DOOm-

DR 238

NVO-130

POST MORTEM FINDINGS IN SOME MARINE MAMMALS AND BIRDS FOLLOWING THE CANNIKIN TEST ON AMCHITKA ISLAND

U.S. PUBLIC HEALTH SERVICE
ARCTIC HEALTH RESEARCH CENTER
FAIRBANKS, ALASKA

JULY 1973

UNITED STATES ATOMIC ENERGY COMMISSION
NEVADA OPERATIONS OFFICE
Las Vegas, Nevada

MASTER

BLANK PAGE

Printed in the United States of America. Available from
National Technical Information Service
U.S. Department of Commerce
Springfield, Virginia 22151

Price: Printed copy $5.45; Microfiche $0.95

BLANK PAGE

NVO-130

POST MORTEM FINDINGS IN SOME MARINE MAMMALS AND BIRDS FOLLOWING THE CANNIKIN TEST ON AMCHITKA ISLAND

Robert L. Rausch

U.S. PUBLIC HEALTH SERVICE
ARCTIC HEALTH RESEARCH CENTER
FAIRBANKS, ALASKA

JULY 1973

Prepared for the

UNITED STATES ATOMIC ENERGY COMMISSION
NEVADA OPERATIONS OFFICE
Las Vegas, Nevada

TABLE OF CONTENTS

ABSTRACT

Animals fatally injured by effects of the Cannikin detonation on Amchitka Island were subjected to post mortem examination, and representative tissues were studied microscopically. From evidence so obtained, in combination with other information, it was concluded that three mechanisms were involved in producing the range of lesions observed: rock-falls; vertical acceleration; and combined effects of over- and underpressures.

Two sea otters, Enhydra lutris (Linnaeus), on the beach at the time of the detonation, were crushed by falling rocks. Eight birds (seven harlequin ducks, Histrionicus histrionicus (Linnaeus), and a pelagic cormorant, Phalacrocorax pelagicus Pallas) also sustained crushing injuries, evidently when exposed rocks upon which they were resting were violently displaced upward by vertical acceleration. Animals that were diving at the time of the detonation were exposed to a pressure-pulse followed by a fall in pressure to cavitation and a subsequent rise to the pre-existing ambient. In ten sea otters and in four harbor seals, Phoca vitulina(Linnaeus), lesions produced respectively by overpressure and underpressure were distinguished, although some of the disruptive changes in the lungs could not be specifically related to one of these factors. Fatal injuries in seven birds, representing six species, were attributed to effects of pressure-changes. In two sea otters, traumatic lesions of undetermined origin were superimposed upon those produced by pressure-changes.

Because of the scarcity of injured but still living mammals, it appears that the rate of mortality was high among those that were diving in the critical areas at the time of the detonation, but those at the surface probably were uninjured.

INTRODUCTION

After the Cannikin Test on Amchitka Island on 6 November 1971, it was possible to investigate the effects on marine animals of the pressure wave generated by an underground nuclear detonation. I was invited to be present in order to examine any that were injured or killed. A search along the beaches, beginning as soon as possible after the test on 6 November and continuing through 8 November, produced several marine mammals and birds that evidently had been killed by effects of the detonation. The present report describes post mortem findings in these animals and attempts to define the means by which the observed injuries were produced.

MATERIALS AND METHODS

Most of the animals were obtained by teams traversing the beaches on foot, beginning at 4:30 p.m. on 6 November, when only about a half-mile of beach on the north side of the island nearest the test-site could be surveyed by nightfall. A severely injured sea otter and several dead birds were found during that evening. Extensive searches along both sides of the island were made during the next two days (Kirkwood and Fuller, 1972). Most of the animals found by the respective crews were brought the following morning by helicopter to a central location, and thus the majority of specimens was received on 8 and 9 November. In all, twelve sea otters, four harbor seals, and sixteen birds representing seven species were in suitable condition for detailed examination. I departed Amchitka on

10 November, since it was considered that degenerative changes in the organs would obscure a judgement as to the cause of death in specimens found after that time.

Prompt examination was essential, in order to lessen the effects of degenerative changes that had begun soon after the death of the animals, notwithstanding the cool climate of the region. All animals were dissected on the day that they were received, but the amount of time that could be devoted to each was less than would have been desirable. The work was much facilitated by the assistance of other investigators, who kindly recorded the findings. Autopsies were done on the sea otters and birds in a small building made available by the Fisheries Research Institute, University of Washington. One seal that could not be retrieved by helicopter was dissected on the beach; the others, because of their large size, were examined outside on the ground. Most of the sea otters were in relatively good condition, having been immersed for a time in cold sea-water. Some of the specimens obtained at the last had been partially eaten by eagles, but the resulting damage was confined to the posterior part of the body. The two seals found last and examined on 9 November exhibited more advanced degeneration, since the enveloping layer of subcutaneous fat caused retention of body-heat. The birds found later than 6 November also showed degenerative changes, but these did not obscure the nature of the injuries sustained.

The total length of each mammal was recorded, and weights were taken of the sea otters. After an external examination, all animals were placed in dorsal recumbency and the ventral tissues were removed to expose the thoracic and abdominal viscera *in situ*. Gross findings were recorded, after which the organs were removed for detailed examination. The brain and spinal cord were the last to be examined. The processing of the animals was uniform, except that circumstances did not permit removal of the spinal cord from the seals. Selected skulls, including those from the seals, were taken to the Arctic Health Research Center in Fairbanks for cleaning and further study. Samples of tissue, the majority from macroscopically abnormal organs, were individually labeled as to origin and orientation and preserved in 10 per cent formalin solution. Selected tissues from ostensibly normal organs also were preserved. For comparisons, two intact, uninjured sea otters kindly provided by Mr. James A. Estes, University of Arizona, were later dissected at the laboratory. The blood vascular system of one was doubly injected with vinyl resin by Dr. F. H. Fay, to permit study of the vessels of the vertebral column.

Tissues were dehydrated and embedded by standard methods, sectioned at 0.005, 0.007, or 0.010 mm, and stained routinely in hematoxylin-eosin and in Mallory's anilin blue collagen stain. Special stains were applied to selected sections. The osteological material was cleaned by means of dermestid larvae and bleached in hydrogen peroxide.

RESULTS

The marine mammals obtained after the detonation had been in good to excellent condition at the time of death, as would be expected during autumn. Some general information concerning the sea otters is summarized in Table I. It might be noted that none of the abnormalities described below was observed in a series of thirty-one sea otters found moribund or dead of natural causes on Amchitka Island during 1949-52 (Rausch, 1953). One of the birds exhibited abnormalities of a chronic nature, and one had died of causes unrelated to the detonation (see below).

In a preliminary report of findings (Kirkwood and Fuller, 1972, p. 9), the injuries sustained by the animals were attributed to three causes: (1) rock-falls; (2) vertical acceleration; and (3) overpressure. The integration of the evidence from the post mortem examinations and from the study of tissue-sections has required modification of the concept of one of these mechanisms as well as different interpretation of the cause of death of certain animals. The three mechanisms as now recognized are (1) rock-falls; (2) vertical acceleration; and (3) the combination of overpressure and underpressure. The findings are presented according to these categories.

I. Animals injured by rock-falls

Two sea otters, Enhydra lutris (Linnaeus), examined on 8 November, had been crushed by rocks falling from the coastal cliffs at the time of the detonation.

Crushing of the skull and left side of the abdomen had caused the immediate death of the first animal. The thorax had not been directly

Table I. General information concerning sea otters examined at Amchitka Island.

Specimen	Sex	Total length (mm)	Weight (kg)	Condition	Remarks
1	♀	1250	19	Lactating.	Injured; alive when found.
2	♀	1180	21.8	--	Pressure-related injuries. Evidence of external trauma (subcutaneous edema and hemorrhage ventrally).
3	♀	--	11.3	Moderate subcutaneous fat.	Pressure-related injuries.
4	♀	1290	19+	Moderate subcutaneous fat. Partly eaten by eagles.	Pressure-related injuries.
5	♂	1165	17.2	Much subcutaneous fat.	Pressure-related injuries.
6	♂	1250	20.4	Little subcutaneous fat.	Pressure-related injuries.
7	♀	1220	18.1	Moderate subcutaneous fat.	Pressure-related injuries; fracture of skull.
8	♀	1270	20.9	Lactating.	Killed by rock-fall.

Table I. Continued:

Specimen	Sex	Total length (mm)	Weight (kg)	Condition	Remarks
9	♀	1225	18.6	Moderate subcutaneous fat.	Killed by rock-fall.
10	♀	1255	20.4	Lactating.	Pressure-related injuries.
11	♀	1280	17.2+	Moderate subcutaneous fat. Partly eaten by eagles.	Pressure-related injuries. Evidence of external trauma (injury to thorax externally; fracture of left zygoma).
12	♀	1310	22.2	Lactating. Moderate subcutaneous fat.	Pressure-related injuries.

injured. The left lung was moderately congested, probably as the result of post mortem hypostasis of blood; the right lung was essentially normal. The abdominal cavity contained much uncoagulated blood and large clots. The spleen had been ruptured, and the liver displaced entirely to the right side.

The second sea otter was immobilized by rocks that had fallen on the right rear leg. The animal was dead when found on 8 November, and the eyes were missing, presumably having been removed by scavenging birds, but it had survived for some time after the accident, having severely abraded the anterior margins of the pads of both fore-feet in its struggles to escape. Signs of trauma were limited to the muscles of the femur, the adjacent abdominal wall, and associated mammary tissue, all of which were hemorrhagic. The subcutaneous tissues over these areas were severely edematous. The abdominal organs exhibited no gross injuries, except for a perforation of the colon at a level about 75 mm anterior to the anus. The pleural cavities contained a small amount of blood-stained fluid, and the lungs appeared to be severely congested.

The left ramus of the mandible had a fresh fracture that began ventrally at the level of the anterior margin of the second molar and extended diagonally anterodorsad to the middle of the second premolar, of which the crown and posterior root were missing. Two beach-pebbles, about 3 and 4 mm in diameter, respectively, were found deeply embedded between the two surfaces of the fracture after the skull had been cleaned. These evidently had become impacted by the animal's chewing surrounding objects in its efforts to escape. The enamel crowns of the remaining teeth were uninjured.

An incidental finding was a cystic area, about 6 x 4 mm, in the right thyroid. Sections showed this to be a cystadenoma.

II. Animals injured by vertical acceleration

Injuries of the type that I now attribute to vertical acceleration were limited to birds. Those affected presumably had been resting on offshore rocks in shallow waters near the test-site at the time of the detonation. All of the specimens exhibiting the characteristic pattern of lesions were found within a restricted area along the north side of the island.

The specimens considered here include seven harlequin ducks, *Histrionicus histrionicus* (Linnaeus), and one pelagic cormorant, *Phalacrocorax pelagicus* Pallas.

Evidence of severe trauma was seen in both the soft tissues and the skeleton of these birds. The external examination revealed injury to the femoral muscles and to the adjacent body-wall in all cases; other findings included fractures of the leg-bones, fracture and separation of the vertebral column, and hemorrhage from the auditory canals. The nature of the injuries suggested that the legs had been driven dorsad by a vertical force.

The tissues of the body-wall around the proximal end of the femur and the medial femoral muscles were hemorrhagic bilaterally. In two ducks, the body-wall just anterior to the femur had been perforated bilaterally, with seepage of fluids from the abdominal cavity. Leg-bones of two ducks had been broken; one had fractures of both tibiae and comminuted fractures of the left femur, and both femora had been broken in the other. As far as could be determined, the fractures of the long-bones were essentially longitudinal in orientation.

The vertebral column had been disarticulated in five ducks, with the separations occurring at the level of the second to the fourth thoracic vertebrae. In one of the most severely injured, the skin over the separated vertebrae had been perforated, with drainage of fluids from the thoracic cavity. Ribs had been broken unilaterally in one duck and in the cormorant.

The viscera of all of these birds had been severely damaged, particularly those situated anteriorly and dorsally. The thoracic contents, excepting the heart, had disintegrated to the extent that pulmonary tissue was unrecognizable. The kidneys also were more or less macerated, but the abdominal organs otherwise were relatively intact. Some degree of laceration of the liver had occurred in two ducks and in the cormorant. In one duck, the proventriculus had ruptured, releasing its contents into the body-cavity; in another, it had separated at the point of junction with the ventriculus. Both of these birds also had perforations of the intestine. The abdominal cavity contained much blood and large clots in all cases.

All of the specimens showed some degree of injury to the superficial or deep tissues of the head and neck. In one of the more severely affected ducks, the tissues covering the cervical vertebrae ventrally were hemorrhagic from the level of the pharynx to the thorax; the trachea was partially crushed, and had been severed at a point about one-third of its length anterior to the point of entry into the thorax. Laceration of the spinal cord where it passed through the foramen magnum had occurred in one case, and subarachnoid hemorrhages were present dorsally on the cerebrum and cerebellum. This bird also had a hemorrhagic area about 50 mm long superficially in the right pectoral muscle. Subarachnoid hemorrhages, in one case involving the optic lobes bilaterally, were present on the brain of three other ducks. Flow of blood from one or both auditory canals was noted in four. Macroscopically, there was no evidence of fractures in the basal portion of the skull in these birds.

III. Animals injured by combined overpressure and underpressure

The injury of one sea otter, and the deaths of nine sea otters, four harbor seals, and seven birds were attributed to the effects of pressure while the animals were submerged.

Sea otters.

The traumatic lesions observed in the sea otters in this category were uniform in pattern and, in the animals found dead, differed within rather narrow limits in degree of severity. The injury of the sea otter found alive was initially attributed to vertical acceleration, but the integrated evidence strongly supports its inclusion here. Findings in this animal are considered separately.

The injured sea otter, discovered late on the evening of 6 November by Mr. James A. Estes and myself, as we returned along the north beach, was lying among large boulders well above water-line, headed away from the sea. The animal made feeble efforts to defend itself by biting, but was no longer capable of locomotion. It was carried back to a vehicle and taken to the laboratory, where the examination was begun at about 9:00 p.m., or approximately ten hours after the detonation had occurred.

External signs of trauma were absent. The thoracic walls were markedly distended and rigid, and breathing was of abdominal type. There were fifteen respirations per minute, with severe inspiratory and expiratory dyspnea. Inspiration was through the open mouth, accompanied by a gasping sound; expiration was prolonged, with forcible expulsion of air through the mouth.

The animal was killed by means of intracardiac injection of potassium chloride solution. When the needle was inserted, air escaped into the subcutaneous tissues. At this time, the sea otter expelled a large quantity of blood-stained urine, so dark in color as to have the appearance of pure blood.

The lungs appeared to be collapsed bilaterally. The pulmonary pleura was intact, with scattered, subpleural areas of hemorrhage. The more severely affected left lung (consisting of two lobes in the sea otter) was relatively uniform dorsally and ventrally; the ventral surface of the right lung (consisting of three lobes) was more nearly normal in appearance. The left posterior lobe had a few islets of inflated, apparently normal tissue along its medial margin. Each pleural cavity contained 10-15 ml of blood-stained fluid. The trachea throughout exhibited severe congestion of the submucosal tissues between the rings. Blood-clots were present anteriorly in the nasal cavity.

The eyes and auditory canals appeared to be normal. The tympanic membranes were intact. The stomach and duodenum contained frothy, semifluid material with many gas-bubbles. Discrete, hemorrhagic areas were present anterodorsally in the wall of the urinary bladder, one medial and one on either side; a smaller hemorrhage was found in the wall on the right side near the neck. The lumen contained large blood-clots. The kidneys and ureters were macroscopically normal. The liver exhibited only numerous pale, round spots up to 4 mm in diameter beneath the capsule on the anterior (diaphragmatic) surface. The wall of the gall bladder was greatly thickened as a result of chronic cholecystitis caused by a trematode that occurs commonly in sea otters (Rausch, 1953). No abnormalities were observed in other organs.

Samples of tissue from four areas in the lungs of this animal were examined microscopically. These were similar in that each showed considerable variation in degree of injury from lobule to lobule.

In the sections from the apex of the left anterior lobe, large areas of alveoli were collapsed or partly collapsed. Some of these areas were severely hemorrhagic and contained many round cavities or spaces that resembled bubbles and presumably had contained gas. That some might have been fat cannot be excluded, since staining for fat was not done. Such cavities were a prominent feature in sections of pulmonary tissue from the various injured animals (see below). Some represented bullous emphysematous spaces formed as a result of traumatic disruption of the tissue, but most were spherical cavities that appeared clearly to represent gas that had been entrapped upon coagulation of blood or hemorrhagic fluid in which it had been suspended. Hemorrhage had occurred also into some bronchi and bronchioles (in the sea otter, cartilaginous elements are present in bronchioles of a diameter down to at least 0.280 mm). The interlobular, subpleural, and peribronchial connective tissue was emphysematous. No hemorrhage had occurred into these interstitial spaces. In the more moderately affected areas, the tissue was congested, frequently with slight hemorrhage into the alveoli. Findings were similar in sections from the medial portion of the left posterior lobe.

In sections from the anterior margin of the right anterior lobe, gas-bubbles were clearly evident in hemorrhagic areas. Focally, groups of round, interconnected cavities apparently filled with air had been produced by rupture and distension of adjacent alveoli. These differed markedly in appearance from the irregular spaces in areas of pulmonary emphysema. Interstitial emphysema had produced wide spaces around bronchi and large veins. Some lobules, or parts of lobules, had been only slightly affected. In sections from the medial portion of the same lobe, atelectatic areas were observed to be more extensive, especially around larger bronchi. Where the alveoli were not completely collapsed, many gas-bubbles often were present. Some bronchi contained blood, usually as a thin layer covering the mucosal surface. Interstitial emphysema was marked, with interlobular septa attaining widths up to 0.480 mm (Fig. 1). Other areas of parenchyma were not severely affected, but usually there was some degree of collapse of the alveoli or emphysema.

The kidneys were severely congested, especially in the cortical zone. Free erythrocytes were occasionally observed in Bowman's space and in the lumina of convoluted tubules. The pale, subcapsular foci on the hepatic surface evidently corresponded to superficial aggregations of vacuolated hepatic cells. Staining to demonstrate fat was not attempted. No microscopic abnormalities were recognized in tissue from other organs.

In the sea otters found dead, the thoracic organs and central nervous system had been severely affected. The observed lesions were relatively uniform in the nine specimens.

The head and associated structures. The examination of the structures of the head was difficult under field conditions, and only the few specimens taken to the laboratory were adequately studied. Fracture of the orbital walls was easily overlocked unless the skull had been cleaned. In the field, injury to the ears was determined from examination of the external auditory meatus and of the tympanic cavity after removal of the roof of the tympanic bulla. Severe injury with hemorrhage in the middle ear had occurred in five animals. The tympanic membranes were considered to be intact in four.

Injury to the contents of the ocular globe was noted in two sea otters. Hemorrhage into the anterior chamber of the left eye had occurred in both, and the right eye of one had been ruptured, an injury that might have been caused by scavenging birds. Fracture of the posterior wall of the orbit was discovered in the cleaned skulls of two animals, in neither of which was there any external evidence of injury to the eyes. In one subadult animal, a round, depressed fracture measuring 20 x 15 mm was present in the right orbital wall directly ventral to the supraorbital process. Comparison of the skull with that of a young sea otter in which synostosis had not yet occurred, showed that the fracture was almost wholly in the frontal bone, with slight involvement of the posterior end of the lacrimal. In an old adult, a hole measuring 19 x 14 mm was present in the same location, and the underlying maxilloturbinates had been shattered (Fig. 2). Fractures of the orbital walls probably were overlooked in some of the other specimens.

Similar injuries had occurred in two animals that presumably had been killed on 6 November but found some time later by University of Arizona personnel. The orbital walls had been fractured bilaterally in a subadult male found on the beach on 26 November. In the cleaned skull, the openings measured 26 x 19 and 19 x 15 mm on the left and right sides, respectively. The upper part of the maxilla had been broken away on the left, but the location of the openings was the same as in the other two described above. In this animal also, the maxilloturbinates and nasal septum had been shattered, and the posterior part of the palatine bone had been broken away. Similar damage to the palatine bone, with fracturing of the maxilloturbinates posteriorly, was observed in a second specimen, found on 7 December. The orbital walls of this animal were intact. The structure of the skull of the sea otter is such that damage of this nature could hardly occur fortuitously after death.

Other external injuries to the head were noted in two animals. In one, a fracture extended from the posterior end of the sagittal crest ventrad across the occipital to the dorsal margin of the foramen magnum. Dorsally, a depressed fragment of the right parietal, 30 x 10 mm, extended from the lambdoidal crest anteriad along the sagittal crest. In the second, the parietal muscles immediately posterior to the supraorbital process were hemorrhagic over an area of about 25 x 25 mm. The maxillary process of the left zygoma had been fractured.

The central nervous system. Lesions observed in the brain and spinal cord were uniform in pattern. Only one animal, the least severely injured of the series studied, exhibited no gross lesions in these organs. The spinal canal of one animal was not opened.

Subarachnoid hemorrhage was a frequent finding in the brain. Hemorrhage attributable to external trauma was observed only once, in the animal having a depressed fracture of the right parietal. Findings in the brains of the sea otters are listed briefly below:

(1) Blood-stained fluid in the lateral ventricles. (2) Epidural hemorrhage on the dorsal surface of the right cerebral hemisphere posteriorly, just lateral to the longitudinal fissure, and corresponding in location to a depressed fracture of the parietal bone. (3) Blood-stained fluid in the lateral ventricles; congestion of the cerebrum. (4) Blood-stained fluid in the lateral ventricles; subarachnoid hemorrhage anteriorly on the dorsal surface of the cerebrum. (5) Hemorrhage into the longitudinal fissure of the cerebrum; blood-clots in the lateral ventricles; hemorrhage ventral to the brain stem and right lobe of the cerebellum; subarachnoid hemorrhage anterodorsally on the left lobe of the cerebellum. (6) Congestion of the cerebellum; hemorrhage ventral to the brain stem; blood-stained fluid in the lateral ventricles (the spinal cord of this animal was not examined). (7) Brain appeared swollen, with massive subarachnoid hemorrhage dorsally over both cerebral hemispheres; blood-clots in the lateral ventricles; subarachnoid hemorrhage dorsally over the cerebellum.

The vascular lesions in the cerebrum varied in extent, but were essentially superficial. No evidence of ruptured vessels in the deeper tissue was observed when slices of formalin-fixed brain were examined under the dissecting microscope. Vessels lying in the sulci also were intact. Findings in sections ranged from congestion of the subarachnoid veins to extensive subarachnoid hemorrhage. The capillaries and larger vessels of the cerebral tissue were usually congested, and focal hemorrhages involving both arteries and veins were occasionally observed peripherally.

The choroid plexuses of the lateral ventricles were usually congested, but rupture and hemorrhage had occurred in one specimen.

Vascular lesions in the cerebellum tended to be more extensive. In areas where subarachnoid hemorrhage had occurred, the vessels in the interfolial sulci were ruptured, with consequent distension of the sulci with blood. In sections of the cerebellum in which the most extensive hemorrhage had occurred dorsally, scattered diffuse hemorrhages were noted in the granular and molecular layers, and less frequently deeper in the white matter (Fig. 3). In one restricted area in the white matter, perivascular hemorrhage had occurred, usually into perivascular spaces. Congestion of the cerebellum was not marked. Focal hemorrhages in the granular layer were present in another animal.

The spinal cord had been injured in seven of the eight animals appropriately examined. The severity of the other lesions observed in the ninth animal indicated that its spinal cord probably had been injured as well. In all seven sea otters there was subdural hemorrhage of the spinal cord, but in no case was there any indication of fracture or dislocation of the vertebrae. The thoracic portion of the cord was consistently affected, with variation in the extent of injury posteriad. In three animals, such hemorrhage was present throughout the thorax, from about the level of the seventh cervical vertebra posteriad. In the remainder, subdural hemorrhage extended farther posteriad, to the level of the third lumbar vertebra or beyond, as in one case where it extended to the level of the sacrum. The cervical spinal cord was abnormal in two cases: epidural hemorrhage had occurred in one of the most severely injured animals, and hemorrhagic fluid was present in the spinal canal of the second.

The injured spinal cords had a tense, smooth dura, through which the underlying blood appeared black. Small areas free of hemorrhage sometimes imparted a mottled appearance. In less severely hemorrhagic areas, it was apparent that both the ventral venous sinus and the dorsal subarachnoid vessels had ruptured. Transection of formalin-fixed specimens showed the spinal cord to be surrounded by an asymmetrical layer of blood ranging up to 2 mm in thickness (Fig. 4), the uneven distribution of which beneath the dura frequently had caused distortion of the cord. Hematomyelia was an occasional finding, with single or multiple hemorrhages into the white matter. Tissue-sections also revealed focal hemorrhages in the gray matter, usually along the columns adjacent to the white matter. Congestion of the gray matter was characteristic.

The respiratory system. Escape of blood from the nose and mouth was noted in five animals. In all cases, massive pulmonary hemorrhage had occurred. The quantity of coagulated blood in the lungs was such that these organs tended to retain their size and normal anatomical relationships when the pleural cavities were opened. This condition facilitated examination of the lungs *in situ*. The significance of lung-weight as an indicator of severity of injury in animals exposed to air-blast has been defined by Richmond and co-workers (White and

Richmond, 1959; Richmond et al., 1968). The lungs of the marine mammals at Amchitka were not weighed, but a marked increase in weight of these organs was clearly evident in all of the animals found dead.

The trachea usually contained blood or stomach-contents, but rarely showed macroscopic lesions. The tracheal mucosa was congested in one case; in another animal, subcutaneous tissues ventral to the larynx and upper trachea were hemorrhagic. Within the thorax, subpleural hemorrhage sometimes occurred around the trachea (see below).

The lungs of all the animals had been injured bilaterally, although the pattern of the lesions varied. Injuries to the anterior lobes tended to be least severe; in three animals, these were relatively normal macroscopically, and in two others the apical portions of these lobes had been little affected. In another specimen, a few normal-appearing islets of inflated alveoli protruded on the lateral surface of the otherwise hemorrhagic lobes; these measured up to 50 x 35 mm and 48 x 30 mm on the left and right lobes, respectively. The lateral or medial surfaces of one or more lobes sometimes appeared to be relatively normal, but the underlying parenchyma was hemorrhagic. Superficial pulmonary lesions of other types were occasionally observed. In one animal, the left posterior lobe having multiple perforations also exhibited numerous gas-filled cavities bulging beneath the pulmonary pleura. On the anterior margin of the right posterior lobe were two inflated bullae, the larger of which measured 45 x 25 mm; the floor of each was formed by a layer of coagulated blood. Subpleural, gas-filled cavities were present on the lateral surface of the left posterior lobe in another animal. In a third specimen, hemorrhage had occurred in the tissues around the two left bronchi where they entered the respective lobes.

In the most severely affected lobes, the pulmonary parenchyma was very dark, and solid in consistency; samples removed for preservation often sank in formalin solution. In some animals, the severely hemorrhagic areas contained macroscopic cavities filled with gas. The bronchi and larger bronchioles in all cases contained blood and, in five animals, ingesta from the stomach as well. Hemothorax was observed in seven animals, with the quantity of free blood in each pleural cavity ranging from about 40 to 150 ml. Large clots, up to 100 ml in volume, were usually present, either free or extending from perforations in the pulmonary pleura. Rupture through the pulmonary pleura of one or more lobes had occurred in the lungs of five animals. The perforations were on the lateral surfaces, and ranged from 3 to 40 mm in greatest diameter. None was observed in the anterior lobes. Single perforations of one or more lobes were present in the lungs of four animals: bilaterally in the posterior lobes; in the right cardiac lobe at the junction with the anterior lobe dorsally, and in the right posterior lobe; in the right posterior lobe near the junction with the cardiac lobe; and in the left posterior lobe. Four perforations were present on the lateral surface of the posterior lobe in the fifth specimen. In another animal, a large fragment of pulmonary tissue had been detached from the apex of the right anterior lobe.

When cut surfaces of preserved samples of pulmonary tissue were examined under the dissecting microscope, areas of severe hemorrhage appeared black, in contrast to the lighter color of adjacent hemorrhagic tissue that had not been so extensively disrupted. These tissues contained scattered, spherical cavities up to 4 mm in diameter, evidently representing entrapped gas-bubbles. After preservation, such tissues had a liver-like consistency, and were not compressible.

Microscopically, the lesions in the dead animals differed from those observed in the animal found alive only in their greater extent and greater severity. Disruption of tissues and extensive hemorrhage were characteristic of the cardiac and posterior lobes. In the more moderately affected areas, the alveoli were uniformly blood-filled; in other areas, the alveolar structure could not be discerned. Bronchi and bronchioles contained blood, and many had been ruptured. Round cavities, representing gas-bubbles, were present in all of the sections examined; where there was extensive hemorrhage, these attained diameters up to 4 mm and sometimes imparted a sponge-like appearance to the tissue. Some lobules contained partly collapsed alveoli, but areas of completely atelectatic alveoli were uncommon. Pulmonary emphysema was a frequent finding. All sections showed evidence of interstitial emphysema, with distension of interlobular septa to widths up to 0.880 mm. Such septa usually appeared as spaces in which remnants of connective tissue were sparse or absent. Occasionally a very loose reticulum remained, in which small numbers of erythrocytes were sometimes present. In the vicinity of ruptures through the pulmonary pleura, the interlobular spaces were filled with blood. Representative lesions in the lungs of these sea otters are shown in Figs. 5-9.

In one of the more severely injured animals, sections of tissue taken from the lateral surface of the right posterior lobe near a 10 mm perforation showed evidence of chronic interstitial inflammation, with wide septa of dense connective tissue marginally infiltrated by lymphocytes. Plasma cells were occasionally observed. The alveoli in this area were hemorrhagic and largely collapsed, but interstitial emphysema was less general.

On 21 November, the body of a sea otter was recovered by a trawl off Amchitka in 30 fathoms of water. The lungs were fixed in formalin by Mr. James A. Estes, who sent the material to me for further study. The tissues stained poorly, but the gross changes in the lungs (extensive hemorrhage; interstitial emphysema), as determined from sections, appeared to be identical with those observed in pulmonary sections from the animals considered above.

The heart and associated structures. Cardiac tissues were preserved from four of the nine animals. In three, the ventral surface of the heart exhibited superficial, subepicardial lesions as follows: ecchymotic and petechial hemorrhages anteriorly on the left ventricle near the coronary groove; an irregular hemorrhagic streak up to 7 mm in width extending over two-thirds of the ventricular length; and a 2 mm hemorrhage at the interventricular sulcus. In two cases, the lesions had been produced by hemorrhage into the subepicardial adipose tissue; the myocardium was also congested, and scattered focal hemorrhages were present. Sections of the third specimen, having only a small superficial lesion, revealed extensive hemorrhage deep in the myocardium of the thinnest part of the ventricular wall, with marked separation of the myocardial fibers (Fig. 10). In the specimen without macroscopic lesions, the myocardium was congested and very few focal hemorrhages could be found. Examination of the endocardium and other structures revealed no other macroscopic lesions, nor were any abnormalities noted in the large vessels.

When the thoracic organs of one sea otter were examined in situ, an "empty" space of considerable extent was observed in a pulmonary vein medially. Although its significance was not appreciated at the time, this was the only macroscopic evidence of possible air-embolism. No air-emboli were recognized in the coronary vessels.

Other thoracic organs. In these animals, other structures situated dorsally in the thorax usually exhibited some degree of injury. Lesions were sometimes present in the mediastinum and in the pulmonary ligaments, and hemorrhage into the subpleural fascia ventral and lateral to the vertebral column had occurred in most cases. Since summarization of the observations is difficult, findings in the individual animals are briefly described:

(1) Retroperitoneal hemorrhage dorsal to the aorta throughout the thorax, and bilaterally extending dorsad lateral to the vertebral bodies; also with hemorrhage dorsally in the mediastinal pleura just anterior to the diaphragm. (2) The mediastinum (not fenestrated in the sea otter)

had been perforated posterior to the pulmonary ligaments, with herniation of the posterior lobe of the right lung into the left pleural cavity. (3) Severe hemorrhage and clot-formation dorsal to the aorta throughout the thorax, extending posteriad to the level of the origin of the psoas muscles; bilaterally with hemorrhage and clot-formation lateral to the vertebral bodies. (4) Rupture of the costal artery anterior to the sixth thoracic vertebra, with a hemorrhagic area 60 mm in length. (5) Hemorrhage and attached clots in the right pulmonary ligament and dorsal to the trachea anteriorly, and with clot-formation dorsal to the aorta opposite the seventh and eighth intercostal spaces; moderate hemorrhage lateral to the vertebral bodies throughout the posterior two-thirds of the thorax bilaterally. (6) Moderate hemorrhage, more severe on the left, lateral to the vertebral bodies throughout the thorax. (7) Slight hemorrhage along the vertebral bodies bilaterally, with some clot-formation; pulmonary ligaments hemorrhagic posteriorly. (8) Anteriorly with hemorrhage in the left pulmonary ligament and extending dorsad along the lateral surface of the trachea on the right; rupture of the intercostal veins and arteries with massive retroperitoneal accumulation of coagulated blood from the level of the sixth thoracic vertebra posteriad on the left. None of these lesions was observed in the ninth animal.

The thoracic walls and diaphragm. The thoracic walls infrequently showed macroscopic signs of trauma. In one of the most severely injured sea otters, having ruptured intercostal vessels, the sixth through ninth ribs on the right had separated at the costochondral junctions, and the surrounding tissues were hemorrhagic. The muscles of the eighth and ninth intercostal spaces dorsally had been nearly perforated immediately lateral to the vertebrae. Externally, in the corresponding area (over the seventh to ninth ribs), tissues ventral to the latissimus dorsi were hemorrhagic. The right side of the diaphragm also had multiple perforations.

In a second animal, subpleural hemorrhage had occurred laterally in the left thoracic wall, along the posterior margins of the fifth to twelfth ribs, increasing in extent posteriad. Sections revealed subpleural and intramuscular hemorrhage, with involvement of only the internal intercostals. On the same side, the thoracic surface of the diaphragm had subpleural hemorrhages extending dorsoventrally along the muscle fibers. Similar lesions were observed on the right thoracic surface of the diaphragm of a third animal.

An unusual combination of lesions was observed in a sea otter found on the south side of the island. In addition to severe intrathoracic injuries, including separation of a fragment from the apex of the right lung (see above), there was evidence of external trauma. The subcutaneous tissues ventrally had been consumed by amphipods (some of which were still present), indicating a longer period of immersion in the sea. Scattered, discrete blood-clots were present over the ventral surface of the abdomen and lower thorax. The subcutaneous tissues were edematous bilaterally along the abdomen, with greater accumulation of fluid on the left. The ventral subcutaneous tissues presumably also had been edematous. Hemorrhage had occurred beneath the pectoral muscles just lateral to the sternum.

The injuries suffered would seem to have been sufficiently severe to have been immediately fatal.

The abdominal organs. Some of the sea otters had been partly eaten by scavenging eagles, which in all cases had opened the abdomen posteriorly. The intestine and part of the right kidney were missing in one; the intestine, both kidneys, and the posterior parts of the liver had been removed from another; the urinary bladder was missing, and the intestine had been mutilated and was largely autolyzed in a third. With these exceptions, all of the abdominal organs were examined.

Some degree of injury to the liver had occurred in eight of the nine animals. The lesions were largely restricted to the anterior (diaphragmatic) surfaces of the four large lobes. Following the nomenclature of Urmanov (1971), these are designated the right and left diaphragmatic and the right and left visceral lobes. In the sea otter, the liver is almost entirely enclosed within the thorax, with only the posteromedial margins of the diaphragmatic lobes extending beyond the costal arches into the subcostal angle. Because of the variation in lesions observed, findings in the individual animals are briefly described.

(1) The surfaces of the right diaphragmatic lobe and of the left diaphragmatic and visceral lobes were lacerated, with fragmentation or maceration of the surrounding parenchyma. Hemorrhage had occurred into the abdominal cavity. (2) The left diaphragmatic lobe had a superficial abraded area just lateral to the midline. The intestine and other

viscera were coated with blood. (3) The left diaphragmatic and visceral lobes had multiple tears and abrasions; the injured area of the diaphragmatic lobe was covered by a thin, adherent clot. The abdominal cavity contained much free and coagulated blood. (4) The surfaces of the diaphragmatic lobes appeared to be severely congested. (5) Both diaphragmatic lobes had been lacerated or ruptured, with maceration of the parenchyma; an adherent clot covered the exposed parenchyma of the right lobe. The abdominal cavity contained free blood. (6) The right diaphragmatic lobe exhibited a subcapsular hemorrhage measuring 70 x 50 mm. (7) The surfaces of the diaphragmatic lobes had areas of apparent congestion, with scattered petechial hemorrhages. The anterior surface of the right visceral lobe had a subcapsular hemorrhage measuring 30 x 15 mm, and a similar area of smaller size was present on the medial surface of the quadrate lobe. (8) The left diaphragmatic and visceral lobes exhibited extensive subcapsular hemorrhage. The posterior portion of the liver was missing in this animal. No macroscopic lesions were noted in the liver of the ninth animal.

Macroscopic examination of the affected lobes showed that the lesions were superficial in all cases, even when extensive laceration or crushing had occurred. Sections from areas that appeared to be congested revealed diffuse hemorrhage superficially in the parenchyma, but the capsule and hepatic cells immediately below were unaffected. Where petechial hemorrhages had occurred, sometimes causing a slight elevation beneath the capsule, the accumulations of blood were separated from the capsule by a thin layer of intact hepatic cells.

Other abdominal organs usually were normal macroscopically. Perforation of the stomach or intestine was never observed. The gastric mucosa appeared to be congested in one specimen. Blood found in the stomach of two animals presumably had been swallowed. In one case, severely congested or possibly hemorrhagic areas were present in the psoas muscles bilaterally.

Harbor seals.

Four harbor seals, *Phoca vitulina* Linnaeus, were found dead. These animals, three females and a male, were adults; their body-lengths were 1210, 1520, 1700, and 1910 mm, respectively. Weights could not be obtained. The spinal canal was not opened in these animals, and tissues were not preserved from the two in which post mortem degeneration was rather advanced. The heads were further examined and cleaned at the laboratory.

The head and associated structures. Hemorrhage into the anterior chamber had occurred in the left eye of one animal, and bilaterally in a second. In another, blood was draining from the mouth and ears.

The posterior walls of the orbit had been fractured bilaterally in the four specimens (Fig. 11). In the least severely injured, a female, the anterior and ventral margins of the orbital part of the frontal bone as well as the adjacent orbital part of the maxilla had been fractured bilaterally (*Phoca* has no distinct lacrimal bone). That part of the maxilla covering the ventral turbinates anteriorly had been fractured bilaterally just anterior to the base of the zygomatic process. The fractures within the orbit as well as of the maxilla were depressed, with most of the fragments remaining _in situ_. Findings in the second female were similar, but the maxilla had been fractured anteriorly only on the left. The injury was more extensive in the remaining two, in which the fractures of the orbital wall also involved the perpendicular part of the palatine bone bilaterally. Some of the bone-fragments had been detached, with the result that the cleaned skulls exhibited large openings in the orbital walls. In one, the posterior portions of the ventral turbinates had been exposed, with some fracturing; in the second, the ventral turbinates on the left had been shattered and almost completely detached. In these two specimens, no fractures had occurred in the maxillae anteriorly.

The middle ear was examined on the left side only, following removal of the roof of the tympanic bulla. The tympanic membrane had been ruptured in all cases. Hemorrhage into the tympanic cavity had occurred in two specimens, with displacement of the auditory ossicles in one. Although the tympanic cavity contained no blood, the auditory ossicles had been dislodged in the male. In the third female, the most severely injured of the seals, the promontory of the pyramid had been shattered on the left side, and the petrosal part of the temporal bone had been broken away bilaterally and displaced dorsad. The resulting openings in the floor of the cranium measured about 30 mm in transverse diameter and about 18 mm in length.

The brain. Subarachnoid hemorrhage of limited extent occurred in three animals: on the dorsal surface of the cerebellum in two, and ventral to the cerebellum in the third. The displacement dorsad of the petrosal bones had caused severe hemorrhage ventrally in the brain of the one specimen. Because removal of the brains of these animals was delayed, no tissues were preserved.

The respiratory system. As in the sea otters, the lungs of the seals had been severely injured bilaterally. The lungs of the harbor seal are not lobed, and appear to be identical. Each is separated into approximately equal anterior and posterior segments by a shallow fissure (Sokolov et al., 1968), but this was not evident in the distended lungs of the specimens examined. A pair of principal bronchi, of which the posterior is longer, arises from the trachea on each side.

In the first animal, found on the morning of 7 November, the lungs were distended and very dark in color, with a somewhat mottled appearance imparted by irregularity of the subpleural hemorrhage. The lungs were more hemorrhagic ventrally, especially in the apical and lateromedial areas. Gas-bubbles in the interlobular septa were visible through the pleura. The transected parenchyma was severely hemorrhagic, with large areas that appeared to be atelectatic. The bronchi and bronchioles contained blood. The pulmonary pleura had been torn around the right posterior bronchus at its point of entry into the lung. The second animal, the most severely injured, was examined on 8 November. Blood was draining from the mouth, and the thorax was filled with uncoagulated blood that had escaped following bilateral rupture of the lungs. The lungs were totally hemorrhagic, with the posterior margins appearing somewhat less severely injured. On the lateral surface of the left lung, posterior to the middle, was a transverse laceration about 70 mm long, within which a ruptured bronchus was clearly discernible. On the right, a round perforation about 20 mm in diameter was present near the middle in the posterior third of the lateral surface. The trachea contained blood, and exhibited a hemorrhagic area in the dorsal wall at the level of the origin of the anterior bronchi.

The two animals examined on 9 November were in relatively poor condition for study. In the first, a large male, the lungs were hemorrhagic, but no unusual lesions were observed. The mouth contained a large blood-clot, indicating escape of blood via the trachea. When the second animal was opened ventrally by means of a median longitudinal incision, entrapped air-bubbles were found under the subcutaneous adipose layer in tissues ventral to the larynx. This condition was evidently interstitial subcutaneous emphysema produced by extension of air along the cervical fascias from the anterior mediastinum. The lungs were hemorrhagic, with the posterior portions most severely injured. Gas-bubbles were abundant in the interlobular septa. The bronchi and bronchioles contained blood.

Freshly cut surfaces of formalin-fixed pulmonary tissues from the first two animals were examined under low magnification, and as in the sea otters, the pattern of the lesions varied from lobule to lobule. In the first specimen, the interlobular septa were much distended, and large, spherical cavities were present in the interlobular connective tissue. In the severely injured animal, the tissue was liver-like and very firm, and when transected had the appearance of a homogeneous matrix within which were many spherical cavities ranging up to 2 mm in diameter. Such cavities had a smooth, uniform lining apparently consisting of coagulated blood. Ruptured bronchi or bronchioles were noted in the vicinity of a perforation of the pleura.

Microscopically, the findings in the lungs of the seals were similar to those in the lungs of the sea otters. The range in the severity of lesions in different parts of the organs was comparable. In Phoca, as compared with the sea otter, the interlobular septa are more strongly defined, glandular tissue in the bronchi and bronchioles is more abundant, and cartilaginous elements are more extensive.

The sections from the severely injured apical region of the right lung of the first seal revealed extensive accumulations of blood, with only bronchioles remaining distinguishable. Large gas-bubbles were present in the coagulated blood. Tissues excised from the lateral surface near the middle of the right lung exhibited pulmonary emphysema in less hemorrhagic areas. Gas-bubbles were numerous in coagulated blood, and single alveoli distended with air were scattered among blood-filled alveoli in some lobules. The interlobular septa were usually distended with blood, but little or none was present in the lumina of the bronchioles. Tissue from the apical region of the left lung was less severely affected. The lobules were distinct, with most of the alveoli filled with blood. Gas-bubbles were numerous, and the interlobular septa were emphysematous in some areas. Findings in the lungs of the second seal were similar. However, sections through the ruptured area of the left lung revealed extensive disruption of tissues and severe hemorrhage. Representative areas from the lungs of the two seals are shown in Figs. 12-15.

Possible tissue-emboli were identified in veins in the lungs of two seals. One (Fig. 16), nearly filling the lumen of the vein, contained strands of fibrin, but the identity and origin of the tissue otherwise could not be determined. In the second case, three small, discrete aggregations of cells were present, embedded among erythrocytes (Fig. 17). The cells most nearly resembled those of the alveolar walls, but their identity could not be clearly established. Neither was considered to be an artefact. With the extensive disruption of the lungs of these animals, the occurrence of emboli derived from pulmonary tissues would not seem remarkable.

The heart. Macroscopic lesions of the heart were recognized only in the most severely injured of the seals. Along the distal margin of the right auricle were several thin-walled, vesicle-like structures, which inflated when pressure was exerted at the base of the auricle before the heart was opened. Sections through the auricular margin showed these vesicles to be subepicardial cavities evidently produced by air in the subepicardial connective tissue. Muscle fibers of the underlying myocardium were torn, but relatively little hemorrhage had occurred.

Although the auricle contained little blood, cavities suggestive of air-emboli were identified in sections of an adherent clot in one area (Fig. 18). An examination of the remainder of the preserved auricle by means of the dissecting microscope revealed similar subepicardial defects more proximally in the auricular wall. It had been recorded at the time of the autopsy that the heart of this animal contained coagulated blood in the left ventricle only.

No other abnormalities were observed in the thoracic organs of the seals.

The abdominal organs. No macroscopic lesions were recognized in the abdominal organs of the seals. In one animal, the esophagus was dilated with semifluid ingesta. The kidneys of the most severely injured animal were found microscopically to be congested.

Birds.

Fatal injuries to seven of the birds are attributed to the effects of pressure. Six species were represented in this series: horned grebe, *Podiceps auritus* (Linnaeus), 1; pelagic cormorant, 2; greater scaup, *Aythya marila* (Linnaeus), 1; oldsquaw duck, *Clangula hyemalis* (Linnaeus), 1; harlequin duck, 1; and common murre, *Uria aalge* (Pontoppidan), 1. The scaup was found on a freshwater lake about 2 km west of the test-site. The other birds were found along the beaches.

Because post mortem changes had occurred generally in these specimens, few tissues were suitable for preservation and later microscopic examination.

The head and associated structures. In the grebe was a small, subcutaneous hemorrhage between the rami of the mandible, ventrally. In the more severely injured cormorant, the tissues dorsal and lateral to the cervical vertebrae were hemorrhagic, and a small hemorrhagic area was present on the left side of the head just anterior to the auditory opening.

The brain. Marked congestion of the brain was noted in the grebe and in the more severely injured cormorant. In the latter, a small hemorrhage had occurred anteriorly on the ventral surface of the brain.

The thoracic organs. In all specimens, the lungs were dark in color and appeared to be hemorrhagic. Retroperitoneal hemorrhage had occurred dorsal to the lungs in the second cormorant. In the harlequin duck, the left lung had been partially detached posteriorly, and its lateral margin had been lacerated. The trachea of the scaup contained blood, and hemothorax was observed in the grebe and in the harlequin duck.

The lungs of the more severely injured cormorant and of the grebe were examined microscopically, after preservation in spite of the evident post mortem degeneration. The blood-content of the organs was greatly increased, but there was little disruption of the tissues. Rupture of vessels, with hemorrhage into secondary bronchi, had occurred in the grebe. Interstitial emphysema seemed to be marked in both birds, and hemorrhage into perivascular tissue had occurred in the grebe. No gas-bubbles could be identified with certainty.

The abdominal organs. Free blood was present in the abdominal cavity of the murre, the oldsquaw duck, the harlequin duck, and one cormorant. A superficial hemorrhagic area was present dorsally on the anterior hepatic lobe of the murre. The left anterior hepatic lobe had been severely lacerated in the grebe, but as determined from sections, the injury was superficial. In the more severely injured cormorant, the right hepatic lobe was lacerated ventrally, with the injured area coated by coagulated blood.

Some chronic lesions found in the scaup were probably attributable to an old shot-wound. A partially calcified subcutaneous abscess was attached to the posterior margin of the sternum ventrally on the left. The ventriculus was adhered to the left abdominal wall, and the anterior hepatic surface was adhered to the surface of the sternum just to the left of the midline. In this area also was an intra-abdominal abscess measuring about 50 x 25 mm, containing caseous material.

A thick-billed murre, *Uria lomvia* (Linnaeus), also found on the beach, had died of causes unrelated to the detonation. The bird was emaciated, with marked wasting of the pectoral muscles. The lungs were macroscopically normal.

DISCUSSION

The two sea otters caught by rock-falls were resting on the beach about 3.2 km southeast of the test-site at the time of the detonation. The lesions attributable to the effects of falling rocks are obvious and need not be considered further, but the fracturing of the mandible in the less severely injured otter is difficult to explain. Signs of trauma in the soft tissues dorsally, as might be expected if the animal had been struck with sufficient force to break the rather massive ramus, were not observed. Vertical acceleration as a possible cause of this injury cannot now be excluded.

The injury of eight birds is attributed entirely to vertical acceleration. The pattern of the lesions and other observations support the conclusion that these birds were subject to the effects of an acceleration pulse of considerable magnitude. Harlequin ducks, seven of the eight specimens included here, commonly rest on exposed rocks near shore and, of the species present at the time of the detonation, would seem to be the most vulnerable to injury by vertical acceleration. All of the birds in this category were found within a small area of the north beach, from about 1.3 to 1.7 km from the test-site. Four ducks and the cormorant were found together at Sand Beach Cove at about 5:00 p.m. on 6 November. These birds presumably had been killed near shore and had washed onto the beach very soon after death. A fifth duck was found at the same location on 7 November, possibly having been overlooked on the previous evening. An acceleration pulse of 10 g was measured on the beach in this area (M. L. Merritt, personal communication; see also Appendix A, Kirkwood and Fuller, 1972). Two last ducks were found on 8 November, near the mouth of White Alice Creek. Their origin is uncertain, since they might have drifted somewhat eastward before being washed ashore.

In mammals considered to have been underwater at the time of the detonation was seen a considerable range of disruptive changes produced by the combined effects of over- and underpressure. These animals presumably were exposed to a relatively slowly rising pressure-pulse followed by a fall in pressure to cavitation and a subsequent rise to the pre-existing ambient. A representative pressure-time curve is shown in Fig. 19.

The number of injured or dead marine mammals found after the detonation was small in proportion to numbers presumed to have been present within the critical areas at the time of the test. Three mammals, a sea otter and two harbor seals, were found along the north coast of the island. The still-living sea otter and one seal, apparently having been injured when near shore, were on the beach adjacent to the area of high overpressures (between Sand Beach Cove and White Alice Creek, see Fig. 20); the second seal came ashore at Crown Reefer Point, evidently having drifted eastward from the area where injury had occurred. Findings on the Pacific side of the island were similar. Three sea otters and one seal were on the beach opposite the test-site; six sea otters and one seal had drifted eastward, some as far as

St. Makarius Bay and, in the case of the seal, St. Makarius Point, before coming ashore. On 21 November, the body of a sea otter was recovered by a trawl in 30 fathoms of water about 2.5 km off the north shore of the island. The changes in the lungs were indistinguishable from those observed in animals killed by effects of pressure (see Results). Consequently, the sinking of some carcasses and the effects of sea-currents were probably important as factors negatively affecting the probability of recovering dead animals. It is perhaps noteworthy that, with the exception of the single sea otter, all of the animals obtained had sustained severe, probably immediately fatal injuries. Among the living animals observed in the area following the detonation, only three (a seal and two sea otters) exhibited signs of injury; none of these could be captured (Kirkwood and Fuller, 1972).

Two variables were important in influencing the severity of the injuries sustained by mammals in waters near the test-site; extent of immersion (i.e., whether at the surface, or diving); and location in reference to the pattern of pressure gradients. The data (M. L. Merritt, personal communication; Kirkwood and Fuller, 1972, fig. A-7) indicate that overpressures of 200 to at least 300 psi were produced at the sea-floor over an area extending about 5 km from the Bering Sea shore, or well beyond the 30-fathom contour, with an extent along shore of about 5.6 km. Beyond this was a zone about 3.5 km wide, extending nearly to the 60-fathom contour, in which pressures ranged from 100 to 200 psi. The area in which cavitation took place extended about 8 km seaward, with a northwest-southeast extent along shore of about 18 km.

Relative underpressure does not increase with depth, and cavitation occurred at depths from the sea-floor nearly to the surface (M. L. Merritt, personal communication). No comparable data are available for the corresponding area on the Pacific coast.

Sea otters can remain submerged for as long as 6 minutes, and may be capable of diving to depths of 50 m (Barabash-Nikiforov, 1947). However, they usually feed in waters up to 15 fathoms deep, and no positive evidence exists that they ever feed in depths greater than 20 fathoms (Kenyon, 1969, p. 69). It may be assumed, therefore, that the majority of sea otters in the waters bounded by Sea Otter Point and Crown Reefer Point (Fig. 20) potentially would have been exposed to overpressures ranging from 100 to more than 300 psi, depending upon their depth in the water at the time of the detonation, and that all would have been within the area of cavitation. Harbor seals are capable of much deeper, longer dives, but the specimens considered in this report were in shallow waters near the shore at the time of the detonation. The post mortem findings indicate that mammals in waters opposite the test-site on the south side of the island were subjected to pressure-changes of a magnitude similar to those occurring on the north side.

Orbital and auditory lesions.

The four seals and some of the sea otters had been subjected to overpressures of magnitude sufficient to cause fracturing of the bones forming the walls of the nasal cavity. In these specimens, that portion of the frontal bone forming the medial wall of the orbit had been fractured and displaced inward. The palatine bone had been broken away posteriorly in two sea otters, and the maxillary bone anterior to the orbit had been fractured in two of the seals.

By means of air-blast, Richmond et al. (1962) and Chiffelle et al. (1968) produced fractures of this type in the orbital walls of dogs. Two parameters considered to be important in producing such fractures were magnitude of overpressure and rise-time (Richmond et al., 1962; White et al., 1971, p. 37). In dogs exposed to air-blast, the orbital walls of some were fractured by overpressures above 140 psi when the interval to maximum pressure was 30 msec or less (Richmond et al., 1962). Of the two possible mechanisms considered by Richmond et al. to account for the pressure-differential that resulted in fracture of the orbital walls, one appears to be pertinent here, i.e., the transmission of the pressure-pulse hydraulically to the intraorbital tissues, resulting in elevated intraorbital pressure, with delayed pressurization of the air-spaces medial to the orbital walls. Lesions of this kind evidently have not been observed previously in mammals exposed to pressure-pulses in water.

Structural differences between sea otters and seals may influence their susceptibility to the effects of pressure on the nasal cavity. Para-nasal sinuses are lacking in both species, probably as a result of adaptation to the marine environment. The skull of the sea otter is compact and relatively massive, with relatively thick orbital walls.

As compared with the river otter, Lutra canadensis (Schreber), the short nasal cavity of the sea otter is both relatively and absolutely much more capacious. The massive maxilloturbinates are situated mostly posterior to the anterior level of the zygomata, and are contiguous with the ethmoturbinates, which have no posterolateral extensions such as are present in the river otter. In old adults, a thin-walled convexity is present in the frontal bone posterior to the supraorbital process bilaterally, just anterior to the level of the relatively large ethmoid. In the sea otter, fracturing of the orbital wall occurred at the level of the posterior portion of the maxilloturbinates or, in more severely injured animals, over the anterior part of the ethmoturbinates as well. The palatine bone in the sea otter is relatively short and quite thin posteriorly.

The orbits of the harbor seal are very large, accommodating correspondingly large ocular globes, and their medial walls are thinner and more fragile than are those of the sea otter. The maxilloturbinates are mostly anterior to the anterior margin of the orbit, and the ethmoturbinates are situated posterodorsally and covered by somewhat thicker bone. The palatine bones of the harbor seal are relatively heavy. In the seals, the orbital walls were fractured more anteriorly, in the area directly lateral to a spacious cavity posterior to the maxilloturbinates. The frontal bone making up the orbital wall in this area has a thickness of as little as 0.1 mm. The maxilla had been fractured in two seals, in one bilaterally, just anterior to the origin of the zygomatic process where a slight convexity is present in the wall of the nasal cavity. As determined by means of transmitted light, the area in which fracturing occurred is the thinnest part of the maxilla. The bone in this area, overlying the maxilloturbinates, is approximately 0.25 mm thick in the adult harbor seal. Thus, in seals exposed to pressure-pulses underwater, not only the orbital walls may be fractured, but the walls of the nasal cavity may give way at weak points elsewhere. Reflex closure of the nostrils occurs in seals upon diving, and this characteristic might have important implications in animals exposed to rapidly rising overpressures. The nasal cavity of the harbor seal contains a relatively large volume of air.

Evidence of injury to the middle ear was noted in five of the sea otters found dead, and in all of the seals. The middle ear was ostensibly normal in four of the dead sea otters, and in the injured otter found alive. Injury to the auditory structures would not be expected if these animals had been only partly submerged at the surface, but this explanation is not compatible with the extent of injury occurring in the lungs of these animals.

In addition to the physical parameters involved, relative susceptibility to injury by pressure-pulses in water depends to some extent upon structural characteristics of the species of mammal exposed. The size of the pinna is reduced in the sea otter, but whether adaptive modification has occurred in structures of the middle ear is unknown. The tympanic bulla of the sea otter is similar in relative size to that of the river otter, but the promontorium is more massive and the tympanic cavity is more capacious.

In Phoca, the pinna is absent, and both the auditory canal and the middle ear have specialized features. The auditory canal is lined by cavernous tissue which may passively become engorged during diving. The external auditory meatus is short and thick-walled, and the tympanic bulla is very large and inflated, with thick walls of dense bone. In the specimens examined, the tympanic membrane was 11 to 12 mm in greatest diameter. The tympanic cavity is lined with cavernous tissue which, if the eustachian tube is closed, may fill with blood and thus equalize pressure during diving; if the eustachian tube is not closed during diving, the connection with the air-passages provides for equalization with the ambient pressure because of the extent to which the lungs are compressible (Møhl, 1968).

The four seals examined would have been in comparatively shallow waters at the time of the detonation, and therefore would not have been physiologically adjusted to high ambient pressure. The process by which the middle ear was injured in these animals is not entirely clear, nor is it possible to determine that all of the seals were affected in the same way. In the most severely injured specimen, the promontorium had been fractured, and the petrosal bones bilaterally had been broken free and forced dorsad into the cranial cavity. The findings indicate that expansion of air during the phase of negative pressure caused the wall of the tympanic cavity to rupture at its weakest point. Rupture of the tympanic bulla under positive pressure would have caused the fragments to implode.

Some indication of the relative effects of maximal and minimal pressures in producing fatal injury to seals can be obtained by the comparison of two of the specimens studied. The most severely injured animal, having ruptured lungs in addition to the auditory lesions mentioned above, was found on the north beach west of the mouth of White Alice Creek, opposite the test-site. If, as is assumed, this animal was washed directly ashore, it would have been killed in an area where overpressures were in the range of 300 psi. In contrast, the seal that drifted eastward to St. Makarius Point was probably the farthest offshore at the time of the detonation, as indicated also by its having been the least severely injured of the four. Its left tympanic membrane had a transverse fissure and the tympanic cavity contained blood, but the auditory ossicles had not been displaced. The medial walls of the orbit had been only slightly fractured, with little displacement of bone-fragments, although bleeding into the anterior chamber had occurred in both eyes. The maxillary bone had been fractured bilaterally, with minimal displacement of fragments mediad. Injury to the lungs of this animal was considered to have been the least severe among the specimens studied.

Thoracic lesions.

Lesions in the lungs and other thoracic structures of mammals exposed to rapidly rising, high-magnitude pressures underwater are considered to be attributable to the establishment of pressure-gradients in tissues of different densities and to implosive effects. The negative-pressure phase probably contributes significantly to the disruptive changes that occur in the lungs of such mammals, but its effects are difficult to distinguish.

Sea otters and seals are adapted structurally and physiologically, although to different degrees, to existence in the marine environment. The musculature of the body-wall of the sea otter appears to be poorly developed, providing little support to the viscera when the animal is out of water. The thorax, with fourteen pairs of ribs, is very capacious, making up about six-tenths of the length of the trunk. The thorax widens rapidly posteriad and, in relaxed dead specimens in dorsal recumbency, has a maximum width about twice the dorsoventral thickness. The thorax is flexible and apparently collapsible. The diaphragm is more oblique than that in terrestrial mustelids. The sea otter has a large hepatic sinus formed by enlargement of the vena cava posterior to the diaphragm (Urmanov, 1971). Other possible modifications of the vascular system of the sea otter have not been investigated.

The thorax of the harbor seal, with fifteen pairs of ribs, is long and capacious, and the diaphragm is somewhat oblique. The long trachea, consisting of about sixty-five complete and incomplete cartilaginous rings, is flexible but comparatively rigid (Sokolov et al., 1968). This structure is not compressible, as it is in other species [e.g., the Weddell seal, Leptonychotes weddelli (Lesson)] that dive to greater depths (Kooyman et al., 1970). The bronchial tree of the harbor seal is supported by extensive cartilaginous elements that extend nearly to the origin of the alveolar ducts; myo-elastic valves also are present in the alveolar ducts (Bélanger, 1940).

Harbor seals exhale immediately after diving (Harrison and Kooyman, 1968). The dead air-space in the lungs is considered to be about one-tenth of pulmonary volume (Scholander, 1940). If the thorax of the harbor seal is collapsible, the lungs during dives would be progressively compressed with increasing depth until, at about 100 m, only the rigid passages would contain air (Scholander, 1964; Harrison and Kooyman, 1968). The blood-volume of seals is proportionally large, being about 60 per cent greater than that of man, and the venous system is very capacious (Harrison and Kooyman, 1968). A differential distribution of blood occurs during diving, when vasoconstriction occurs in peripheral vessels as a reaction to apneic stress (Scholander, 1964). A large hepatic sinus, formed by enlargement of the posterior vena cava, is present in the harbor seal, with blood-flow through the sinus controlled by the caval sphincter situated just anterior to the diaphragm. Thus, the hepatic sinus and large veins in the abdomen may serve as a reservoir for returning peripheral blood during diving (Harrison and Kooyman, 1968).

These adaptations indicate that seals and, to a lesser extent, sea otters can be expected to respond differently from terrestrial mammals when exposed to under- and overpressures underwater. In contrast to the findings in sea otters, it was observed that thoracic lesions in seals were essentially restricted to the lungs. This may be attributable at least in part to their much larger size. Adult harbor seals around Amchitka have been found to weigh from about 90 to 130 kg, the males being largest.

The lungs of both the seals and the sea otters killed by effects of pressure had been extensively disrupted, although the severity of the lesions differed rather widely from area to area, or even from lobule to lobule. Whether the pattern of pulmonary lesions was related to the distribution of the larger, more rigid air-passages could not be determined. Although the alveoli in atelectatic areas were congested, little intra-alveolar hemorrhage had occurred. This observation suggests the possibility that only the air-filled alveoli were severely affected during the phase of negative pressure. Also, sudden death might have inhibited extensive hemorrhage. In any event, the abundance of gas-bubbles within the pulmonary tissue as well as in the more extensive accumulation of blood and other fluids seems to indicate that distribution of gas in the lungs was significantly affected by the negative phase. Bronchi and bronchioles were found commonly to be filled with blood. However, as observed in tissue sections, some bronchioles that had ruptured were filled with gas, and communicated directly with gas-filled cavities in the surrounding parenchyma. Perforation of the pulmonary pleura in at least some cases clearly involved rupture of bronchioles distally, near the lateral surface of the lobe. Findings in thick sections through such lesions, examined by means of the dissecting microscope, indicated that internal pressure had caused the bronchioles to rupture.

Hemorrhage into surrounding tissues in such lesions appeared to be secondary.

Whether rupture of pulmonary lobes was related to expansion of air during the negative phase might also be considered. It is perhaps significant that, in sea otters, the perforation occurred on lateral surfaces of posterior lobes or, in two cases, in interlobar fissures. Five posterior lobes had been ruptured in the sea otters, and perforations were present posteriorly in both lungs of the seal that probably had been exposed to the greatest overpressure. Since the thorax in both species is relatively rigid anteriorly, overexpansion of the lungs would seem possible only posteriorly, where the margins of the thorax are quite flexible, and where the diaphragm would be displaced posteriad.

In addition to disruptive effects related to the intrinsic inertia of tissues and to spalling, alternating phases of acceleration and deceleration of the thoracic wall produce pressure-differentials that may cause hemorrhaging into air-filled tissues (White et al., 1971). Such hemorrhaging might be expected to occur especially in mammals such as sea otters and seals, in which an unusually large volume of blood is contained by the hepatic sinus and by the large veins in the abdominal cavity. The pressure-differentials may also cause air to enter fluids in the lungs. Findings in sections indicate that venous-alveolar fistulae were numerous in the lungs of these animals. In addition, ruptured bronchioles were frequently observed within extensive hemorrhagic areas; these would seem also to be an important source of air-emboli.

Stomach-contents were found in the larger airways of the lungs of most of the sea otters found dead, and the esophagus of one of the seals was distended with ingesta. Compression of the abdomen evidently caused the stomach to empty, forcing the contents into the esophagus and pharynx. Inspiration of the ingesta then occurred during a subsequent interval of negative thoracic pressure. The exact sequence of events involved is not clear.

In the four dead sea otters that had no recognized lesions in the middle ear, the anterior pulmonary lobes exhibited only slight to moderate injury, while the posterior lobes had been severely affected. In reference to the effects of air-blast, Desaga (1950, p. 1289) expressed the opinion that there is little possibility of severe blast-injury without rupture of the tympanic membranes. However, recent investigations (Richmond _et al_., 1968; White _et al_., 1970) have shown that considerable variation is to be expected in the response of the tympanic membrane in animals exposed experimentally to overpressures of short duration. The findings of White _et al_. suggested that injury to the tympanic membrane is more likely to occur with rapidly rising overpressures in mammals of some species, but a clear correlation between physical parameters and expected degree of injury has not yet been established. White _et al_. pointed out that severe injury to the lungs without rupture of the tympanic membranes may occur in animals exposed to air-blast. In the sea otter, structural adaptations of the external ear may afford much better protection against pressure-pulses underwater as compared with the ear of terrestrial mammals subjected to air-blast.

Sea otters commonly swim or rest on their back at the surface, and they are often seen also in vertical position with only the head extended above water. In any area, a considerable proportion of otters in the water would be expected to be at the surface at any given time. In the case of conventional explosions underwater, the instantaneously changing pressure-pulse arriving at the air-water interface is reflected as a tension-pulse, and those parts of an animal's body below the surface are exposed to maximum pressure as well as to the effects of the tension-pulse (Wakeley, 1945). Severe injury to persons exposed to such pressure-pulses at the surface has been frequently observed. When fatal injuries occur under these circumstances, post mortem findings include extensive pulmonary hemorrhage and alveolar rupture, with significant increase in lung-weight (Huller and Bazini, 1970). In the case of the Cannikin detonation, the effect of the pressure-pulse at the surface was quite different. With the refraction at the sea-floor as the incident wave passed from rock to water, the slowly changing pressure-pulse approached the surface from below, its magnitude decreasing inversely with depth. The pressure at the surface would be near zero, and submerged portions of animals at the surface would thus be exposed to negligible overpressure (M. L. Merritt, personal communication). In contrast, the effect of acceleration would be greatest at the surface, so that partially submerged animals might have been thrown violently from the water. The possible effects of acceleration and of cavitation at the surface are unknown.

Injuries to other intrathoracic structures in the sea otters, with the exception of some of the vascular lesions, may be attributed to shearing and tearing caused by acceleration and deceleration of the thoracic wall (White et al., 1971), which would seem to account for injuries to the mediastinum, pulmonary ligaments, and other associated structures. The specific cause of subepicardial and myocardial hemorrhages in blast-injured mammals has not been determined (Chiffelle et al., 1968). The subepicardial vesicles observed on the right auricle of one of the seals were presumably related in some way to air-embolism. It would appear that emboli could have entered the right side of the heart only by way of the pulmonary artery.

Mammals injured by air-blast frequently exhibit a pattern of intermittent hemorrhage beneath the pulmonary pleura. Such hemorrhages, usually designated "rib-markings," evidently correspond to intercostal spaces, rather than to ribs (Clemedson and Granstöm, 1950). In some cases, corresponding hemorrhagic areas are also present beneath the parietal pleura in the intercostal spaces. Schardin (1950, p. 1213) reported that fracturing of ribs and tearing of intercostal muscles may occur in animals exposed experimentally to air-blast. Rössle (1950, p. 1272) observed intercostal hemorrhage in a man who had been killed by blast during an air-raid. The severe injuries sustained by the sea otters and seals resulted in subpleural hemorrhage so extensive and diffuse as to obscure any pattern that could be related to structures of the thoracic wall. Also to be considered is the possibility that "rib-markings" would not have been produced if the pulmonary injuries were essentially decompressive in origin.

Hemorrhages in the intercostal spaces were observed only on the left side of one otter. In this animal, the left side of the thorax had been severely injured, with separation of some of the costochondral junctions and tearing and hemorrhage of the thoracic muscles externally. The left zygomatic arch also had been broken. In a second animal, the ventral body-wall had injuries indicative of external trauma, and a portion of the anterior lobe of the right lung had been torn away. Since animals diving near the sea-floor would be in density-equilibrium with the surrounding water, it seems improbable that such injuries could be caused by acceleration, although animals diving in shallow water perhaps could be injured by rocks displaced upward. Both of these otters also exhibited thoracic lesions attributable to effects of pressure-changes.

Extensive retroperitoneal hemorrhage was observed lateral to the vertebral column in most of the sea otters examined. In two animals, such hemorrhage was partly caused by rupturing of dorsal intercostal vessels, one case involving both veins and arteries. The extensive, more diffuse hemorrhage lateral to the vertebral bodies appears to be attributable to rupturing of numerous small tributaries of the internal vertebral system of veins. The retroperitoneal hemorrhage thus appears to have been caused by the same process that produced lesions in the central nervous system (see below).

Lesions of the central nervous system.

In the sea otter, as compared with the dog or with man, the internal vertebral system of veins is more highly developed and complex (Fig. 21). The system is still more specialized in *Phoca* and in some other marine mammals (Harrison and Tomlinson, 1956; Watson and Dowd, 1972). Such modification is apparently related to adaptation to the marine environment. The system of vertebral veins provides an alternate route for return of blood to the heart by way of the azygos vein.

In the sea otter, the azygos vein enters the anterior vena cava on the right side at the level of the fifth intercostal space (Fig. 21). Three large veins arising from the internal vertebral veins on the right combine with one from the left side, forming a common trunk that extends a short distance ventrad and joins the azygos anteriorly. The three veins on the right contain valves. The left vein, lacking a valve, arises from a large internal vertebral sinus. An anterior connection between the vena cava and the internal vertebral veins is provided by the costocervical vein, which runs slightly posteriad, joining the anterior vena cava at about the level of the first rib. The internal vertebral system communicates with the dural sinuses in the skull (Barnett *et al.*, 1958), and the veins of the spinal cord are tributaries to this system.

Compression of the vena cava, including the hepatic sinus, and its tributary vessels in the abdominal cavity would force blood into the internal vertebral system, mainly by way of the left tributary of the azygos and the costocervical vein. If the pressure were sufficient, rupturing of some of these vessels might result. The extensive retroperitoneal hemorrhage seen lateral to the vertebral column in the sea otter is attributed to rupture of small, extravertebral tributaries to the internal vertebral system of veins. Internal rupturing of vessels would seem to account for the subdural and subarachnoid hemorrhage in the spinal cord and brain. Thoracic lesions attributable to injury of vessels of the internal vertebral system were severe from about the level of the azygos vein posteriad, a pattern that would seem to be a result of increased pressure posterior to the large internal vertebral sinus from which the left branch of the azygos arises. Similarly, the lesions in the brain would result when blood entering from the costocervical vein was forced anteriad in the internal vertebral veins.

Increased hydrostatic pressure in the internal vertebral system, with the consequences described above, is attributed to compression of the capacious vessels in the abdomen. A similar hypothesis was proposed by Young (1945) to account for increased intracranial pressure in man exposed to air-blast. However, because of their specialized azygos venous system, sea otters and seals appear to be much more susceptible to pressure-induced injury to the central nervous system than are man and other terrestrial mammals.

Lesions in abdominal organs.

In man, immersion-blast usually produces injury to intra-abdominal organs, especially those containing gas. A frequent finding is perforation of the large intestine (Wakeley, 1945; Huller and Bazini, 1970). Parenchymatous organs seem rarely to be affected in man. Perforations or other lesions of the intestine or stomach were not observed in the sea otters and seals killed by pressure-effects. The absence of such injury might be attributable to a lack of gas in the alimentary canal of these mammals.

In eight of the nine sea otters killed by effects of pressure, traumatic lesions were present in the liver. These ranged from subcapsular hemorrhage to laceration or maceration of the superficial tissue of the anterior surfaces of the larger lobes. Since the thorax of the sea otter appears to be collapsible dorsoventrally, especially posteriorly, the hepatic lesions seem probably to be attributable to the crushing effect exerted by the costal margins when the thorax was suddenly compressed. During the positive-pressure phase, the liver evidently would have been immobilized by simultaneous compression of the abdomen, preventing any displacement of the organ posteriad.

The severe congestion observed in the kidneys of some animals was probably a result of compression of the posterior vena cava and renal veins.

Conclusions.

The lesions observed in marine mammals injured by underwater effects of the Cannikin detonation are attributed to both over- and underpressure. The role of negative pressure in the genesis of disruptive changes in the lungs is not entirely clear. The pattern of lesions observed is considered to be related in part to structural characteristics of species of mammals adapted to the marine environment. In some of the sea otters, injuries produced by an unknown mechanism were superimposed upon those caused by effects of pressure.

The small number of injured but still living sea otters observed following the detonation suggests that the mortality was high among those that were exposed to pressure-changes within limited areas (e.g., between Sea Otter Point and Crown Reefer Point along the Bering Sea coast). A high proportion of the local population in the two critical areas potentially would have been exposed to lethel pressure-changes because sea otters frequent relatively shallow waters (i.e., usually within the 20-fathom contour), and thus would tend to be concentrated relatively near shore. Thus, diving animals within certain, still undefined limits would be exposed to severe pressure-changes; animals at the surface presumably would not be injured.

ACKNOWLEDGEMENTS

At Amchitka Island, Mr. James A. Estes (Cooperative Wildlife Research Unit, University of Arizona, Tucson), Dr. Theodore R. Merrell, Jr. (Auke Bay Biological Laboratory, National Oceanic and Atmospheric Administration, Auke Bay), Dr. M. L. Merritt (Sandia Laboratories, Albuquerque), and Mr. Charles A. Simenstad (Fisheries Research Institute, University of Washington, Seattle) by recording information and assisting in other ways, helped greatly to speed and ease the work in the post mortem examinations. Dr. Clayton S. White, President-Director of the Lovelace Foundation for Medical Education and Research, Albuquerque, gave generously of his time for discussion of blast-injury and made helpful suggestions concerning this study. He and his colleagues at the Lovelace Foundation, Dr. Robert K. Jones (Department of Hematology), Dr. Thomas L. Chiffelle (Department of Pathology), and Dr. Donald R. Richmond and Dr. Edward G. Damon (Department of Comparative Environmental Biology), critically reviewed a draft of this report. Dr. M. L. Merritt in addition on several occasions provided important information in questions concerning pressure-effects, permitted inclusion of some data, and also reviewed the manuscript. At the Arctic Health Research Center, I am indebted to Mrs. Karen B. Coady, who prepared many tissue-sections, and to Mr. Christopher Ankrom, who continued the histological work; to Dr. Francis H. Fay, who assisted with an interpretation of some of the findings and critically reviewed the manuscript; and to Mr. Gerald C. Kelley, who assisted in the photography. Dr. Rollo W. Van Pelt, Institute of Arctic Biology, University of Alaska, Fairbanks, kindly provided consultation on some of the tissue-sections. Finally, I am grateful to Mr. Ernest D. Campbell, Chief of the Bioenvironmental Branch, U. S. Atomic Energy Commission, Nevada Operations Office, Las Vegas, who requested that the investigation be undertaken and who made all arrangements for its support. I express my thanks to these individuals for their contributions to this study; I assume sole responsibility for any imperfections or errors in the interpretations and conclusions presented.

REFERENCES CITED

Barabash-Nikiforov, I. I. 1947. Kalan. Sov. Min. RSFSR, Glavnoe Upravlenie po Zapovednikam. Nat. Science Found., Israel Program for Scientific Translations, Jerusalem, 1962.

Barnett, C. H., R. J. Harrison, and J. D. W. Tomlinson. 1958. Variations in the venous systems of mammals. Biol. Rev. 33: 442-487.

Bélanger, L. F. 1940. A study of the histological structure of the respiratory portion of the lungs of aquatic mammals. Amer. J. Anat. 67: 437-461.

Chiffelle, T. L., R. K. Jones, D. R. Richmond, and E. G. Damon. 1968. The biologic and pathologic effects of blast injury. pp. 90-105 in Accident Pathology, K. M. Brinkhous (Ed.). U. S. Govt. Printing Office, Washington, D. C.

Clemedson, C.-J., and S. A. Granstöm. 1950. Studies on the genesis of "rib markings" in lung blast injury. Acta Phys. Scand. 21: 131-144.

Desaga, H. 1950. Blast injuries. War experiences concerning blast effects on man. pp. 1274-1293 in German Aviation Medicine, World War II, Vol. 2. U. S. Govt. Printing Office, Washington, D. C.

Harrison, R. J., and G. L. Kooyman. 1968. General physiology of the Pinnipedia. pp. 211-296 in The Behavior and Physiology of Pinnipeds, R. J. Harrison, R. C. Hubbard, R. S. Peterson, C. E. Rice, and R. J. Schusterman (Eds.). Appleton-Century-Crofts, New York.

Harrison, R. J., and J. D. W. Tomlinson. 1956. Observations on the venous system in certain Pinnipedia and Cetacea. Proc. Zool. Soc. London, 126, pt. 2: 205-233.

Huller, T., and Y. Bazini. 1970. Blast injuries of the chest and abdomen. Arch. Surg. 100: 24-30.

Kenyon, K. W. 1969. The Sea Otter in the Eastern Pacific Ocean. N. Amer. Fauna 68. U. S. Govt. Printing Office, Washington, D. C.

Kirkwood, J. B., and R. G. Fuller. 1972. Bioenvironmental Safety Studies, Amchitka Island, Alaska. Cannikin D+2 Months Report, BMI-171-147, Biology and Medicine. Nat. Tech. Inf. Service, U. S. Dept. Commerce, Springfield, Virginia.

Kooyman, G. L., D. D. Hammond, and J. P. Schroeder. 1970. Bronchograms and tracheograms of seals under pressure. Science, 169: 82-84.

Møhl, B. 1968. Hearing in seals. pp. 172-195 in The Behavior and Physiology of Pinnipeds, R. J. Harrison, R. C. Hubbard, R. S. Peterson, C. E. Rice, and R. J. Schusterman (Eds.). Appleton-Century-Crofts, New York.

Rausch, R. L. 1953. Studies on the helminth fauna of Alaska. XIII. Disease in the sea otter, with special reference to helminth parasites. Ecology, 34: 584-604.

Richmond, D. R., E. G. Damon, E. R. Fletcher, I. G. Bower, and C. S. White. 1968. The relationship between selected blast-wave parameters and the response of mammals exposed to air blast. Ann. N. Y. Acad. Sci. 152: 103-121.

Richmond, D. R., D. E. Pratt, and C. S. White. 1962. Orbital "Blow-out" Fractures in Dogs Produced by Air Blast. Tech. Prog. Rept., DASA-1316. Defense Atomic Support Agency, U. S. Dept. Defense, Washington, D. C.

Rössle, R. 1950. Pathology of blast effects. pp. 1260-1273 in German Aviation Medicine, World War II, Vol. 2. U. S. Govt. Printing Office, Washington, D. C.

Schardin, H. 1950. The physical principles of the effects of a detonation. pp. 1207-1224 in German Aviation Medicine, World War II, Vol. 2. U. S. Govt. Printing Office, Washington, D. C.

Scholander, P. F. 1940. Experimental investigations on the respiratory functions in diving mammals and birds. Hvalråd. Skr. 22: 1-131.

Scholander, P. F. 1964. Animals in aquatic environments: diving mammals and birds. pp. 729-739 in Handbook of Physiology, Sect. 4: Adaptation to the Environment. Amer. Physiol. Soc., Washington, D. C.

Sokolov, A. S., G. M. Kosygin, and A. P. Shustov. 1968. Stroenie legkikh i trakhei u Beringovomorskikh lastonogikh. pp. 252-263 in Lastonogie Severnoi Chasti Tikhogo Okeana. Trudy VNIRO, Vol. 68. Pishchevaia Promyshlennost', Moscow.

Urmanov, M. I. 1971. Sravnitel'no-morfologicheskaia kharakteristika pecheni lastonogikh i kalana. pp. 289-300 in Morskie Mlekopitaiushchie (Kotiki i Tiuleni). Trudy VNIRO, Vol. 82. Otdel Nauchno-Tekhnicheskoi Informatsii, Moscow.

Wakeley, C. P. G. 1945. Effect of underwater explosions on the human body. Lancet, 9 June 1945, pp. 715-718.

Watson, A. G., and D. A. Dowd. 1972. The azygos veins of the Weddell seal, Leptonychotes weddelli (Lesson). Canad. J. Zool. 50: 1583-1587.

White, C. S., I. G. Bowen, and D. R. Richmond. 1970. The relation between eardrum failure and blast-induced pressure variations. Space Life Sci. 2: 158-205.

White, C. S., R. K. Jones, E. G. Damon, E. R. Fletcher, and D. R. Richmond. 1971. The Biodynamics of Airblast. DNA 2738T. Defense Nuclear Agency, Washington, D. C.

White, C. S., and D. R. Richmond. 1959. Blast Biology. Tech. Prog. Rept., Biology and Medicine, TID-5764. U. S. Atomic Energy Comm., Oak Ridge, Tennessee.

Young, M. W. 1945. Mechanics of blast injuries. War Med. 8: 73-81.

FIGURE SECTION

In order to consolidate the color presentations in this document, it was necessary to rearrange some of the figures out of normal numerical sequence. For this reason, the figure numbers and their location are given below.

FIGURE 2. Skull of old adult sea otter, showing perforation of the medial orbital wall. Chronic periodontal disease caused perforations around roots of teeth.

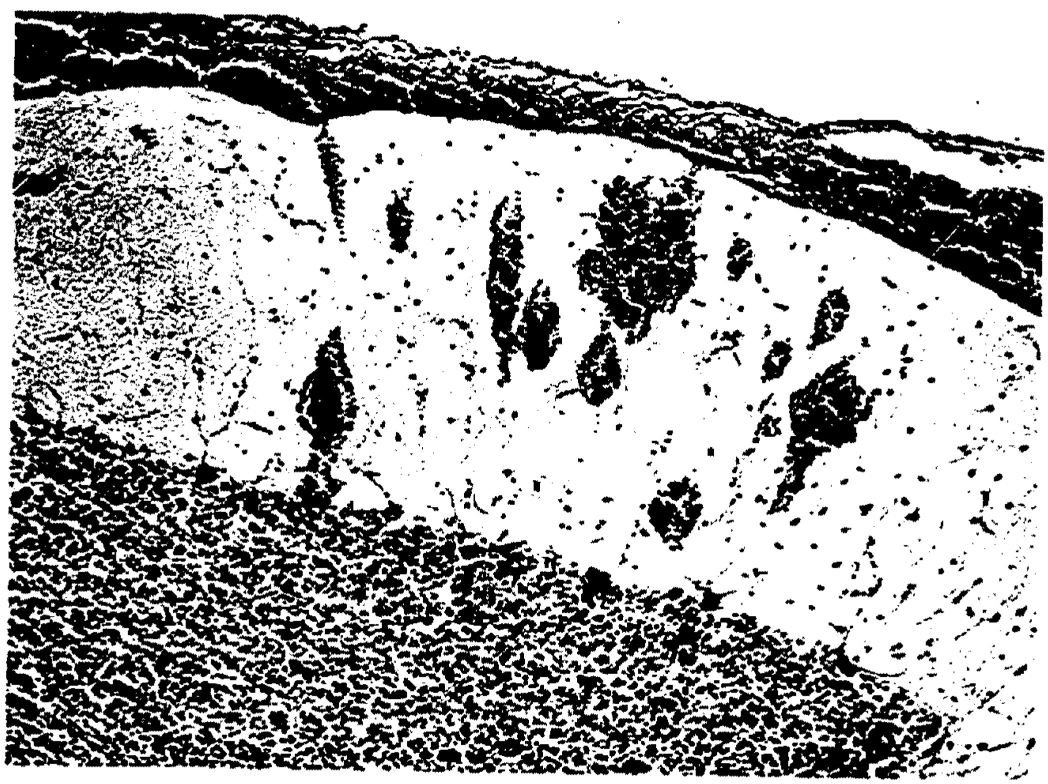


FIGURE 3. Subarachnoid hemorrhage and hemorrhage in molecular layer of cerebellum in sea otter. Hematoxylin-eosin. 95 X.

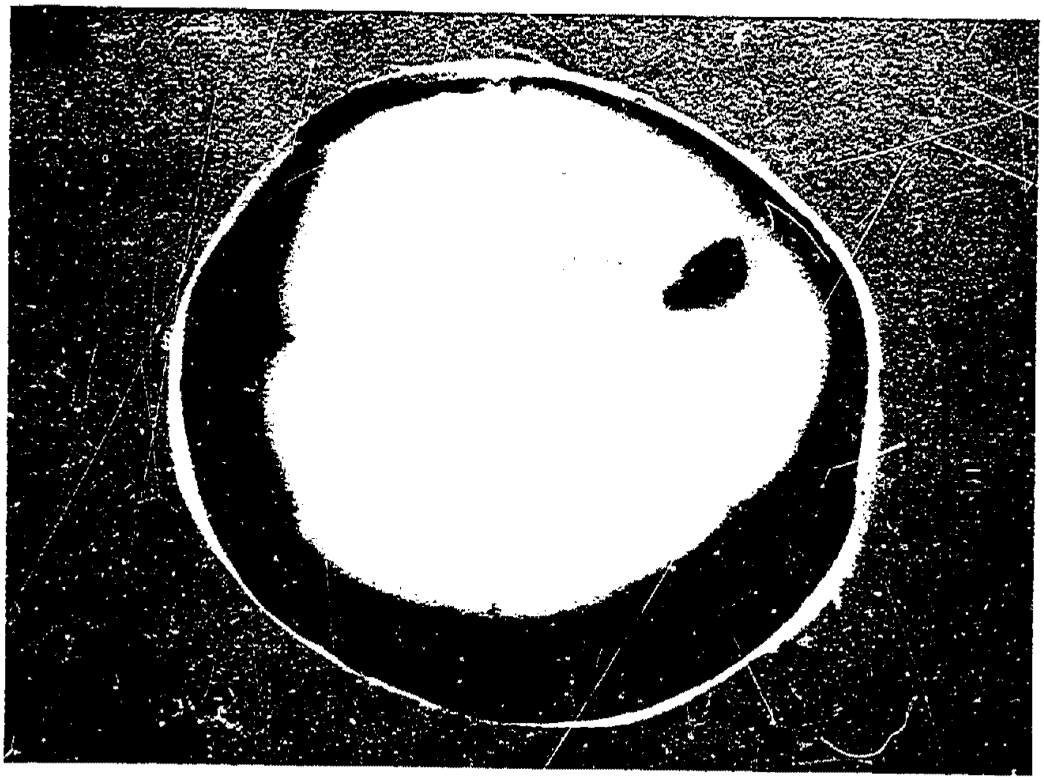


FIGURE 4. Section across formalin-fixed spinal cord of sea otter, showing subdural hemorrhage and hematomyelia.

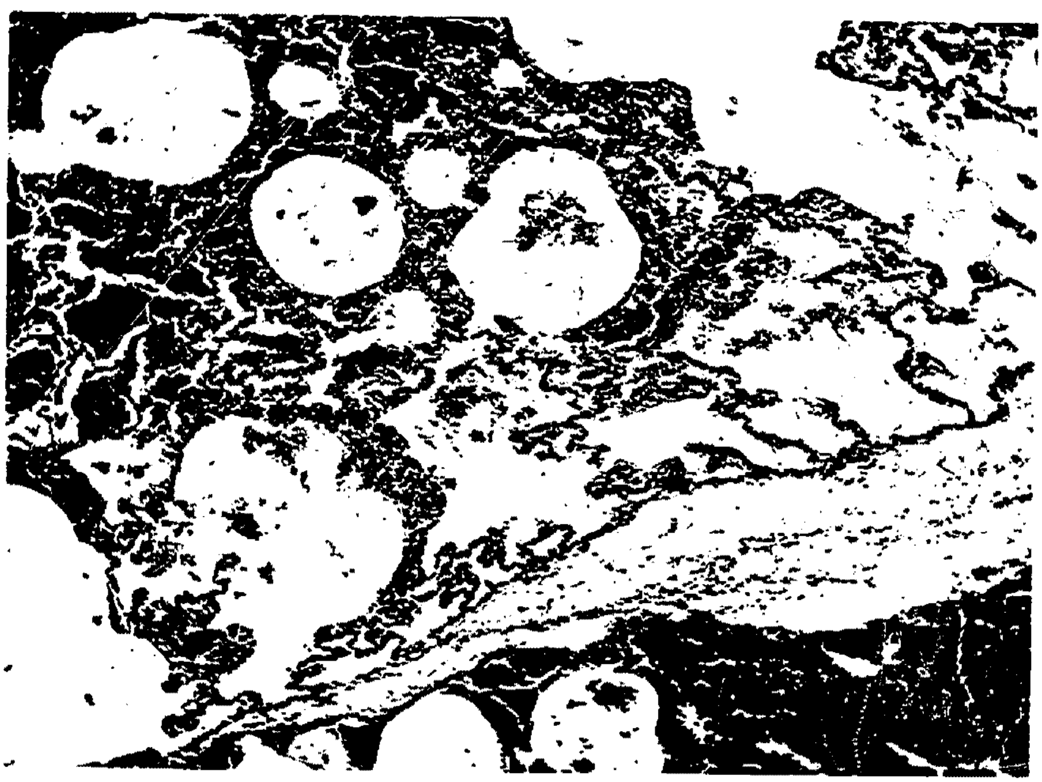


FIGURE 9. Large gas-filled cavities and interlobular emphysema in lung of sea otter. Hematoxylin-eosin. 80 X.

FIGURE 10. Interstitial hemorrhage in myocardium of sea otter. Mallory's anilin blue collagen stain. 225 X.

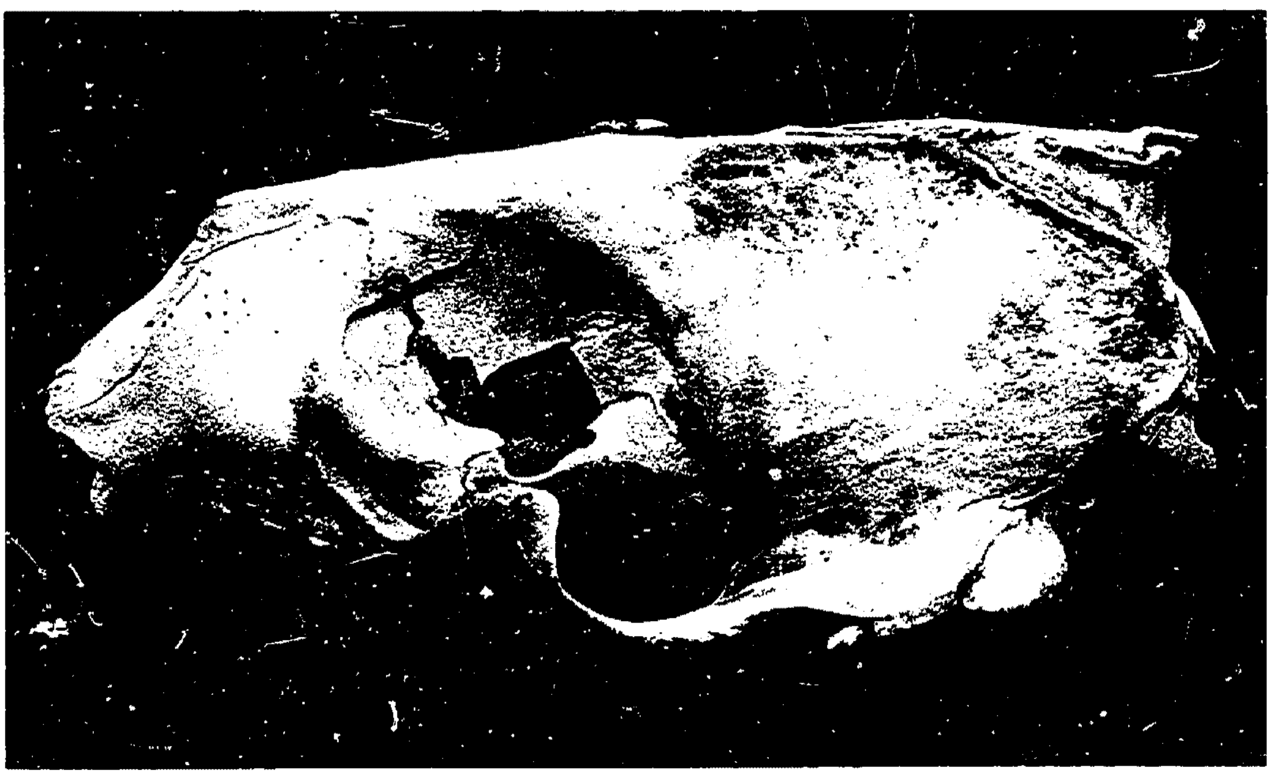


FIGURE 11. Skull of harbor seal, showing fracturing of medial orbital wall.

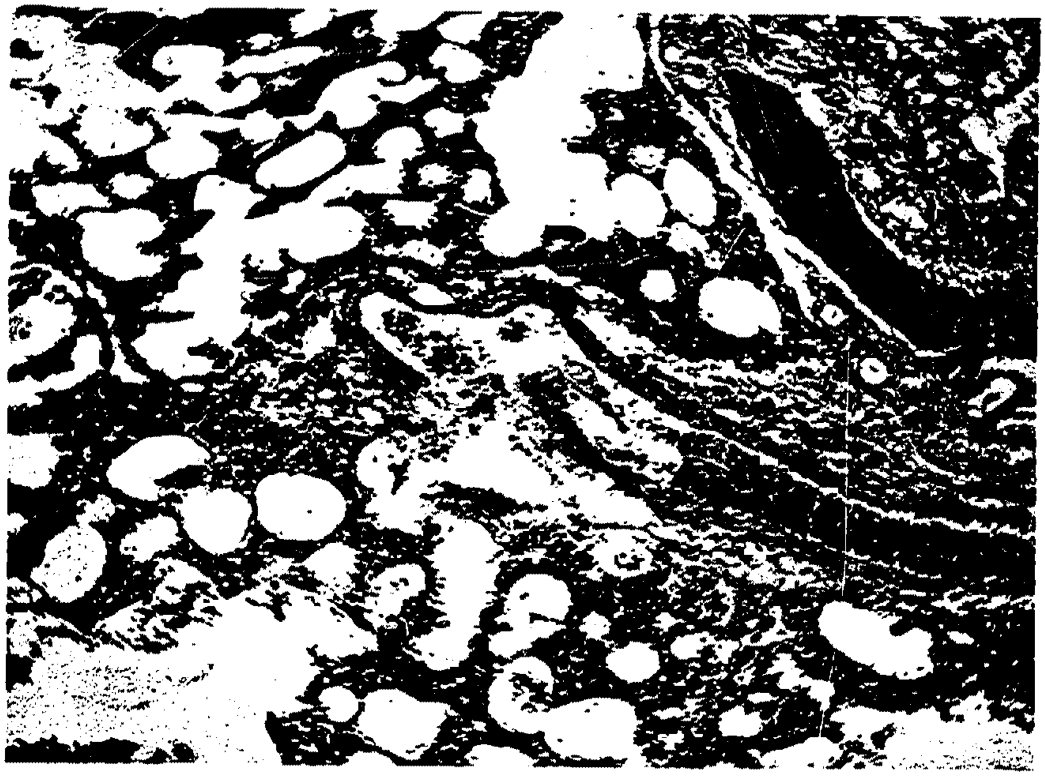


FIGURE 15. Venous-alveolar fistula in lung of harbor seal. Hematoxylin-eosin. 85 X.

FIGURE 16. Embolus of unidentified tissue in vein in lung of harbor seal. Hematoxylin-eosin. 240 X.

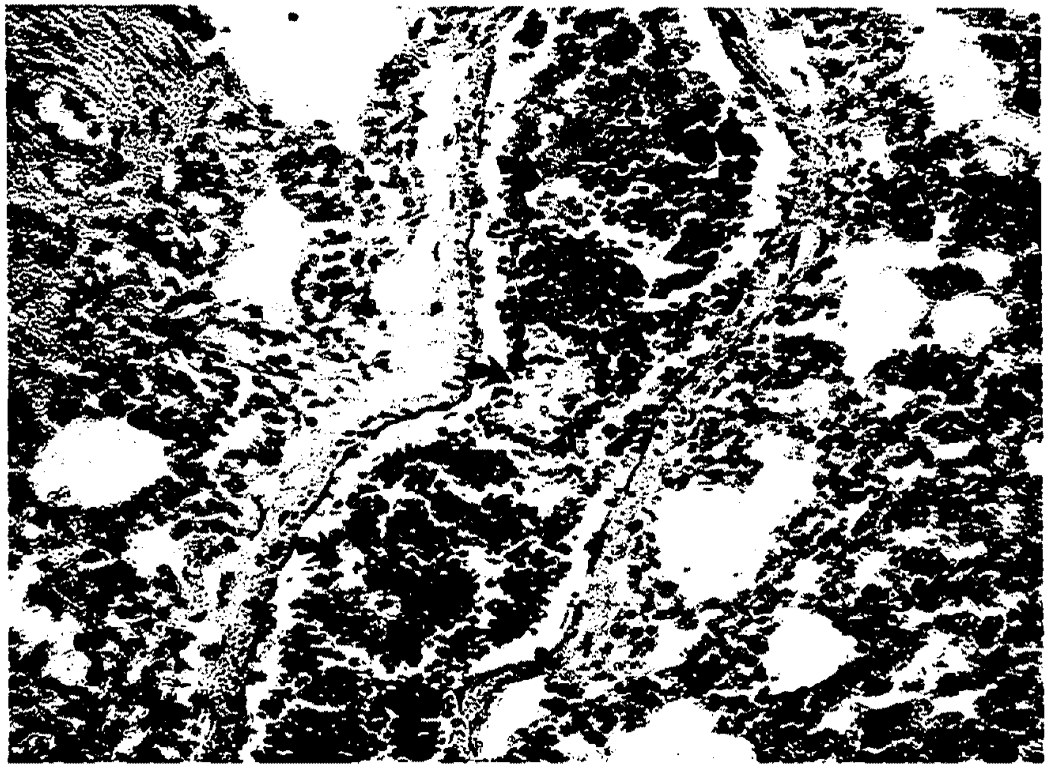


FIGURE 17. Emboli of probable alveolar tissue in vein in lung of harbor seal. Hematoxylin-eosin. 230 X.

FIGURE 18. Probable gas-emboli in coagulated blood in right auricle of harbor seal. Hematoxylin-eosin. 110 X.

C O L O R   P L A T E S

FIGURE 1. Interlobular emphysema and hemorrhage in lung of sea otter. Hematoxylin-eosin. 65X.

FIGURE 5. Ruptured bronchiole and gas-filled cavities in lung of sea otter. Hematoxylin-eosin. 70X.

FIGURE 6. Ruptured vein forming venous-alveolar fistula in lung of sea otter. Weigert-van Gieson. 65X.

FIGURE 7. Intra-alveolar and subpleural hemorrhage in lung of sea otter. Hematoxylin-eosin. 70X.

FIGURE 8. Severe intra-alveolar hemorrhage with ruptured vein in lung of sea otter. Hematoxylin-eosin. 265X.

FIGURE 12. Subpleural emphysema with hemorrhage, and intra-alveolar hemorrhage in lung of harbor seal. Hematoxylin-eosin. 70X.

FIGURE 1.

FIGURE 5.

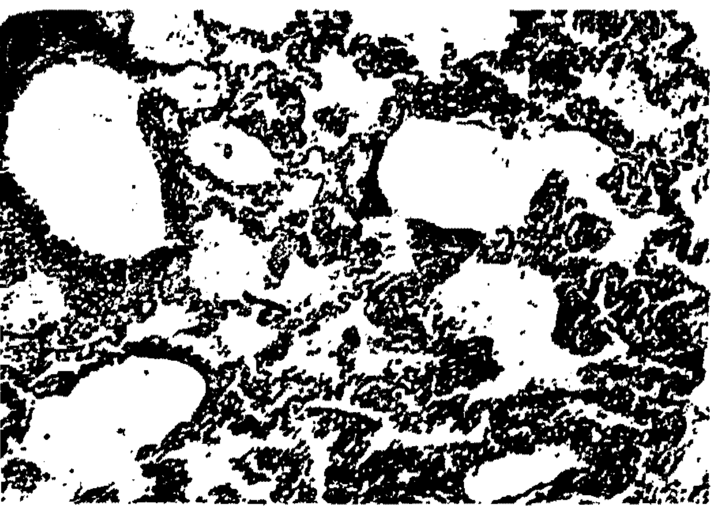


FIGURE 6.

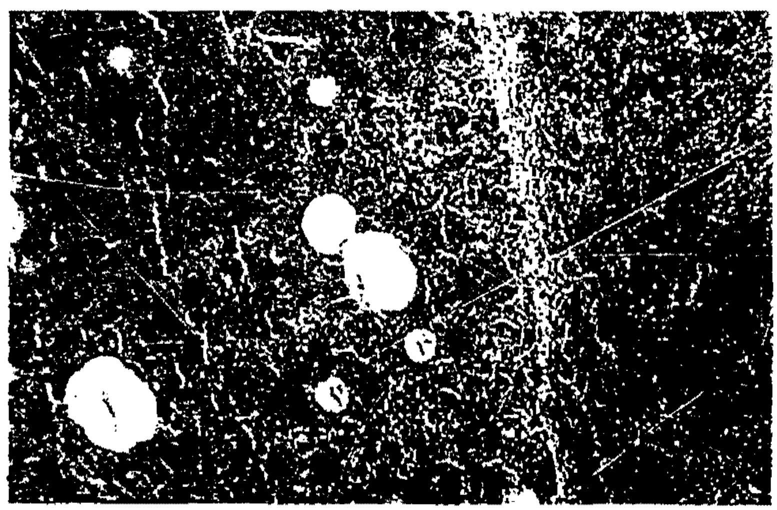


FIGURE 7.

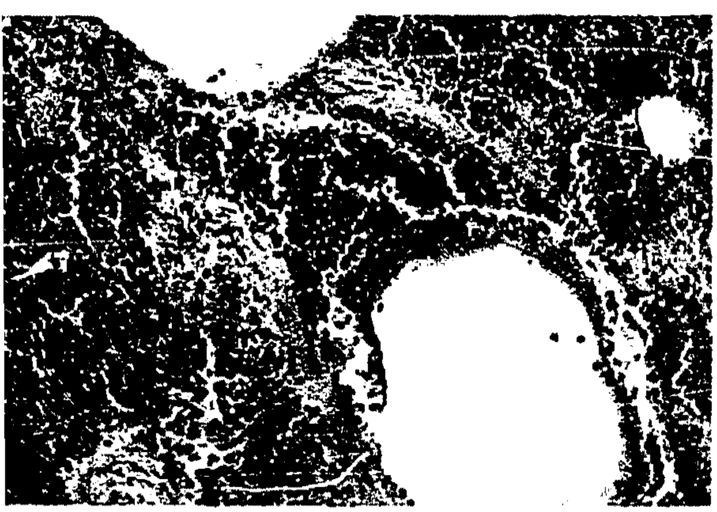


FIGURE 8.

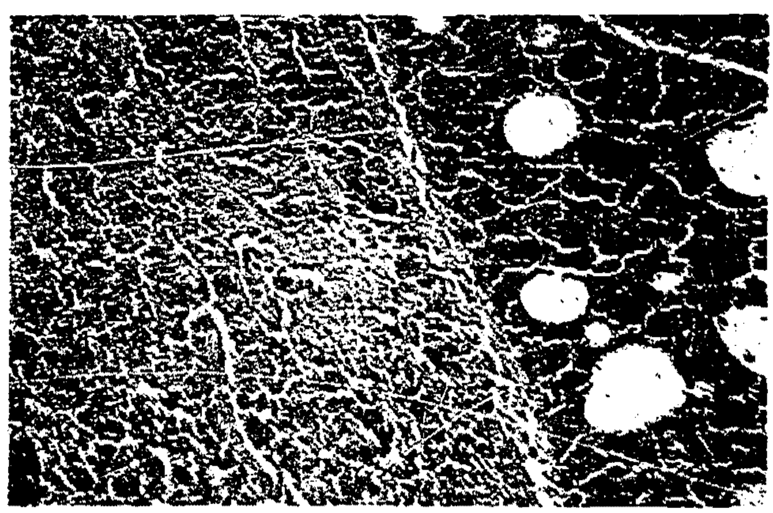


FIGURE 12.

FIGURE 13. Ruptured vein and perivascular emphysema in lung of harbor seal. Hematoxylin-eosin. 65X.

FIGURE 14. Perivascular and peribronchial hemorrhage in lung of harbor seal. Hematoxylin-eosin. 65X.

FIGURE 21. Double-injected circulatory system of sea otter, showing relationships of intravertebral venous system in neck and thorax. Veins - yellow; arteries - red.

FIGURE 13.

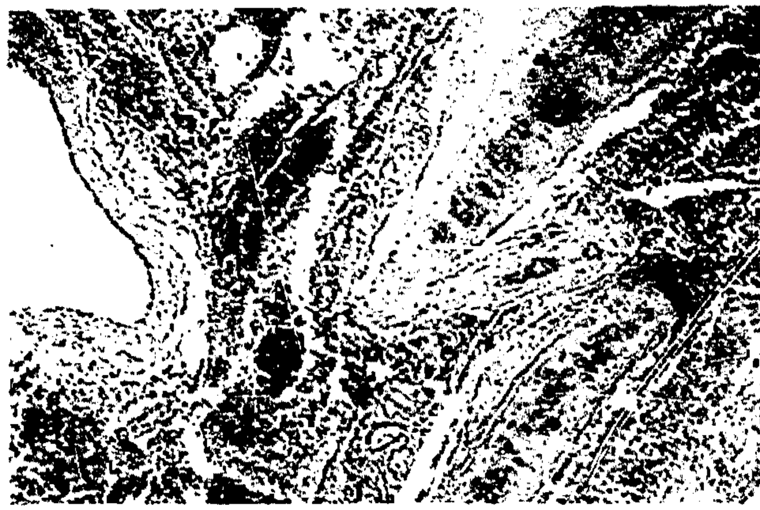


FIGURE 14.

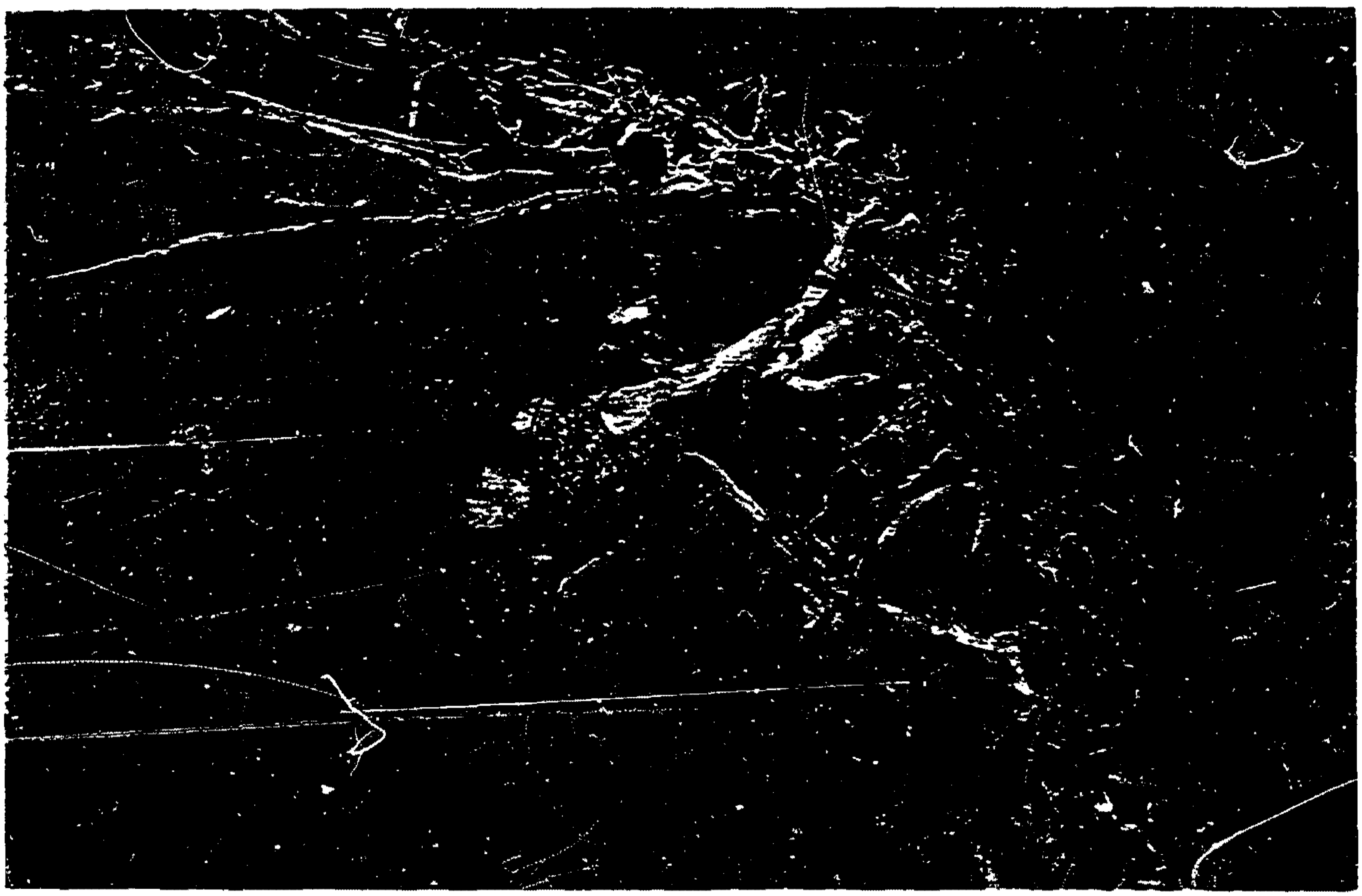
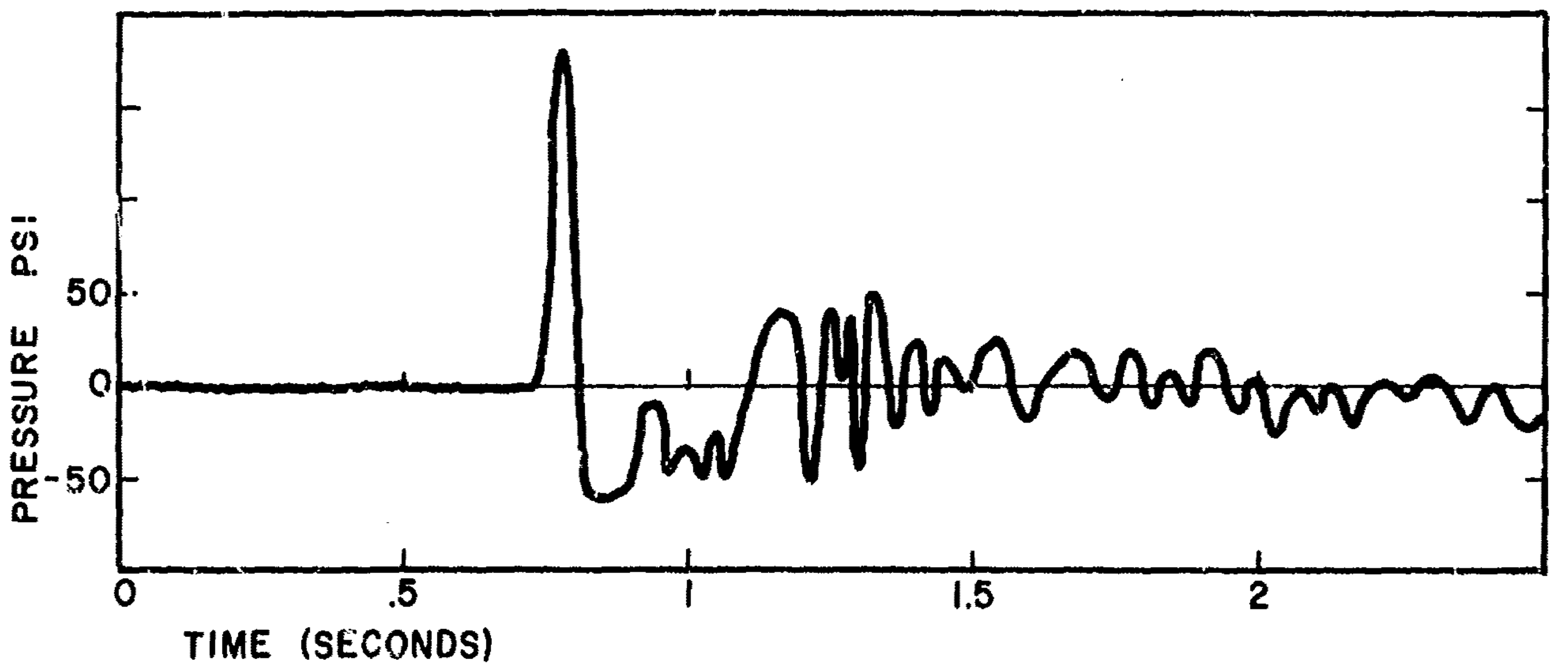


FIGURE 21.

FIGURE 19. Representative pressure-time curve. (Redrawn from original provided by M. L. Merritt.)



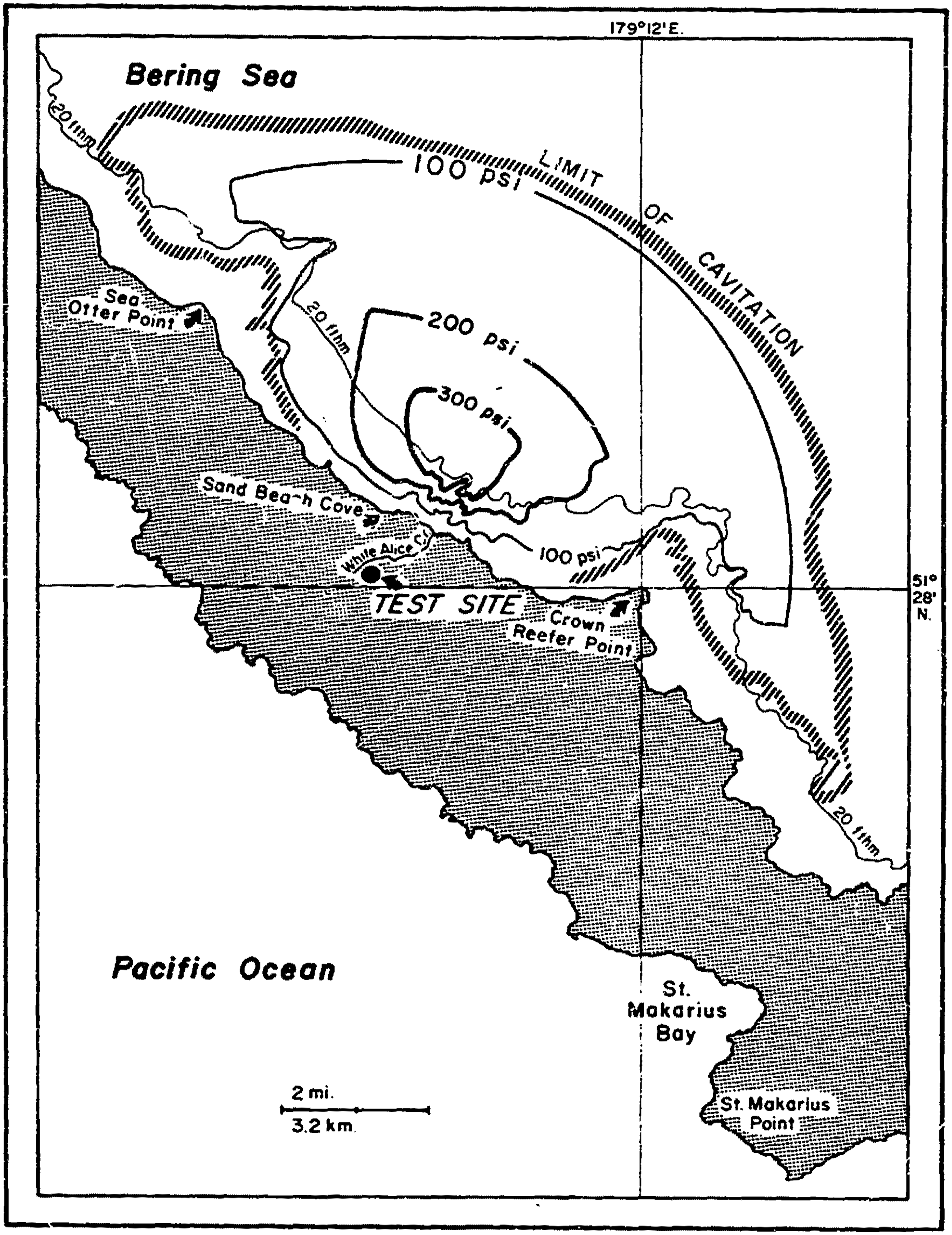


FIGURE 20. Part of Amchitka Island and adjacent Bering Sea, showing predicted overpressure-contours and limit of cavitation, with place-names mentioned in the text. (Redrawn from map provided by M. L. Merritt.)