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BIOLOGICAL EFFECTS OF UNDERGROUND NUCLEAR TESTING ON MARINE ORGANISMS. I. REVIEW OF DOCUMENTED SHOCK EFFECTS, DISCUSSION OF MECHANISMS OF DAMAGE, AND PREDICTIONS OF AMCHITKA TEST EFFECTS

Presented by

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BIOLOGICAL EFFECTS OF UNDERGROUND NUCLEAR TESTING ON MARINE ORGANISMS. I. REVIEW OF DOCUMENTED SHOCK EFFECTS, DISCUSSION OF MECHANISMS OF DAMAGE, AND PREDICTIONS OF AMCHITKA TEST EFFECTS

Merritt (1972, 1973, and this Conference) has described the characteristics of the waterborne shock waves created by the Milrow and Cannikin underground nuclear detonations. The following introduces a portion of the investigations conducted to relate the effects of these perturbations upon organisms in the marine environment adjacent to the Amchitka Island test site. Since 1967, as a component of the US AEC's Bioenvironmental Safety Program on Amchitka, coordinated by Battelle Memorial Institute's Columbus Laboratories, the University of Washington's Fisheries Research Institute (FRI) has performed task-specific studies concerning the effects of the nuclear testing on the area's marine fish, invertebrate, and algal communities. This discussion presents (1) the level of our understanding of nuclear-induced or similar shock wave effects upon fish, (2) the potential mechanisms of damage applicable to the Amchitka situation, (3) the resulting prediction of effects formulated preparatory to the Cannikin test, and (4) our experimental approach to documenting the actual effects of the test. John Isakson (this Conference) continues this discussion with the empirical results of our experiments, observations, and studies and will summarize the effects of the Milrow and Cannikin tests on marine organisms as assessed by FRI and some of the other biological investigators.

An underground nuclear detonation-induced shock wave can produce a variety of biological effects on the adjacent marine environment. It may directly inflict physical damage on coastal marine organisms by stressing their body tissues, organs, and contained air spaces either mechanically or through the effect of pressure differential. It may also alter the habitat, especially in the ecologically sensitive intertidal zone, and thereby indirectly produce distinct changes in species composition, distribution and/or abundance.

Localized habitat disruption associated with nuclear tests on Amchitka previous to Cannikin has been well documented (Seymour and Nakatani, 1967; Kirkwood, 1970; and Merritt, 1970). The biological effect on bottom organisms caused by the movement of the sea floor may be significant, but little definitive documentation existed. The correlation between measured shock wave hydrostatic parameters and observed biological effects had not been adequately established for shock conditions similar to the Amchitka situation. Thus, the characteristics of waterborne shock waves specific to biological damage were assessed through the literature in order to better predict the Cannikin effects and to design the experiments to test those predictions. This survey revealed comparable effects on fish originating from three sources exclusive of nuclear tests: (1) underwater explosions, (2) earthquakes, and (3) specific problem-oriented laboratory experiments.

Previous relevant investigations into the effects of underwater explosions on fish and other marine life have occurred in conjunction with naval ordnance tests in Chesapeake Bay (Bennett, 1947; U. S. Navy, 1947; Chesapeake Biological Laboratory, 1948; and Coker and Hollis, 1950), seismic oil exploration in the Gulf of Mexico (Gowanloch and McDougall, 1945 and 1946), Lake Erie (Ferguson, 1961), in California (Alpin, 1947 and 1962; Baldwin, 1954; Fitch and Young, 1948; Fry and Cox, 1953; Hubbs and Rechnitzer, 1952; and Hubbs et al., 1960), Oregon-Washington (Oregon

Fish Commission, 1962; and Rulifson and Schoning, 1963) and British Columbia coastal waters (Kearns and Boyd, 1965) and in Alaska (Bright, 1957; Alaska Department Fish & Game, 1959; and Roguski and Nagata, 1970) and concerning marine engineering activities such as the removal of Ripple Rock from Seymour Narrows in British Columbia (Thompson, 1958). Dr. Young (Young, 1973) has recently summarized, in part, the results of these investigations. Related experiments have also been performed in Philippine (Thiemmedh, 1949; and Ronquillo, 1950 and 1953) and Japanese coastal waters (Koyama, 1954; and Kuroki and Kumanda, 1961) and in the Soviet Union (Tsvetkov et al., 1972; and Drabkina and Vodovozova, 1973). Natural shock waves induced by earthquakes have also been known to produce fish mortalities (Kachadoorian, 1965; and Kirkwood and Yancey, 1965) as was observed in the 1964 Alaska earthquakes. In an analogous situation, shock wave induced or related fish mortalities have also been examined in association with the passage of fishes through dam siphons (Hogan, 1941), pumps (Foye and Scott, 1965), or hydroelectric power turbines (Muir, 1950; Rowley, 1955; Holmes and Donaldson, 1961; Cramer and Oligher, 1964; and Sutherland, 1972).

Two mechanisms of biological damage applicable to fish were indicated in these studies; (1) changes in pressure over (overpressure) and under (underpressure) ambient hydrostatic pressures, and (2) bulk cavitation. High explosives detonated underwater were shown to create instantaneous pressure changes which were especially injurious to classes of fish possessing air bladders. While many investigations correlated the rapid rising (i.e. 1-2 msec) peak pressure components of the shock with observed fish mortality or damage, a number of the more refined studies, such as the two conducted by Dr. Carl Hubbs and his associates (1952 and 1960), suggested that the negative pressure phases accompanying such high explosive shock waves (see Cole, 1948) were responsible for the more deleterious mechanisms of damage to fish. Other, slower burning explosive shock waves (i.e. risetimes of 6-7 msec) which produced up to four times the peak overpressures as high explosive waves but which did not produce underpressures or decompression pulses, were essentially non-injurious.

Risetimes for nuclear-induced shock waves traveling through rock (Merritt, 1973a and this Conference) are exceedingly longer than in explosion produced waves, e.g. in the order of 70-100 msec for the Milrow test (Merritt, 1971). So, even if rapidly rising overpressure is a factor in explosion produced fish mortalities, it appears not to be the dominant factor in the case of nuclear-induced shock waves. Nuclear shock waves do, however, most commonly have a component of negative pressure in their wave form. Figure 1 illustrates the differences between the three types of documented shock waves - high explosive induced waves (50-1,000 cycles), slower burning black powder-produced waves (140-170 cycles), and low frequency nuclear-induced waves (10-15 cycles). Earthquakes produce shock waves in the order of 15-25 cycles.

While fish can apparently tolerate compression pulses of high amplitudes with risetimes longer than 1-2 msec, the longer a decompression phase persists, the more detrimental its effects. Thus, the first specific mechanism of potential damage from a nuclear-induced shock wave is associated with the negative pressures produced in the refraction cycle (Wentzell et al., 1969; Merritt, 1973a & b; and this Conference) of the shock wave as experienced by an organism positioned in the water column. As noted in the majority of literature-documented fish kills, the anatomical morphology, i.e. whether or not the fish has an air bladder[1] and

[1]The air bladder is a highly vascular, usually single-chambered, hollow, gas-filled organ located immediately below and along the length of the vertebral column between the alimentary canal and kidneys.

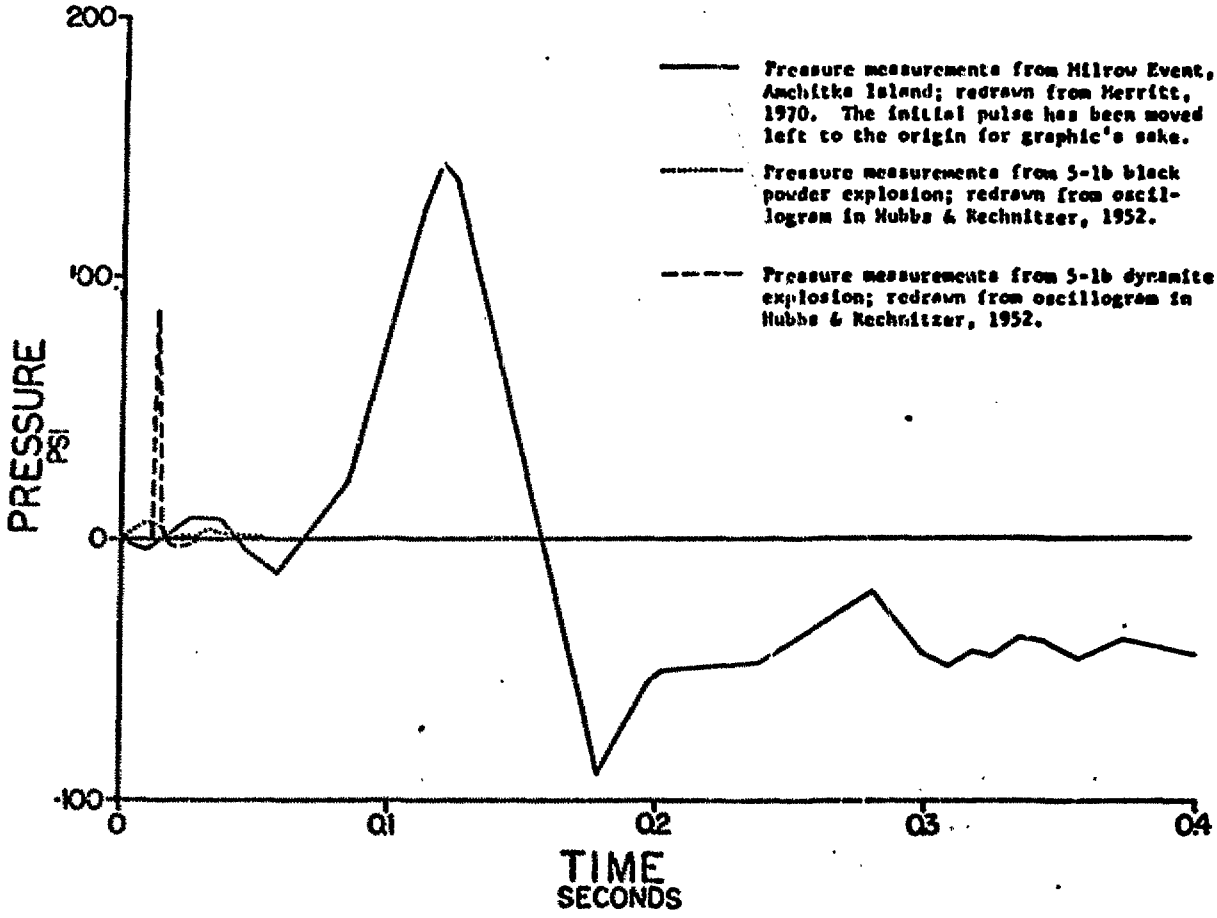

Figure 1. Pressure distributions and wave forms from Milrow Event, black powder and dynamite explosions.

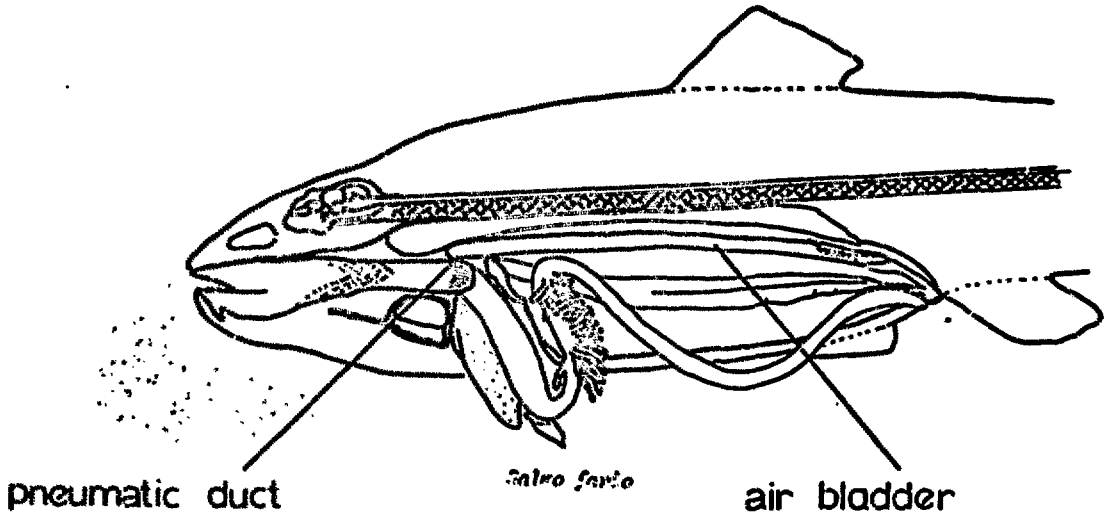

Figure 2. Diagrammatic illustration of physostomous fish, Salmo fario; physoclistous fishes lack the pneumatic duct connection between the air bladder and the esophegus. Redrawn from: Harmer, S.F. & A.E.Shipley (ed.) 1904. Cambridge Natural History, Vol VII. Hemichordata, Ascidians and Amphioxus, Fishes. MacMillan & Co., London. p. 255.

the form of the bladder, determines the scale of shock wave effect on the organism. Fishes without air bladders, predominantly those species living along the shore, in deep water or associated with the bottom (Brown, 1957; Jones, 1951, 1952 and 1957; and Jones and Marshall, 1953), are least susceptible to mechanical decompression damage. Lacking this sack of gas these fishes apparently do not suffer from the effects of the gas expansion during the passage of the decompression phase of a shock wave. Fish with an air bladder are of two forms - "physostomus," those possessing an open duct connecting the air bladder to the alimentary canal (Figure 2), usually at the pharynx; and "physoclistous," those forms without the pneumatic duct. Physostomus forms are prevalent among the pelagic surface dwelling fishes and the physoclistous forms among those inhabiting the midwater environment.

Although physostomus fishes are able to expel air bladder gas rapidly through the pneumatic duct, the volume of the air bladder generally cannot be manipulated rapidly (Sundnes and Bratland, 1972); although, there are indications that in some physoclists muscular tensions around the bladder can provide short-term volume regulation (Sundnes and Gytre, 1972). While the physoclistous fishes suffer potential rupture of the air bladder wall with significant decompression, the physostomus fishes will be less likely to suffer rupture as long as the passage of gas out through the pneumatic duct is at a volumetric rate higher than the expansion of that volume of gas still within the bladder. It is unknown whether or not this is possible over the potential decompression phase of a nuclear-induced shock wave. Both forms, however, are capable of losing buoyancy under minimum decompression conditions and it has been suggested that a 1% change in buoyancy is sufficient to disable a fish (Jones, 1952).

The decompression effect upon a fish with an air bladder will be a function primarily of: (1) the form of air bladder, (2) the tensile strength of the air bladder wall, (3) the resistance that the body wall and internal organs offer to the expansion of the bladder gas, and (4) the percentage volume of the air bladder gas relative to the ambient pressure. Generally, it has been suggested that in physoclistous fishes, a 3/5 reduction in relative pressure will be sufficient to rupture the air bladder wall (Jones, 1951 and 1952). Decompressions of from 14 to 50 psi have been documented to be lethal to physostomus fishes (Bishai, 1961a & b; Hogan, 1941; and Muir, 1959) and, theoretically, even lower values will apply to physoclists. Such threshold values should be considered in the light of the ambient pressures (position in the water column) in all cases. For, while the percentage volume expansion of the air bladder gas decreases with depth and the bladder at depth is thus more tolerant of expansion during decompression, the threshold underpressure value triggering cavitation (and thus limiting the potential underpressure) increases with depth and thus at depth the fish is subjected to a mechanically more intense underpressure stress than in a shallower situation.

To this point discussion has considered only the potential effects of the mechanical expansion of air bladder gas during decompression. The second potential mechanism relates to the phenomenon of bulk cavitation discussed by Cole (1948), Ackerman (1953), Cushing (1961 and 1969), Cushing et al. (1962), Walker (1966), Waldo (1969), Wentzell et al. (1969), Snay (1970), Gaspin and Price (1972), and Merritt (1973 and this Conference). At a threshold level, assumed to be absolute 0 pressure, gas bubbles expand to collapse with the extraction of energy from the source of decompression. It is becoming more apparent that cavitation of an organism's body fluids, those that contain gases, is also a potential mechanism of physiological damage under the correct circumstances. It has been theorized to be a factor in documented explosion-induced fish kills (Hubbs and Rechnitzer, 1952), as a mechanism of medical concussion (Ward et al., 1968) and in the nitrogen

supersaturation problem in the Columbia River system (D'Aoust and Smith, in prep.). Two aspects may be involved: (1) the accumulation of gas bubbles, especially nitrogen, in the vascular system of the organism, resulting in embolism, and (2) the expansion of the gas bubbles to the point of inflicting physiological harm upon the blood vessels and organs. There is no experimental data establishing whether or not the decompression phase of a nuclear-induced shock wave is of long enough duration to allow nitrogen or other gas bubbles to accumulate to the point of embolism damage. Theoretically, these gases should be completely redissolved upon initiation of the following compression phase of the shock wave and will not remain in the vascular system. The relatively short period of the decompression may also limit the volume of gas coming out of solution within the fish body fluids. The instantaneous expansion of any such gases, however, within the vascular system and organs may be carried to the point of rupturing vessel and organ walls, initiating lesions and hemorrhaging if not initially inflicting fatal damage. Such damage in the circulatory system, gill membranes or certain portions of the central nervous system would be immediately fatal but sublethal damage to the peripheral nervous system or the gas exchange system for the air bladder would also place the fish in a situation of likely predation and eventual indirect mortality. There is no data available, that I know of, biologically documenting this mechanism and the need for a comprehensive experiment is essential if we are to determine whether or not it is a real factor of shock wave phenomenon as related to biological injury.

Two little understood phenomenon of hemolymph changes in insects (Newcombe, 1966) and leukocytic composition of fish blood (Drabkina and Vodovozova, 1973), have also been linked to shock wave pressure changes but the mechanism is not well established and may not be applicable.

This is the point of information at which FRI stood preparatory to the Cannikin test. The results from the Long Shot and Milrow tests were inconclusive and sometimes ambiguous in the light of this knowledge. The only predictive experiments conducted preparatory to the Milrow test, involving mechanical compression of the sea otter, Enhydra lutra, and a few fish species (Wright, 1968), disregarded underpressure. Milrow test-time effects experiments (Kirkwood, 1970; and Merritt, 1970) lacked the comprehensiveness (i.e. no air-bladdered fish were utilized) to project the results to Cannikin. Thus, with the aid of Dr. Merritt and the Sandia Laboratories, we used the Milrow physical shock wave data and theoretical pressure functions to produce a map of the over- and underpressure regime as predicted for the marine environment adjacent to the Amchitka Cannikin test site (Figures 3 and 4). With a conceptualization of the distribution of peak pressures and limits of cavitation we superimposed this on our accumulated knowledge of the nearshore and offshore fish communities, their constituents (see Isakson et al., 1971 for description) and their known or suspected susceptability to shock waves.

Using this procedure, we predicted that sizable numbers of the endemic physoclistous forms, the Pacific cod, walleye pollock, and rockfish species, would probably be killed in the offshore (> 36 m) waters within the portion of that region subjected to high underpressure and cavitation (Kirkwood and Fuller, 1971). With the combined effect of underpressure cutoff by cavitation, surface spall and the predominantly physostomus forms of fish (mostly salmonids), the pelagic surface community was considered to be less vulnerable and potentially unaffected. The midwater fishes, about which we know the least, were potentially susceptible to lethal pressure changes in the case of the physoclists, but no quantitative predictions were possible. The bottom fishes, usually lacking an air bladder, were

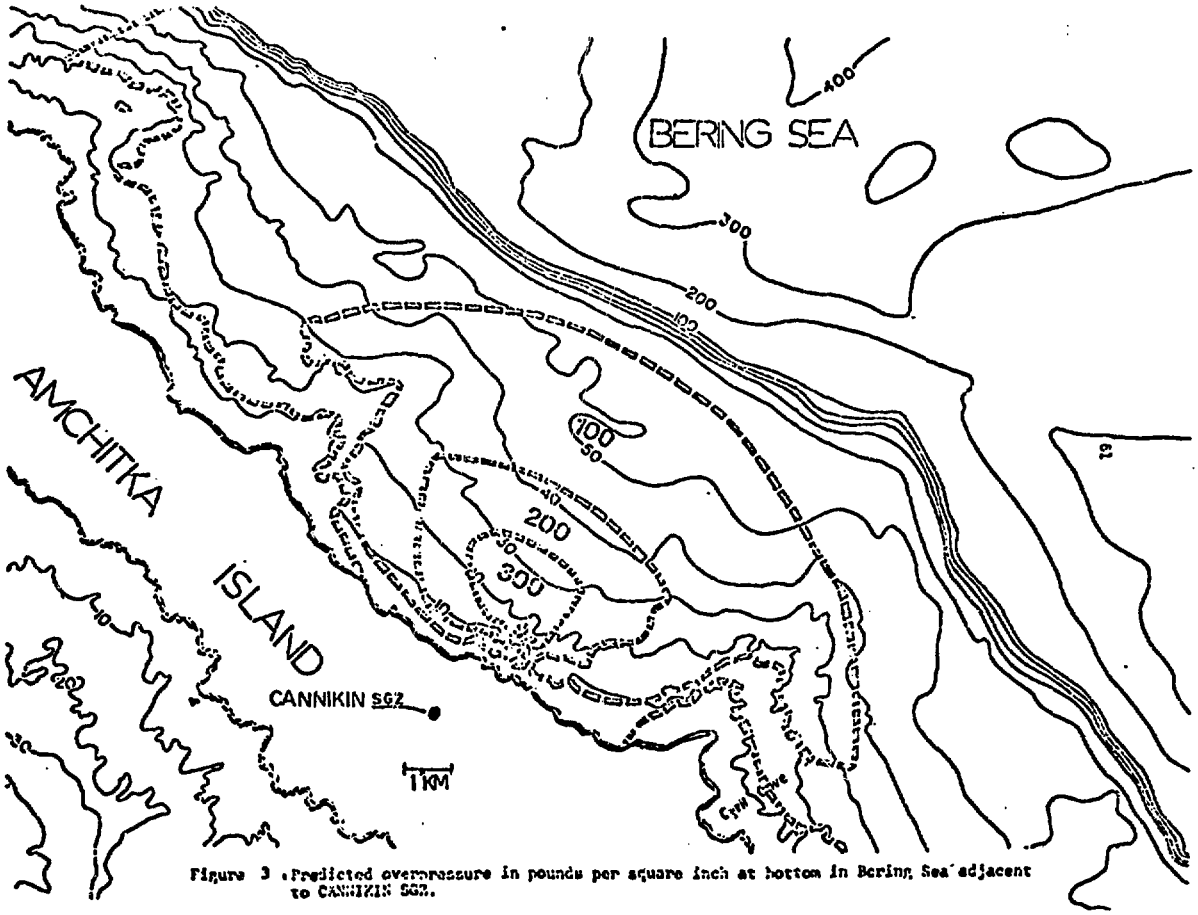

Figure 3 - Predicted overpressure in pounds per square inch at bottom in Bering Sea adjacent to CANNIKIN SGZ.

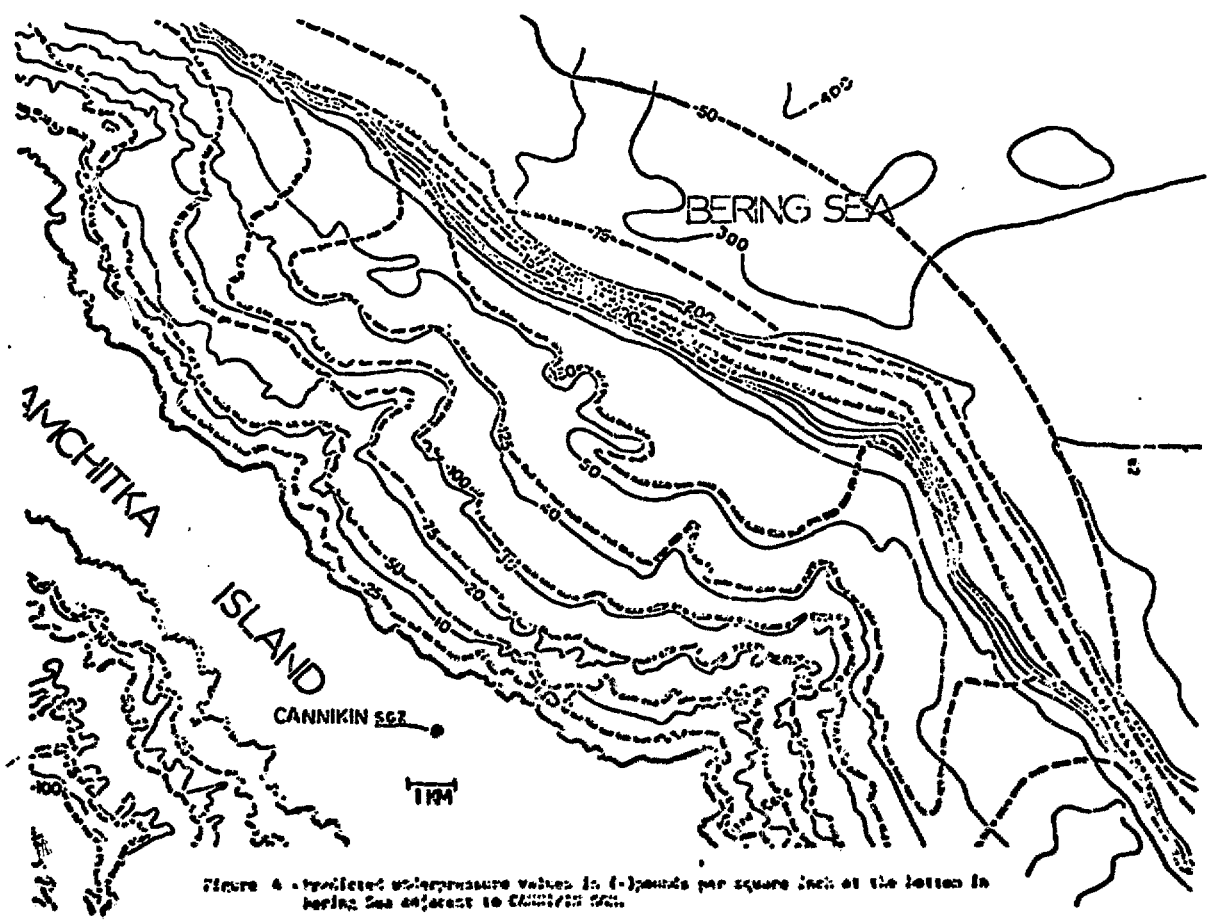

Figure 4 - Predicted overpressure values in (-)pounds per square inch at the bottom in Bering Sea adjacent to CANNIKIN SGZ.

assumed to be least susceptible to shock wave mortalities. The two nearshore communities were considered similarly but in the light of higher overpressure and lower underpressure values than further offshore. The nearshore physoclists, the Pacific cod and dusky rockfish, were considered to be in moderate danger of fatal shock wave pressures if attenuation within submarine canyons or other possible mechanisms of focusing underpressure should occur. Physostomus and non-air bladdered fishes were not predicted to be fatally inflicted. The prediction summarized by saying that, considering the relatively low percentage of Amchitka's fish communities involved, the reduction of its fish populations would not be irreversible and might, in fact, be undetectable.

With predictions in hand we then designed an experiment to test them. A system of live-cage strings (Figure 5) designed to hold fishes at the surface, bottom and at midwater were built to be set at varying distances from the test site in positions to maximize the spectrum of peak pressure changes (Figure 6). Fishes representative of the different communities were collected, tagged and were to be placed in their respective live-cages in the water column. Each cage was to be instrumented by Sandia Laboratory using passive gauges recording peak over- and underpressures. Unfortunately, recording active gauges which were used during the Milrow test documentation could not be used for various reasons. But the assumption that wave forms would be similar and could be extrapolated from the peak pressure values and land-based acceleration data is probably valid. This system of live-cages with the experimental animals was to be set in place the night prior to the Cannikin detonation but due to winds gusting over 80 knots pushing 30 ft seas the live-cage strings could not be set as planned and the test schedule proceeded without this documentation. Mr. Isakson will continue the discussion of Cannikin effects in this light.

In summary, the potential mechanisms of biological damage to fish resulting from a nuclear-induced shock wave appear to involve (1) mechanical damage from bottom acceleration and rockspall, (2) the synergistic effect of compression to decompression producing the mechanical expansion of gas spaces within the organism, (3) effects of cavitation, and (4) possibly the alteration of blood constituents. The indirect effects of the shock wave should also be considered in a truly ecological approach. Loss of fish or other marine organisms may reduce food resources for other species and place an unusual stress upon the community's food web and increased predation created by the influx of a formerly minor constituent may also be a real consideration. The determinants of biological damage, as concluded from our studies involve the (1) anatomical morphology, and (2) ecological characteristics of the various members of the fish community, and (3) the physical characteristics of the environment as produced by the introduced shock wave.

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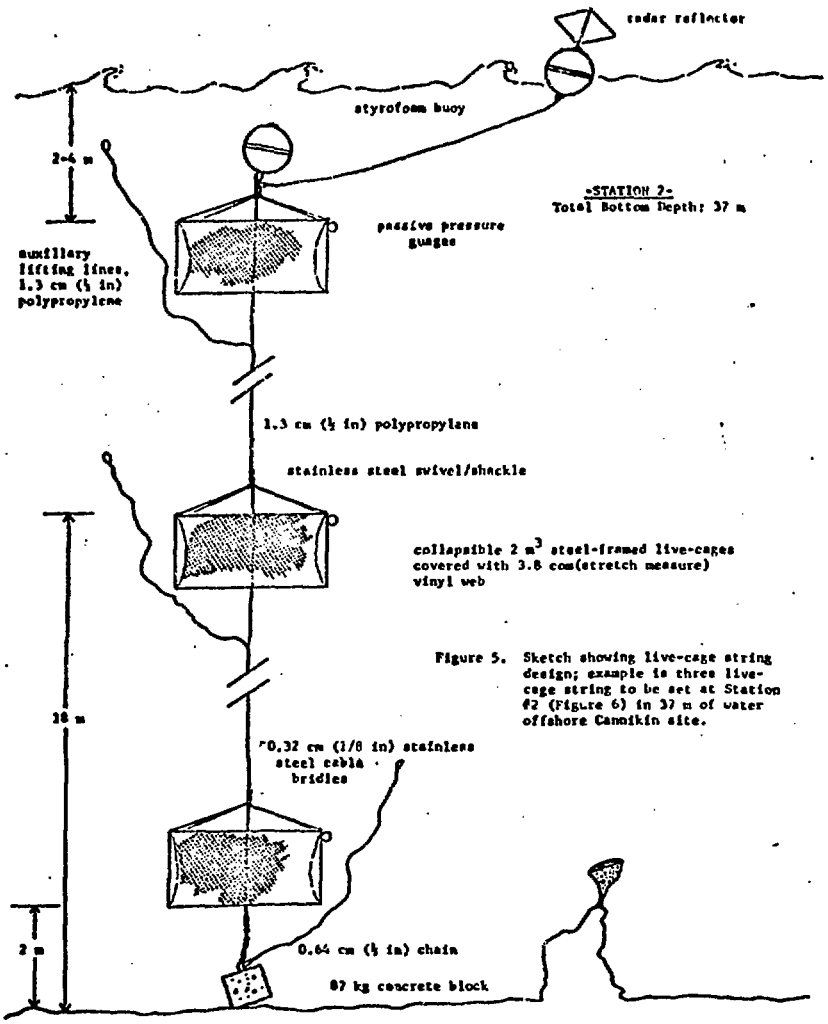

Figure 5. Sketch showing live-cage string design; example is three live-cage string to be set at Station #2 (Figure 6) in 37 m of water offshore Cannikin site.

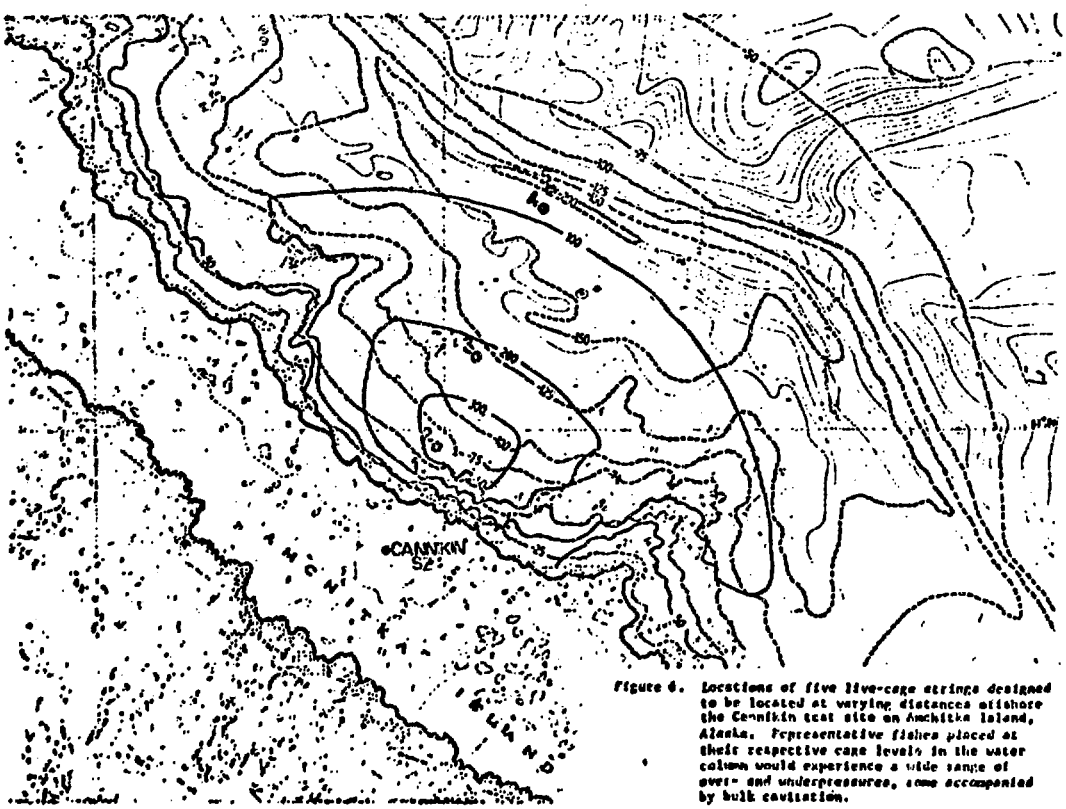

Figure 6. Locations of five live-cage strings designed to be located at varying distances offshore the Cannikin test site on Amchitka Island, Alaska. Representative fishes placed at their respective cage levels in the water column would experience a wide range of over- and underpressures, some accompanied by bulk cavitation.

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Paper Modifications Conf-730581--2

November 20, 1973

Dr. James B. Kirkwood
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Duxbury, Massachusetts 02332

Dear Dr. Kirkwood:

Enclosed is a copy of the letter sent to Dr. George Young, Naval Ordnance Laboratory, Silver Spring, Maryland which corrects our papers submitted for the NOL EEEE Conference proceedings in accordance with AEC and BCL comments as received by us 16 November.

We feel several points should be taken into consideration relative to the Battelle comments.

1. These papers were not originally to be prepared (only written abstracts) and it wasn't until after that we received word to prepare polished summaries of our presentations.

2. Papers were submitted to Dr. Merritt upon agreement with Dr. Merritt as the papers were a series (i.e. I, II, III) that would need to be reviewed as such. Also, as per clearance procedure, BCL was not supporting travel so the thoughts of Douthett's Oct. 25th letter, second paragraph, best explains our action.

3. We covered subjects and organisms not covered by the FRI contract because no one else responded or participated in the conference and, as the Navy requested an overall view of the test's impact on the environment, we felt qualified to include these aspects in our presentations.

As to specific comments about the papers.

Simenstad This paper

1. I think that the word "disregarded" is appropriate to the experiments by Wright (as presented in BMI-171-130) in relation to underpressure. The experiments were designed around overpressure only, the results discussed only overpressure effects, and the model's criteria was based only on pressure increases. Other than two confusing sentences on page 11 of BMI-171-130, no mention of the possible role of underpressure or the synergistic effect of the wave form is made.

a) Concerning the discussion of the live-cage experiments, the conference was designed to help defense contractors and the military evaluate the effects of their operations in the environment. Our approach, as well as our results, was of interest to this audience. In this light, I do not consider one paragraph in my paper and one in Mr. Isakson's as excessive elaboration.

Isakson

1. The bottom trawls compared for effects-evaluation included trawls from off Kirilof Bay-Cyril Cove. See Figure 2 and Appendix Table A-2 in BMI-171-150. Specific mention of Kiriloff Bay is made on pages 8, 9 ("area B"), 10, and 68 of FRI's final draft on our summary report (to become BMI-171-156) which may not have been seen yet by AEC reviewers.

2. The revised statement for #4 on page 5 attempts to say that a test such as Cannikin can kill thousands of marine fishes and these are "replaced" by immigration from adjacent and unaffected areas. The cumulative impact of numerous, successive tests would be to limit this method of recovery by reducing those areas that remain unaffected.

3. I regret not seeing Mr. Estes final report when this was prepared. I do not know what happened to the report sent on July 2. I received a copy from Mr. Estes after requesting one in September. My sources for these numbers were personal communications from Estes. Estes and Smith (1973) is now incorporated into the NOL paper.

We sincerely hope these changes and comments rectify any incongruities and irregularities in the papers as submitted to NOL earlier. Thank you for your considerate evaluations.

Charles Simenstad
Marine Biologist

John Isakson
Biologist

CS/JI:mm

Enclosure: letter to Dr. Young