NEUROPATHOLOGIC OBSERVATIONS OF HEAD TRAUMA IN THE NORTHERN FUR SEAL

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Abstract: The skulls and brains of 19 northern fur seals *(Callorhinus ursinus)* were studied. These animals had been rendered unconscious by blunt head trauma as part of the harvest procedure on the Pribilof Islands of Alaska. All seals had depressed, comminuted fractures of the skull. Epidural, subdural and subarachnoid hemorrhage were frequently present. In addition, the brains showed varying prevalence and degrees of laceration, superficial contusion and hemorrhage in deep midline or paramedian structures and ventricular system. Lesions observed were related to the head trauma.

INTRODUCTION

The Pribilof Islands of Alaska lie in the Bering Sea, between that state and the Soviet Union. These islands are the summer home and breeding grounds for approximately 80% of the world's northern fur seal population.[7] Almost from the time of discovery of the Pribilofs by Russian explorers in 1786-87, these seals have been killed for their pelts. The history of this activity and its effect upon the fur seal herd have been reviewed.[7]

There has been growing concern among United States citizens about the humaneness of methods used in harvesting these animals.[9] Because of this, a panel of six veterinarians (see acknowledgments) was invited by the National Oceanographic and Atmospheric Administration of the United States government to evaluate this aspect of the fur seal harvest.[9] The panel visited St. Paul Island in the Pribilofs during July, 1971.

This paper describes the craniocerebral lesions of animals rendered unconscious during the 1971 harvest by a single stunning blow to the head. It constituted an addendum to the report of the veterinary panel noted above.[9]

METHOD OF HARVEST

The fur seal harvest procedure as observed by the veterinary panel has been described,[9] and will be summarized below. The report of this panel[9] should be consulted for more detailed data. A somewhat similar description of the 1967 harvest has been given by Simpson.[8]

Bachelor male seals, mainly 3-4 years of age (a few 2, 5, and 6 years) were driven a short distance inland from the Pribilof Island beaches, where they tend to congregate during summer. They were herded into relatively large groups, from which smaller numbers (usually 5-6) of these animals were driven toward a group of men equipped with tapered hardwood clubs resembling elongated baseball bats. These clubs were approximately 155 cm long and weighed 2.2 kg.[8] (there probably was some increase in weight during the harvest due to the uptake of moisture by the club*). These men rendered the animals unconscious with a blow to the head, using the blunt end of these clubs. The animals were supporting themselves in a semi-erect posture when struck, and their heads were not restricted in movement. In almost all instances a single blow was sufficient to produce uncon-

* Keyes, M. C. — Personal communication.

sciousness. Within 60 seconds after being struck, the unconscious animals were killed by exsanguination (incision of heart and great vessels) and then skinned.

Five of the 19 animals in this study were used by the veterinary panel for physiologic studies after being rendered unconscious. These animals either died or were killed at times ranging up to 7 minutes after the blow. There were no significant differences in craniocerebral lesions between these five seals and the ones killed within 60 seconds after the blow.

Determination of unconsciousness of the seals following the head trauma was made by pinching or tugging at their flippers, making loud noises, touching the cornea or placing objects in the animal's mouth. The seals did not respond to these stimuli.[9]

MATERIALS AND METHODS

The skulls and brains of 19 animals which had been rendered unconscious by a single blow were examined. Members of the harvest crew had no prior knowledge of which animals were to be selected. Care was exercised to avoid damaging the heads during the skinning process.

The skin and muscles of the dorsal and lateral aspects of the head were dissected away. Lesions of the skull were noted. Following removal of the calvarium, the meninges and exposed surfaces of the brain were observed. Brains were removed, fixed in neutral buffered 10% formalin, and later dissected. In most cases the olfactory bulbs were not available, having been artifactually separated from the brain.

Tissue for histologic examination was selected from each brain, dehydrated in graded alcohols, embedded in paraffin, cut at 6μ and stained with hematoxylin and eosin (H&E). Certain sections were also stained by Bodian's nerve fiber and Mahon's myelin techniques. A seal which had been killed by a non-traumatic method served as a control.

RESULTS

Gross Lesions

Traumatic lesions were found in the skull and brain of all animals. Depressed comminuted fractures on the dorsal, dorso-lateral or postero-dorsal aspect of the skull were present in all seals. The animals were separated into two groups based on the fracture location. In seven animals, the primary site of these lesions was in the bones overlying the nasal cavities or adjacent structures rostral to the brain (Fig. 1). The remaining 12 animals had fractures primarily of the calvarium, overlying the brain (Fig. 2). These fractures are termed nasal and calvarial respectively in this paper.

The fractures were graded + or ++ on the basis of their size and the degree of downward displacement of bony fragments. In + lesions, the area of comminution measured up to 5 cm in greatest dimension and there was relatively slight downward displacement of bony fragments (Fig. 1). These + graded fractures were observed in seven animals (five nasal, two calvarial). Fractures graded ++ were more extensive, with major zone of comminution measuring 5 to 17 cm in greatest dimension, and greater downward depression of bony fragments (Fig. 2). The ++ lesions were noted in 12 animals (two nasal, ten calvarial). All fractures had linear extensions from the comminuted site, frequently seen as suture separations. In the ++ lesions these linear extensions sometimes reached the floor of the cranial cavity and the tentorium. The latter has a considerable osseous component in *C. ursinus*.[3]

Lesions in the meninges and brain were associated with these fractures (Table 1). Epidural hemorrhages were directly related to fractures overlying the dura, but were less extensive than those in other meningeal spaces. They were noted in 10 of 12 animals with calvarial fractures and two of the seven with nasal fractures. In the latter two animals, the fractures extended from the nasal region to the bony coverings of the cranial cavity. Subdural hemorrhages

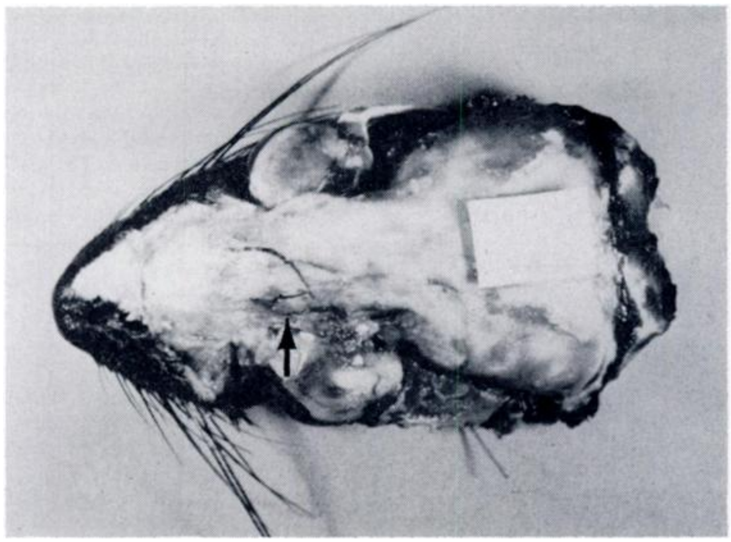

FIGURE 1. Dorsal surface of skull of seal showing + fracture over nasal cavity (arrow). The skin and muscles have been partially dissected away. Nose at left, occipital region at right (this type of preparation and its orientation also hold for Fig. 2).

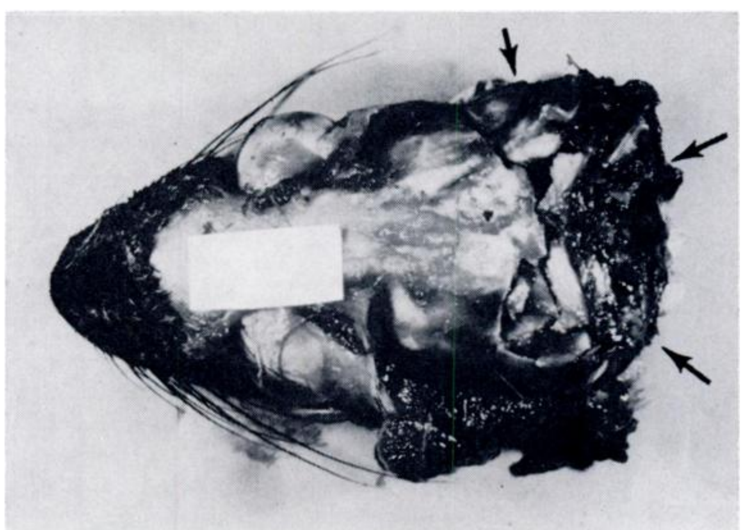

FIGURE 2. Dorsal surface of skull of seal with ++ fracture and associated defect in the posterior portion of calvarium (arrows). Note the extent of fracture and degree of depression of bony fragments.

were the most common meningeal lesions, being found in all 19 animals. These were of marked or moderate extent in all 12 seals with calvarial fractures and in four of seven with nasal fractures.

Subarachnoid hemorrhages were also common, prominent lesions (Figs. 3, 4). They were only absent in two animals, both of which had + nasal fractures. Although the subarachnoid hemorrhages were often diffuse, some pooling of blood at the base of the brain, especially in the midline, was common. Tears in the dura, with laceration of underlying cerebral cortex (Figs. 3, 4), were observed only in seven animals with ++ calvarial fractures. Separation of hemorrhage into specific meningeal compartments was difficult in these lacerated regions.

Gross lesions within the brain were considered under four categories: 1) lacerations (loss in continuity of brain surface); 2) superficial contusion (bruising of cortex and white matter without loss of surface continuity); 3) hemorrhage into deeper midline or paramedian structures; 4) intraventricular hemorrhage.

Lacerations and associated softening of the brain were only observed in animals with ++ calvarial fractures (eight of ten), usually related to severe depression of bony fragments and tears in overlying dura. These lacerations involved cerebral cortex and white matter in regions beneath or adjacent to fracture sites (Figs. 3, 4), while one animal had an associated mesencephalic laceration. The cerebellum was lacerated in three animals, in one of which this was the only such lesion.

Contusions of varying degree in the cortex and underlying white matter were common. Such lesions, represented by multiple small hemorrhages (Fig. 5), were observed in all seven animals with nasal fractures and 11 of 12 with calvarial fractures. Definitive patterns of distribution of these superficial lesions were difficult to evaluate. In general they were more extensive in regions directly beneath or adjacent to the fracture site, although other cortical areas were also involved. In some animals a few small hemorrhages were noted in the cortex of the medial, inferior, posterior aspect of the cerebrum, lying adjacent to the tentorial incisure. Cerebellar contusions most frequently involved the vermis and rostral aspect of the hemispheres.

Hemorrhages in deep midline or paramedian structures, such as diencephalon, basal ganglia and adjacent white

TABLE 1. Frequency of intracranial lesions associated with skull fractures in seals.

Location & severity of fracture	Meningeal lesions				Brain			
	Epidural hemorrhage	Subdural hemorrhage	Subarachnoid hemorrhage	Dural tears	Laceration	Superficial contusion	Deep midline or paramedian hemorrhage	Intra-ventricular hemorrhage
Nasal +	1/5	5/5	3/5*	0/5	0/5	5/5*	5/5*	0/5
Nasal ++	1/2	2/2	2/2	0/2	0/2	2/2	2/2	2/2
Calvarial +	1/2	2/2	2/2	0/2	0/2	2/2*	2/2*	1/2
Calvarial ++	9/10	10/10	10/10	7/10	8/10	9/10	9/10	9/10

Numerator—number of animals with the indicated meningeal or brain lesion. Denominator—number of animals with indicated fracture.

* Affected animals have a relatively high percentage (50% or more) of slight lesions.

matter, mesencephalon and/or pons, were surprisingly common and extensive in view of the relative distance of such structures from the site of trauma. These hemorrhages were multifocal and varied in size from pinpoint to 1 cm in diameter (Figs. 4, 5). They were noted in 18 of 19 animals, and were generally more extensive in those seals with ++ fractures. These lesions were bilateral in 17 seals (Fig. 5), but were sometimes more severe on the side of the fracture (Fig. 4). When the pons and the mesencephalon were affected, periaqueductal gray matter and the colliculi were often involved (Fig. 5). These deeper hemorrhages were minimal in five of the 18 seals in which they were demonstrated. Two of these five had calvarial fractures and the other three had nasal lesions. In the latter three animals these hemorrhages were restricted to the mesencephalon and/or pons.

Significant intraventricular hemorrhage was present in 12 of the 19 seals. It was seen in 10 animals with + or ++ calvarial fractures and two with ++ nasal lesions. In two animals with calvarial fractures cerebral laceration into a lateral ventricle probably caused the intraventricular hemorrhage (Fig. 4). Cerebral edema was not observed in this study.

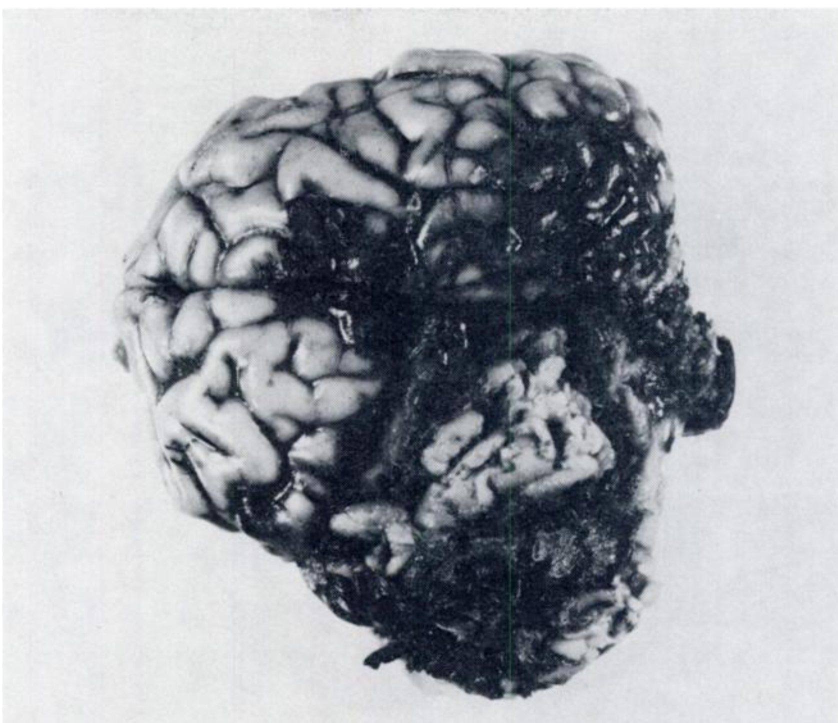

FIGURE 3. Dorsal surface of brain. The seal had a ++ calvarial fracture with laceration of underlying brain tissue. Masses of blood and traumatized brain tissue are present in the laceration in the left parieto-occipital region. There is extensive associated subarachnoid hemorrhage (frontal pole at left, medulla at right).

Light Microscopic Lesions

Microscopic study of the hemorrhagic regions of the brains revealed blood restricted to the perivascular space as well as within the parenchyma. Shrunken, dark staining neurons were noted adjacent to some hemorrhagic regions, but similar cells were also present in the control animal. In some superficial contusions, continuity between cortical hemorrhages and the surface of the brain was observed. Despite the presence of these "microlacerations", the gross classification of such lesions as contusions was maintained.

There was focal loss of ependyma, and in one animal this was noted in relationship to intraventricular hemorrhage. Displaced fragments of brain tissue were seen in some lacerations. Two animals had such fragments in their ventricular systems. Unclassified microfilaria in blood vessels and regions of hemorrhage were observed in five animals.

DISCUSSION

The seals in this study were subjected to a single blow to the head by a blunt instrument, a blow of sufficient force to cause unconsciousness.[9] Resulting lesions in the skulls and brains of these animals were related to forces initiated by the blow and acting upon craniocerebral tissues. It was of significance that the heads of these seals were not resticted in movement at the time of trauma (that is except for its cervical attachment, the head was able to freely move in response to the blow).

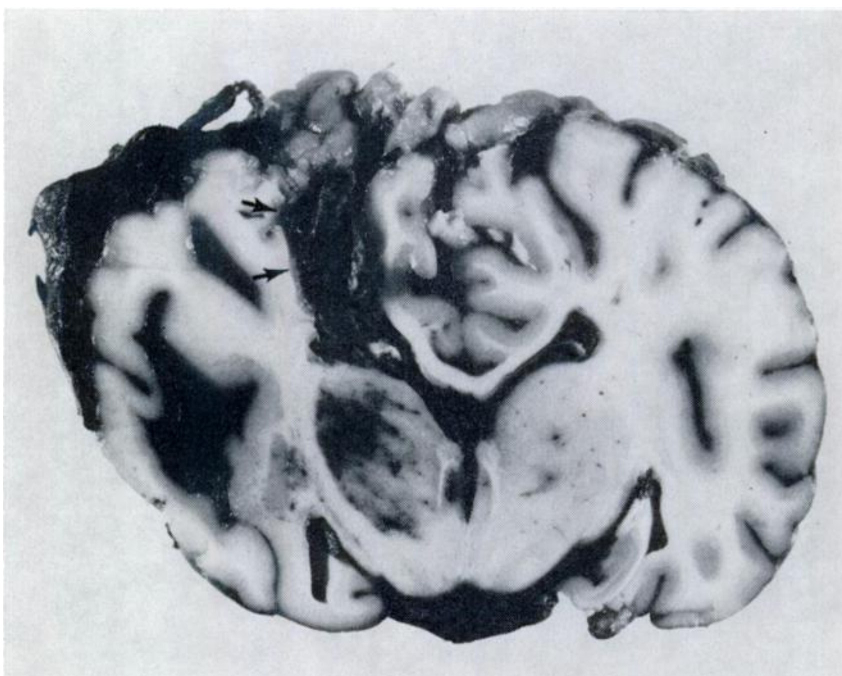

FIGURE 4. Coronal section of cerebrum of a seal which had a ++ calvarial fracture in the right parietal region. A laceration extends from dorsal surface of brain to right lateral ventricle (arrows). There is extensive intraventricular and subarachnoid hemorrhage. The right thalamus contains multiple hemorrhages.

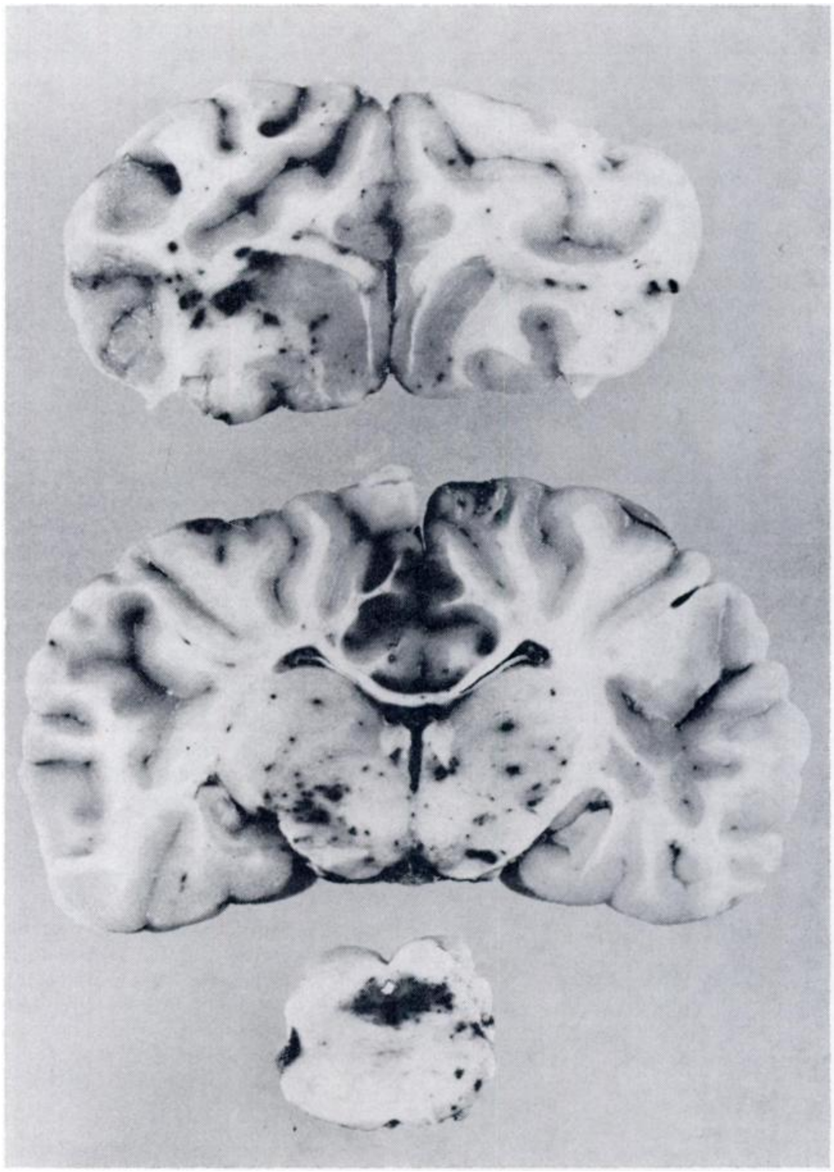

FIGURE 5. Three coronal sections of the brain of a seal which had a + nasal fracture. Multiple hemorrhages are prominent in cerebral cortex and white matter, thalamic region and periaqueductal gray matter.

Energy entering such a freely moveable head on impact is thought to be partitioned in three interrelated ways: 1) skull distortion; 2) rotation of the head; 3) translation (movement in a straight line) of the head.[9] The former two are thought to be of greater significance.[9] In the absence of fracture the initial distortion is followed by several oscillations of the affected regions of the skull,[10] but this is reduced if fracture occurs.[5]

Blunt trauma to a freely moveable head also initiates movements of the skull relative to the brain.[1,2,10] The latter often undergoes some rotational movements within the skull.[1,10] This asynchrony of movement can result in laceration and superficial contusion of the brain when its gliding movement is interfered with by bony prominences of the inner table of the cranial cavity, as in the anterior and middle fossae of man.[1,10] The traumatically-induced local bone deformation and discordant movement of skull and brain result in rapidly (0.04 to 1.3 milliseconds) oscillating negative and positive intracranial coup (directly below the trauma) and contrecoup (180° from the site of trauma) pressures, as noted in human cadaver studies.[2] Impact head injury subjects the intracranial contents to compression, tension (tearing apart of tissues) and shear (sliding of portions of tissues over one another).[4]

A significant traumatic lesion present in these seals was the depressed comminuted skull fracture observed in all animals. These were evidence of a blow of sufficient magnitude to cause excessive momentary indentation of the skull, an event leading to fracture.[10] The fracture location would seem to indicate that the primary site of impact in these seals was on the dorsal aspect (or portions of its periphery) of the nasal or calvarial regions of their skulls. Simpson[8] noted cranial fractures in 98.1% of the 1,121 northern fur seal carcasses she examined during the 1967 Pribilof Island harvest. Fractures of bone covering the brain can lead to increased direct brain injury, such as the lacerations of brain tissue seen in the seals in the present study.

Lesions of varying extent were present in the meninges and brains of all 19 animals subjected to the blunt head trauma. Although all seals were rendered unconscious, the animals with calvarial fractures generally had more severe meningeal and brain lesions than those with fractures over the nasal cavities, and more profound brain lesions were observed with more severe fractures.

Some of the intracranial gross lesions observed in these animals, such as meningeal hemorrhages, dural tears, superficial contusions and lacerations of brain tissue are not surprising when one considers the apparent energy of the causal blunt trauma and extent of skull fracture. The presence of multiple, frequently bilateral hemorrhages in midline or paramedian structures, such as diencephalon, basal ganglia, white matter, mesencephalon and pons, deserves additional comment. Lindenberg and Freytag[5] noted similar hemorrhages in some deep structures of the human brain, associated with depressed skull fracture. They indicated these lesions were caused by shearing force in such regions generated by a mass shifting of the brain predominantly along the line of impact.

A specific potential source of intraventricular hemorrhage, laceration extending from the cerebral cortical surface to the lateral ventricle, was noted in two animals. In the remaining seals with intraventricular bleeding, the sources were probably either direct traumatic hemorrhage from the choroid plexus or leakage from periventricular sites.

Acknowledgements

I am indebted to Drs. C. R. Smith, W. V. Lumb, L. E. McDonald, A. F. Moreland and W. M. Wass, my colleagues on the veterinary panel which evaluated humane aspects of the northern fur seal harvest, for their interest, encouragement and assistance in this study. I also appreciated the cooperation of Dr. M. Keyes of the National Oceanographic and Atmospheric Administration. This study was supported in part by the National Oceanographic and Atmospheric Administration, U.S. Department of Commerce and by a general research support grant from the College of Medicine and Dentistry of New Jersey (DHEW GRS SO1 05393).

LITERATURE CITED

1. ANGRIST, A. A. 1955. Traumatic intracranial hemorrhage: some broad pragmatic and theoretical considerations. Jew. Mem. Hosp. Bull. (N.Y.) 1: 3-11.
2. EDBERG, S. and A. ANGRIST. 1967. Intracranial pressure changes following impact of human cadaver heads. I. Brain surface measurements. J. Forens. Sci. 12: 60-67.
3. FISH, P. A. 1898. The brain of the fur seal Callorhinus ursinus; with a comparative description of those of Zalophus californianus, Phoca vitulina, Ursus americanus and Monachus tropicalis. J. Comp. Neurol. 8: 57-91.
4. GURDJIAN, E. S., L. M. THOMAS, V. R. HODGSON and L. M. PATRICK. 1968. Impact head injury. GP 37 (No. 2): 78-87.
5. LINDENBERG, R. and E. FREYTAG. 1960. The mechanism of cerebral contusions. A pathologic-anatomic study. Arch. Path. 69: 440-469.
6. OMMAYA, A. K., R. L. GRUBB, Jr. and R. A. NAUMANN. 1971. Coup and contrecoup injury: observations on the mechanics of visible brain injuries in the rhesus monkey. J. Neurosurg. 35: 503-516.
7. ROPPEL, A. Y. and S. P. DAVEY. 1965. Evolution of fur seal management on the Pribilof Islands. J. Wildl. Manag. 29: 448-463.
8. SIMPSON, E. 1967. More humane way with seals. Nature 216: 1237-1238.
9. SMITH, C. R., B. S. JORTNER, W. V. LUMB, L. E. McDONALD, A. F. MORELAND and W. M. WASS. 1972. Report of the veterinary panel evaluating humaneness of the northern fur seal harvest in the Pribilof Islands. Pages 456-471 *in* the Ocean Mammal Protection Hearings before the Subcommittee on Oceans and Atmosphere of the Committee on Commerce, United States Senate, 92nd Congress, 2nd session, Serial no. 92-56.
10. TOMLINSON, B. E. 1964. Pathology. Pages 93-158 *in* Rowbotham, G. F. ed. *Acute Injuries of the Head. Their Diagnosis, Treatment, Complications and Sequels.* 4th ed. E.&S. Livingstone, Edinburgh/London.

Received for publication 21 August 1973