stretching of the vessel wall with little or no formation of a temporary cavity. Because of this difference, proper assessment of the wound and knowledge of the nature of the wounding missile are necessary requirements in proper management of missile wounds.

SUMMARY

1. Historically, the effect of the temporary cavity upon arteries has been controversial.
2. We have presented angiographic and photographic documentation of low velocity and high velocity injuries to arteries.
3. The low velocity missile pushes the blood vessel ahead stretching it slightly before penetration and can be pushed aside with little or no formation of the temporary cavity.
4. The high velocity missile neatly cuts the arterial wall at impact but then the explosive effect of the temporary cavity crushes the ends of the artery against its walls.

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5. Severity of arterial damage is proportional to the velocity of the wounding missile. The energy transferred from the high velocity missile forms the temporary cavity. Other factors, such as, the configuration of the missiles vary the size and shape of the temporary cavity and extent of injury.

6. Varied tissue destruction by identical missiles at equal velocity can be explained by the shape of the missile, its motion in flight, and most important the angle to the tissue at moment of impact.

7. Knowledge of the ballistic behavior of high velocity missiles in causing arterial damage is necessary for the proper evaluation, and management of missile wounds.

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LEGENDS OF FIGURES

Figure 1. Angiogram of the femoral artery within microseconds after impact. Impact velocity was 3,000 feet per second.

Figure 2. Angiogram of the femoral artery compressed against the wall of the maximal temporary cavity.

Figure 3. A segment of femoral artery is neatly sheared by a 16 grain sphere at 3,000 feet per second.

Figure 4. The initial phase of the temporary cavity is seen. The arterial segments are bent laterally.

Figure 5. An intermediate phase of cavitation is demonstrated. Pressures as high as 100 atmospheres have been reached.

Figure 6. Maximum formation of the temporary cavity. Note the curled segment of vessel in the upper half of the cavity.

Figure 7. High speed photograph of a segment of femoral vessel being approached by a sphere at 1000 feet per second.

Figure 8. The artery is shown stretched forward prior to being severed by the missile.

Figure 9. A M-16 bullet was fired at 3,250 feet per second missing the femoral artery. The artery however is slightly stretched.

Figure 10. The vessel which was missed by the M-16 is thrashed about and distorted laterally by the temporary cavity.

Figure 11. A direct hit with an M-16 bullet severs the vessel and compresses the ends forcefully. The crushed segments can be seen against the wall.

Figure 12. The distal and proximal ends of the femoral vessel sway within the undulating temporary cavity until it comes to rest.

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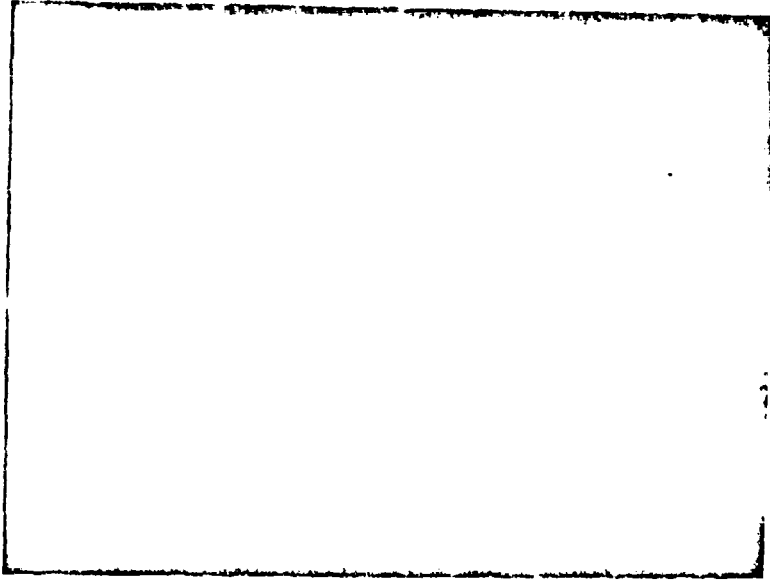


Figure 1. Microseconds after impact at 3000 feet per second.

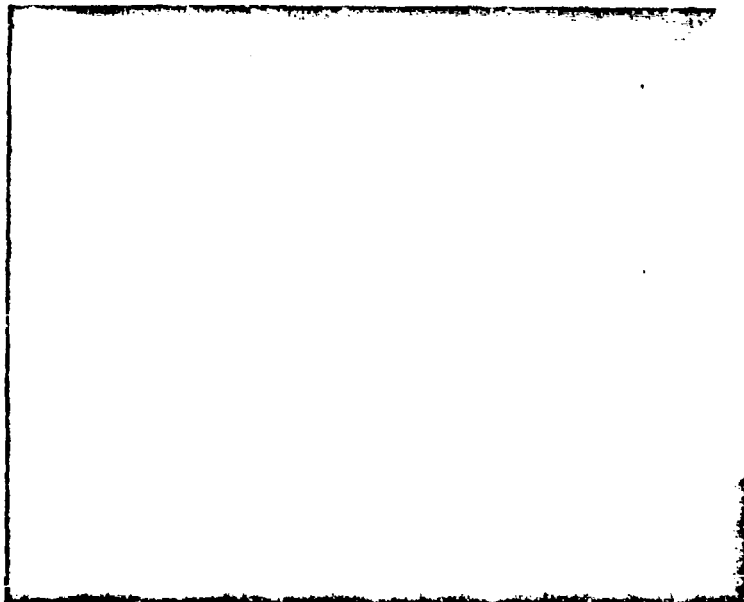


Figure 2. Maximum formation of the temporary cavity.

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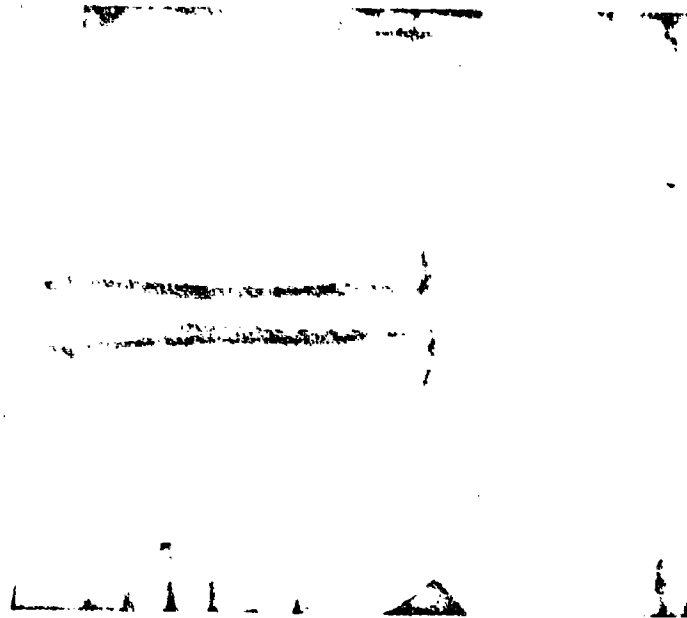


Figure 3. The vessel is transected by the missile in gelatin.

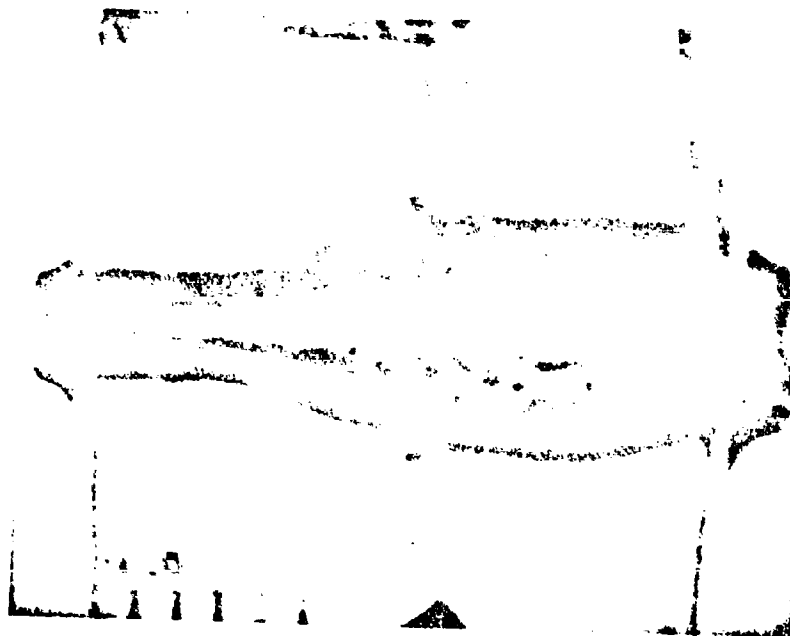


Figure 4. Early formation of temporary cavity.

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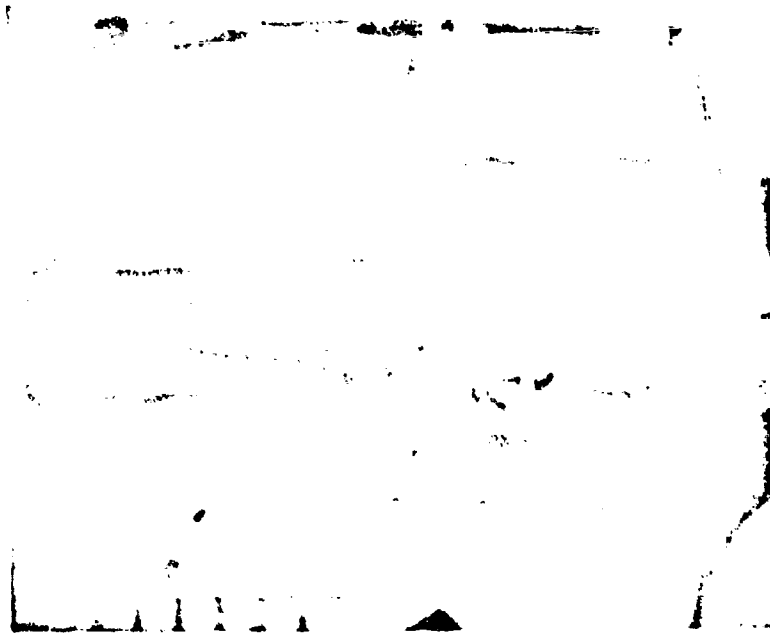


Figure 5. Intermediate phase of temporary cavity.

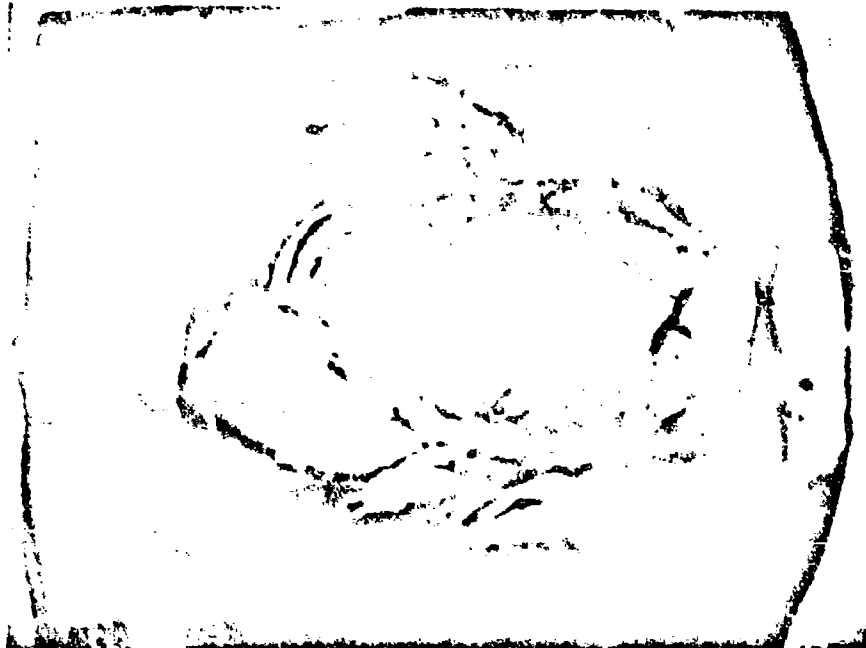


Figure 6. Maximum formation of the temporary cavity.

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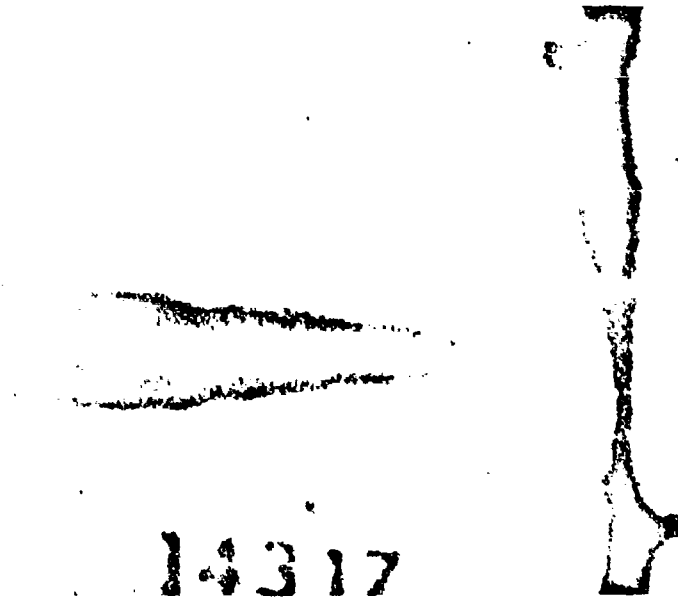


Figure 7. Vessel is approached at 1000 feet per second.

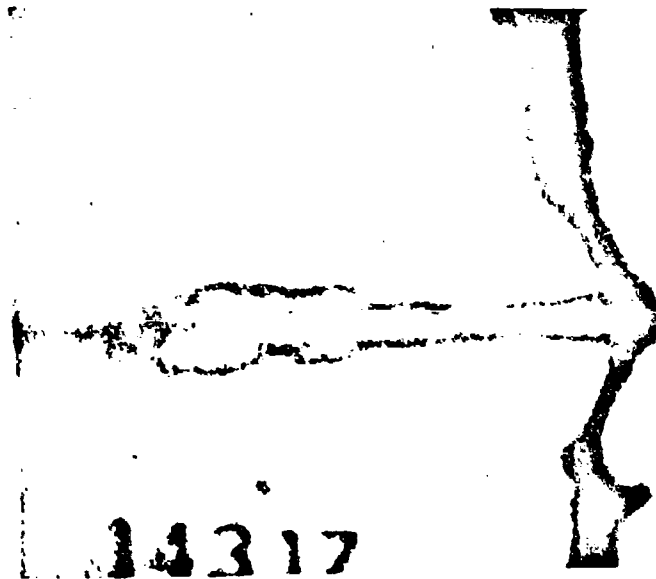


Figure 8. Vessel is stretched by the low velocity missile.

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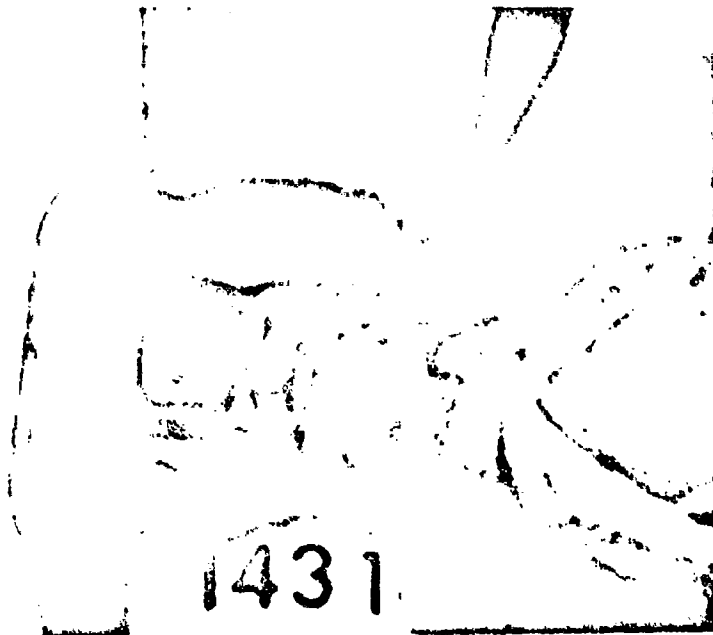


Figure 9. Near miss of vessel with M-16 bullet.

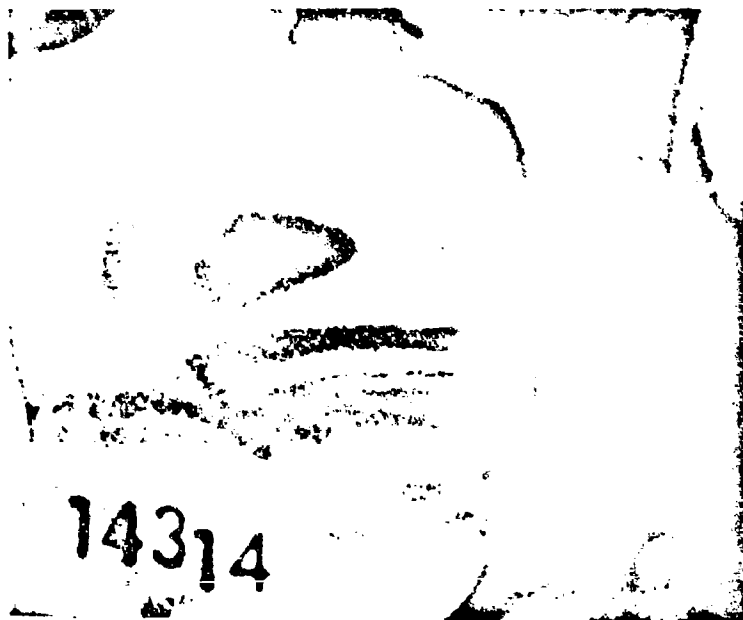


Figure 10. Stretching of the vessel - near miss M-16 bullet.

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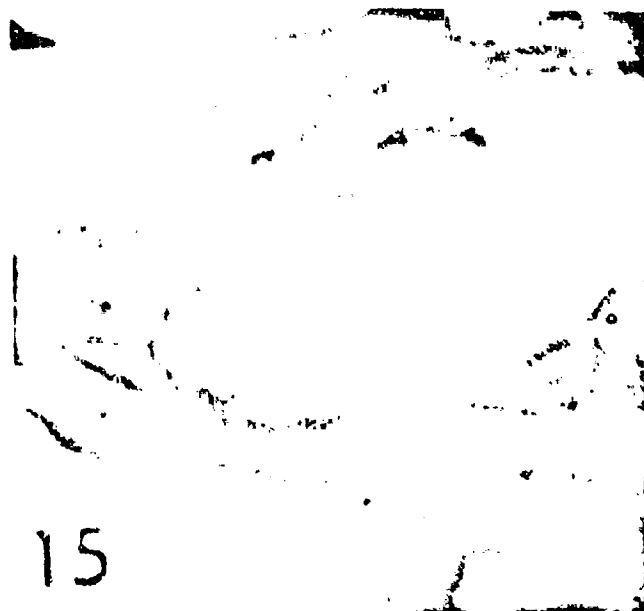


Figure 11. Direct hit with the M-16 - vessel is compressed laterally.

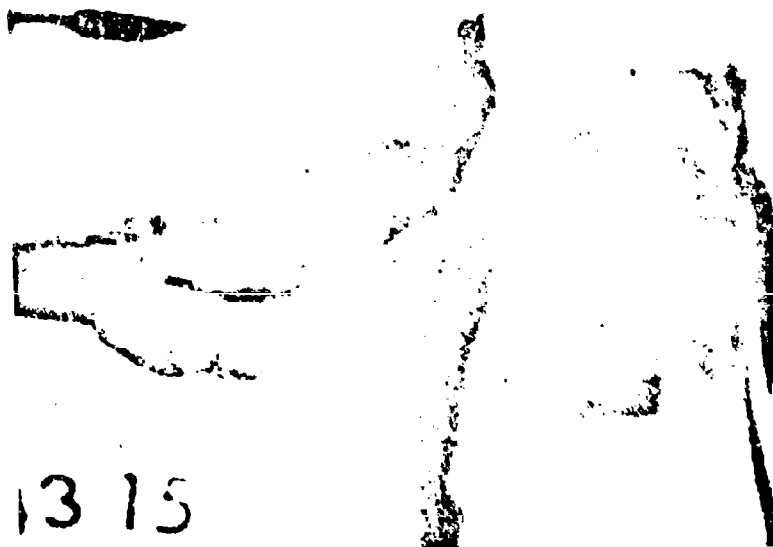


Figure 12. M-16 direct hit - vessel is thrashed about.

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