CEREBRAL AND CEREBELLAR INVOLVEMENT OF TREMATODE PARASITES IN DOLPHINS AND THEIR POSSIBLE ROLE IN STRANDING

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CEREBRAL AND CEREBELLAR INVOLVEMENT OF TREMATODE PARASITES IN DOLPHINS AND THEIR POSSIBLE ROLE IN STRANDING

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Abstract: Seven dolphins (*Delphinus* sp.) that were found stranded near Point Mugu, California between 1966 and 1970 were given complete necropsy examinations. In all seven cases a similar pathological picture was observed. The findings included adult trematodes in the bile and pancreatic ducts, severe liver damage, and massive brain necrosis due to the presence of numerous trematode ova in the brain tissue. We suggest that the stranding and subsequent death of all seven animals resulted from this disease.

INTRODUCTION

From time to time various cetaceans are found on beaches in most parts of the world. A single individual is more frequently encountered but at times there may be a large group. Such cetaceans are generally termed stranded animals. Often the specimen is found alive and well-meaning individuals will sometimes exert considerable personal energy in towing the beast out to sea, only to have it come back on shore again.

Aristotle[1] about 2,300 years ago apparently observed that dolphins were air-breathing mammals quite different from the fish of the sea. In addition, he was probably the first to publish a report on stranding: "It is not known why they sometimes run aground on the sea shore; for it is asserted that this happens rather frequently when the fancy takes them and without any apparent reason." Even today strandings have still not been explained to everyone's satisfaction. A complete review of the literature on the stranding phenomenon is beyond the scope of this paper, but a few of the more recent theories and findings should be mentioned.

At the First International Symposium on Cetacean Research in 1963*, Dudok Van Heel[4] advanced a theory that strandings are the result of the animal having its echolocation system fail because of the acoustic conditions near a sloping, sandy beach. He suggested that sloping, sandy beaches would reflect echoes less efficiently than rocky shoreline. Thus the animals blunder onto the beach because they do not know where the shore is. Backus and Schevill (quoted by Norris[14]) questioned the acoustic basis of Dudok Van Heel's theory.

Breland and Breland[2]** advanced a more comprehensive theory that took into consideration the idea that cetaceans evolved from land-dwelling mammals. The ancestors of the Cetacea probably lived along the shore, and at first might well have retreated to the beach if danger

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**This theory was first advanced by Keller Breland and F. G. Wood at a Symposium held at the University of Florida in 1961.

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threatened from the sea. Thus if an animal or animals were badly frightened or perhaps ill, they might have fled to what was for their ancestors a safe retreat.

Wood (quoted in Norris[11]) mentioned several cases of stranded animals that he had observed. He pointed out that the animals almost invariably came back to shore and refused to accept freedom when they were pushed back into the water. When the animals were put into tanks after stranding they thrashed against the wall as if trying to get out of the water.

Disease is also thought to play a part, especially when lone individuals are found stranded. Fraser, quoted in Norris,[14] pointed out that the pterygoid sinuses of cetaceans are frequently infested with nematodes and that their skulls very often show evidence of inflammation and abscesses. He suggested that as a result of the isolation of the essential organs of hearing the animal might lose its sense of direction and run aground. Reysenbach De Haan[10] and Caldwell and Caldwell[3] also mention parasites found in the middle ear cavities of singly stranded cetaceans.

Ridgway (quoted in Norris[11]) reported finding a stranded Pacific common dolphin, *Delphinus bairdi* and upon examination of the brain found large necrotic areas containing unidentified ova. This case, including histopathology, was more fully reported by Ridgway and Johnston.[19] Norris[11] found about half a handful of flukes around the spinal cord at the foramen magnum of a stranded pilot whale.

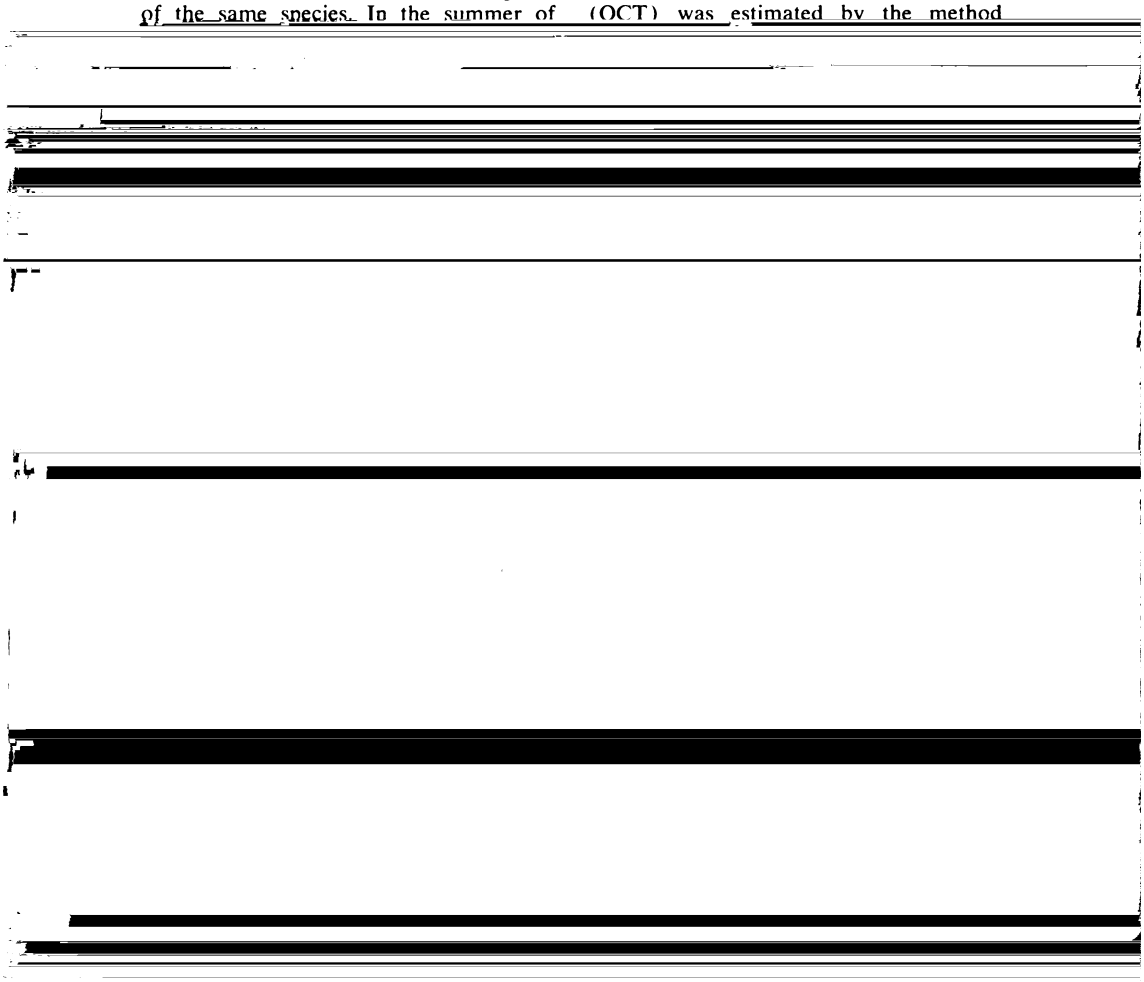
MATERIALS AND METHODS

Between August 1966 and late July 1970 we collected seven common dolphins (Fig. 1) that came to the Los

FIGURE 1. Stranded **Delphinus** sp. at Oxnard, California.

Angeles and Ventura County beaches within 15 miles to the north and about 12 miles to the south of Point Mugu, California. Although a female of the same species was collected in 1963,[19] the seven animals that we examined were males. Judging from the body lengths and testis weights (400-1000 gms) six of the seven dolphins were mature. Case 8 was a female Pacific white-striped dolphin *Lagenorhynchus obliquidens* that died suddenly about 2 weeks after being captured at sea with four other dolphins of the same species. In the summer of

Three of the stranded dolphins were recovered from the beach alive and blood was taken for analysis. The blood was collected from the central vessels on the ventral aspect of the tail fluke.[17] Three normal dolphins captured at sea were also sampled. A 30 ml heparinized disposable plastic syringe was used for collection. After removal of a sample for hematology the remainder was centrifuged (1,000 to 1,500 r.p.m. in a 25 cm centrifuge) to separate plasma from cells. Plasma ornithine carbamyl transferase (OCT) was estimated by the method



common dolphins caught at sea for a study of comparative cephalization.[18] In the summer of 1970, three dolphins were captured for normal blood studies and two of them were used for circulatory tests. In June 1971, eight dolphins were examined from a group caught at sea. In all, the brains of 14 newly captured dolphins were examined for comparison

moles of carbon dioxide. Plasma glutamic pyruvic transaminase (GPT) was estimated by the Reitman-Frankel method and reported in Henry units.[5] Plasma glutamic oxalacetic transaminase (GOT) activity was estimated by an autoanalyzer and reported in Henry units.[11] Bilirubin was estimated by the standard technique and reported in mg/100 ml. The hemo-

the animal swam back and forth just outside the zone of breaking surf. Finally it got into the breakers, and a large wave simply rolled the animal on shore much as a log or piece of driftwood would come in. One of these dolphins survived overnight in captivity and the other survived for 2 days. In both of these cases the animals swam aimlessly and were rolled on shore. They did not appear to swim actively ashore and they were not left high and dry by an outgoing tide as the word stranding suggests. The term "cast ashore" as sometimes employed by Fraser,[6,7,8] and by Nishiwaki[13] is probably more appropriate to these strandings.

There are three additional cases of direct observation of strandings of captive or domesticated cetaceans. In 1966 a large, old, male Pacific bottlenose dolphin *(Tursiops gilli)* that had been in captivity in land based pools at Point Mugu for about 9 months was released in Mugu Lagoon. The Lagoon is a natural stretch of ocean water over a mile long and several hundred yards wide that opens to the Pacific Ocean through a narrow inlet. The second day after the dolphin was released it was found stuck in a shallow mud flat at one end of the lagoon in water 25 cm deep. It was pulled free and turned into deep water where it remained for another 24 hours, after which it beached itself on fairly rocky shore adjacent to the pools from which it had been taken. The dolphin was then transported back to one of the pools. The second event involved a highly trained female Pacific white-striped dolphin. It had been captured in 1964 about 5 miles off Point Mugu. In 1968 and early 1969 it was being trained for diving experiments that required it to be completely free in the open ocean. During one of the training sesions, about half a mile off shore, it abruptly left the trainer's boat and very rapidly swam for shore and somewhat to the trainer's amazement swam right up to the beach. The trainer radioed the Marine Bioscience Facility and a truck was dispatched to retrieve the dolphin from the beach. She was simply dusted off and put in a pool. Subsequent examination and blood tests indicated that she was in normal health. The only explanation that the trainer could give was that he had seen some migrating gray whales nearby and he thought that perhaps his dolphin had been frightened by one of them.[9]

The third instance occurred in a fenced lagoon where dolphins and small whales are kept for an oceanarium show. A pilot whale was given a sedative injection and it later beached itself on a small sand beach at one end of the lagoon enclosure (D. Kenney, Personal comm., 1965).

All of the stranded dolphins that we found were examined by necropsy as soon as possible after death save two which were omitted from our list (Table 1) because neither brain lesions nor trematodes were demonstrated. One animal was found after it had apparently been on the beach for at least several weeks. The other was found dead on the

TABLE 1. Dolphins that were stranded near Point Mugu and given complete necropsy.

	Date stranded	Sex	Length	Weight	Remarks
Case no. 1	8 August 1966	♂	213 cm	90 kg	Died on beach
Case no. 2	12 April 1967	♂	194 cm	76 kg	Died on beach
Case no. 3	1 August 1967	♂	162 cm	40 kg	Died on beach
Case no. 4	26 July 1969	♂	210 cm	85 kg	Lived 36 hours in tank
Case no. 5	5 March 1970	♂	229 cm	105 kg	Died on beach
Case no. 6	20 April 1970	♂	191 cm	77 kg	Lived 24 hours in tank
Case no. 7	10 July 1970	♂	196 cm	67 kg	Lived about 10 hours

beach and stored in a freezer for about 6 months prior to examination. Autolysis was too advanced for an adequate examination of the brain. In neither of these cases could the presence of trematodes or ova be confirmed or denied. The post mortem results were similar in the remaining seven of *Delphinus* sp. cases (Table 1). There was a generalized jaundiced appearance that was especially apparent in cases 1, 4, 6, and 7. On gross examination the liver appeared congested and slightly swollen. Liver section revealed moderate to severe fibrosis surrounding the bile ducts. Adult flukes were found in the bile ducts of case 1 and in both the bile and pancreatic ducts of cases 4 through 7. In cases 2 and 3 the adult flukes were either absent at the time or necropsy or overlooked.

Adult flukes from the liver and pancreatic ducts were identified as *Campula rochebruni*. Ova from the brain and ova within the adult flukes were found to be identical.

The mesenteric lymph nodes in the region of the pancreas and liver showed a mottled or peppered appearance with black dots and splotches apparent over their surface. Slicing the nodes revealed these same black spots throughout. On one occasion similar black spots were also seen in thoracic lymph nodes.

In some cases large dark areas (Fig. 2) were apparent on the surface of the cerebrum and/or cerebellum. Slicing the brain into 1 cm sections revealed numerout dark areas ranging in diameter from 0.1-0.4 cm. Lesions were most numerous in the cerebellum and in the posterior half of the cerebral hemisphere. No lesions were found in the anterior one-third of the cerebrum.

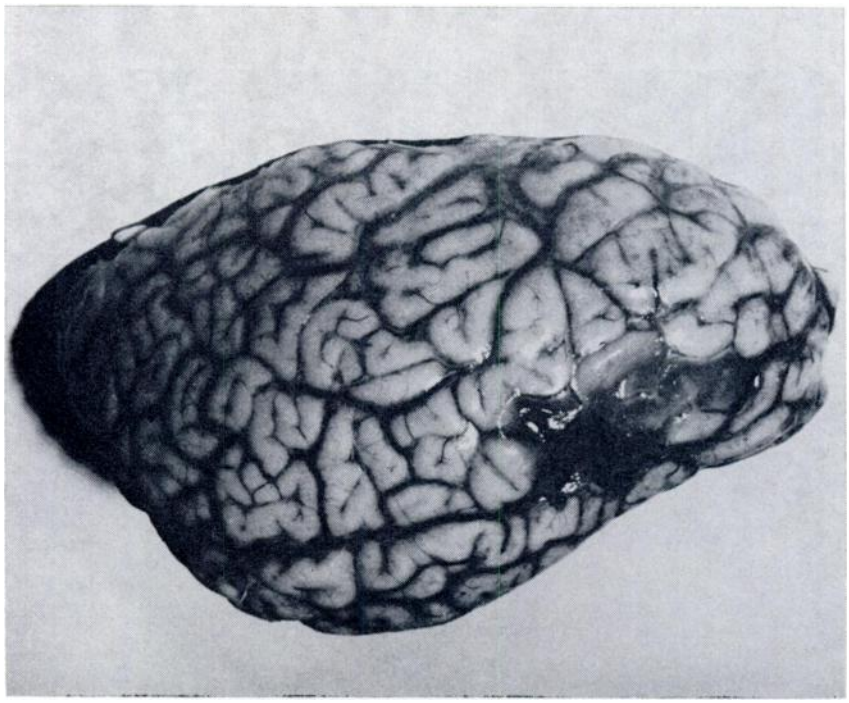


FIGURE 2. Gross view of a cerebral hemisphere of one of the common dolphins showing a dark necrotic area containing numerous aggregations of ova.

Histopathologic examination of sections of the cerebral and cerebellar lesions revealed areas of focal necrosis showing extensive liquefaction with numerous polymorphonuclear leucocytes and large foamy cells resembling macrophages. Often there was focal hemorrhage and perivascular lymphocytic and polymorphonuclear leucocytic infiltration in the adjacent brain tissue. The most striking finding in the necrotic areas was numerous yellow to golden brown triangular-shaped ova (Fig. 3). On the periphery of the necrotic areas were numerous hemosiderin-laden macrophages and Hortega cells that appeared to be cleaning up debris. There was gliosis and perivascular cuffing by lymphocytes. Some of the astrocytes were large with deep acidophilic cytoplasm. Cholesterol clefts were sometimes seen as a by-product of fatty necrosis that resulted from the presence of large numbers of ova (Fig. 4).

The livers had prominent portal fibrosis with large numbers of ova often found in the fibrotic areas. The fibrotic areas also contained focal lymphocytic aggregations. Biliary hyperplasia was very prominent. Most of the remaining hepatic parenchyma, however, was normal.

In the pancreas there was hyperplasia of the pancreatic duct. Other pathologic features included interlobular fibrosis, limited inflammatory cell infiltration and areas of lobular necrosis. Generally the lesions involved no more than 25% of the total gland and probably adequate amounts of viable pancreatic parenchyma remained.

The mesenteric lymph nodes near the pancreas coalesced, forming a large mass. The node capsules were thickened and contained foci of pigment deposition adjacent to cortical sinuses. This pigment was also seen within macrophages and in intercellular spaces. The pigment was dark golden brown — apparently hemosiderin. This was the apparent cause of the black dotted or peppered appearance

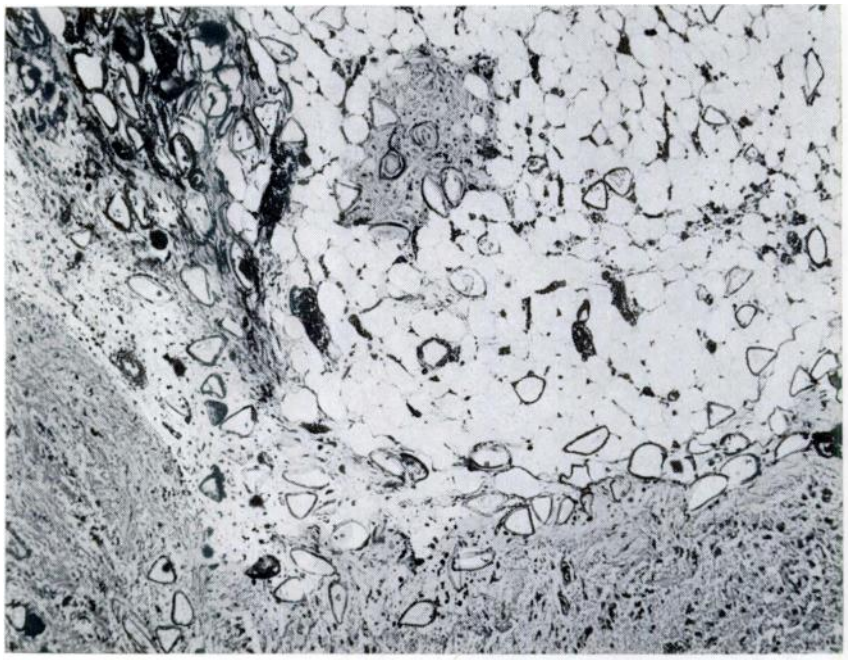


FIGURE 3. Ova in the brain of a common dolphin with attendant fatty necrosis. (x 75).

of the lymph nodes observed on gross examination.

Case 8 was a 95kg female *Lagenorhynchus obliquidens* that was captured in late December, 1968 along with four other animals of the same species. This particular animal adapted very rapidly to taking food from the keeper's hand. It would also come to the side of the pool and allow the attendant to stroke or rub it. This behavior is not usually shown until the dolphin has been in captivity for several months. One day, about 2 weeks after capture, this dolphin suddenly began showing signs of incoordination and tended to list to one side. It died during the night.

Necropsy revealed numerous necrotic brain lesions with massive numbers of ova scattered throughout the cerebrum and cerebellum. In this case the ova were round in cross-section rather than triangular (Fig. 5). No trematodes have been reported from this species of dolphin. Since the ova were round in cross-section, it is suspected that they are from the genus *Zalophotrema,* the only member of the family Campulidae with ova of this type.

In cases 4, 6, and 7, the animals reached the Marine Bioscience Facility alive. Blood was taken for hematology and blood chemistry. The values are presented in Table 2. In each of these cases the plasma appeared icteric. The plasma enzyme and bilirubin values for the stranded dolphins are high compared with values from three dolphins that were sampled soon after capture at sea. The elevated plasma values no doubt reflect the liver lesions that were observed. Hematology values were different only in the differential analysis. Segmented neutrophils were increased in the stranded animals and the other cells were decreased (Table 2).

FIGURE 4. Showing cholesterol clefts and ova in the brain of a common dolphin. (x 75).

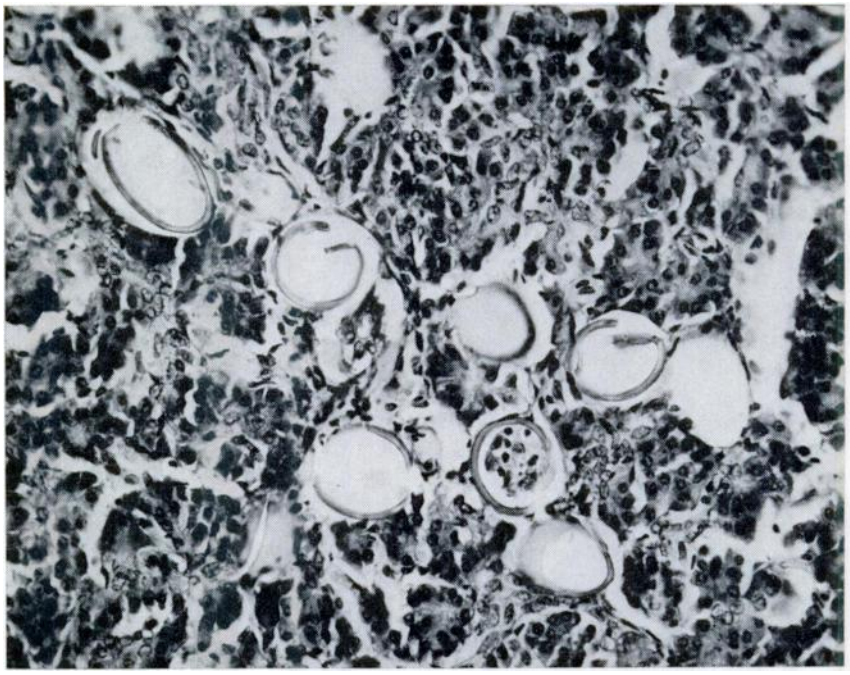


FIGURE 5. Section of the white-striped dolphin's brain showing trematode ova that are round in cross-section. (x 250).

TABLE 2. Blood chemistry values for normal and stranded common dolphins (**Delphinus** sp.)

	OCT	GPT	GOT	Bilirubin
Three normal dolphins captured at sea	5	94±60	184±46	0.3±0.2
Case #4	325	520	1600	4.3
Case #6	801	680	2280	4.8
Case #7	105	1120	1350	5.0

Hematology of Normal and Stranded Common Delphins

	WBC	PCV	Seg	Stab	Lymph	Mono	Eos	Baso
Three normal dolphins captured at sea	6.8±9	50±3	63±5	2±1	25±4	4±1	5±2	1
Case #4	6.3	48	85	—	14	1	—	—
Case #6	6.9	50	84	—	14	2	—	—
Case #7	7.9	51	90	—	9	1	—	—

* Abbreviations:
OCT = ornithine carbamyl transferase
GPT = glutamic pyruvic transaminase
GOT = glutamic oxalacetic trensaminase
WBC = white blood cells
PCV = packed cell volume
Seg. = segmented cells
Stab = Stab cells
Lymph. = lymphocytes
Mono. = monocytes
Eos. = eosinophils
Baso. = basophils

Case 7 had extensive bite marks over the trunk of the body and the tail (Fig. 6). Casual observers would probably have concluded that this animal's stranding was the result of a shark attack. However, when a blood sample was drawn, the rapid sedimentation rate and yellow color of the plasma gave a clue that there were more important reasons. Adult flukes were found in the bile and pancreatic ducts as well as numerous necrotic foci in the cerebrum and cerebellum.

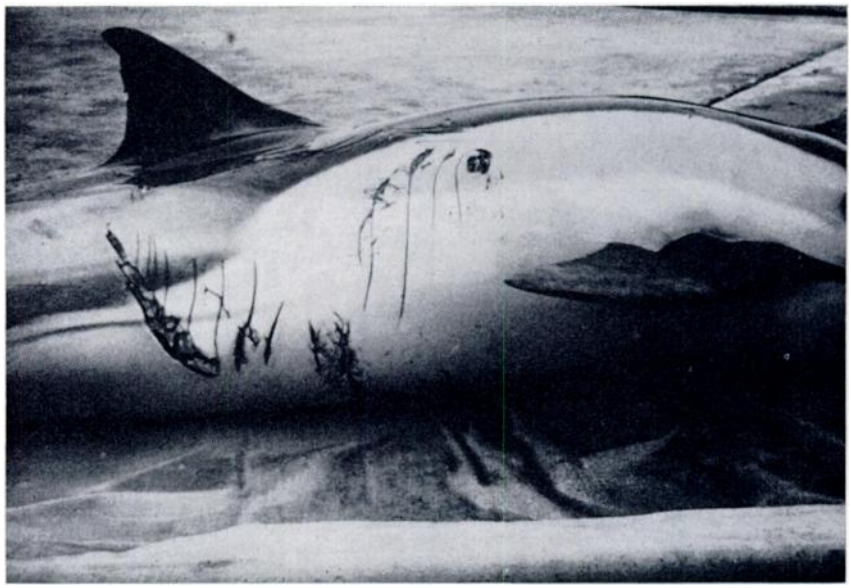


FIGURE 6. Case 7, a common dolphin had been attacked by a large shark. The bite marks were probably produced by a large mako **(Isurus oxyrhynchus)** since the lesions produced are in accord with the shape and spacing of the mako's teeth.

DISCUSSION

Woodward et al.[22] reported finding trematode ova in areas of cerebral necrosis in an Amazon river dolphin *(Inia geoffrensis)*. This animal was unable to swim and floated with its left side up. In this case the adult flukes *Hunterotrema caballeroi* were found in the major bronchi of the lung. Again it was not clear how the ova were transported to the brain. Woodard et al.[22] suggest that the ova could have been deposited in the brain tissue by a wandering fluke or they could have been deposited in the circulation at some distant point (lung). Our studies with microspheres of approximately the same size as the ova indicate that if the ova were placed in the blood stream near the liver or pancreas they would end up in the lungs rather than in the brain. Ova deposited in a pulmonary vessel would be in a better position to get into the brain.

Wood et al.[21] have noted that commercial fishermen along the Florida coast sometimes refer to porpoises and sharks as the "dogs and cats of the sea", and tell of seeing battles between the two, with blood staining the sea red. As Wood et al.[21] suggest, many of these shark-porpoise battles may have involved sick animals such as our case 7.

There appear to be four possible explanations for the presence of ova in the brain, but we do not have enough evidence to prove any of these. First, the migrating adult flukes could go into the brain tissue, lay their eggs, and leave or die and become absorbed. Despite careful gross examination of the entire brain no adult flukes have been found. Microscopic examination of numerous affected areas has failed to reveal any extensive tracts like those produced by other parasites migrating through the central nervous system,[15] although in some instances the ova appear to be lined up as if deposited by an adult fluke migrating through. Second, the ova could somehow get into the circulation after being shed by adults in the liver or pancreas and could be carried to the brain by the circulation, in some way bypassing the lungs. The structure of the circulation is altered somewhat during diving.[10] Dolphin's lungs collapse during deep diving.[20] Thus it appears possible that circulatory changes occurring during the rapid pressure changes of a deep dive in the ocean might allow ova to get to the brain via the blood or lymphatic circulation. We have occasionally found ova in blood vessels of the brain although this could represent a processing artifact. Thirdly, the eggs could be shed by adults in the liver or pancreas and by some means get into the lymphatic vessels, and be carried to some point in the circulatory system that allows them to bypass the lungs. Lastly, the cerebrospinal fluid could be involved in transmission of the ova to the brain.

At least three species of trematodes parasitic in dolphins shed ova which in some unknown way get to the brain, causing extensive damage. This damage leads in incoordination and eventual death. Effected animals that survive long enough are cast ashore or become stranded. Stranded cetaceans, whether found singly or in a group, should be examined for disease. The necropsy should include an investigation of the ears and brain.

Acknowledgements

We thank Mr. W. G. Gilmartin for supervising the circulatory tests with the Sr 85 microspheres and for other technical assistance. Dr. John G. Simpson reviewed the histopathology and made many helpful comments. Mr. F. G. Wood reviewed the manuscript and gave us the suggestion regarding the species of shark that attacked our case 7.

LITERATURE CITED

1. ARISTOTLE. 1883. *Histoire Des Animaux,* Librairie Hachette, Paris, Tome 3 (Translated in French.)
2. BRELAND, K., and M. BRELAND. 1968. *Animal Behavior,* MacMillan, New York, pp. 82-83.
3. CALDWELL, M. C., and D. K. CALDWELL. 1971. Senses and Communication in *Mammals of the sea: Biology and Medicine,* S. H. Ridgway Ed. Thomas, Springfield, p. 485.
4. DUDOK VAN HEEL, W. H. 1966. Navigation in Cetacea in *Whales, Dolphins and Porpoises,* K. S. Norris Ed., Univ. of California Press, Berkeley, pp. 596-602.
5. FRANKEL, S., and S. REITMAN. 1963. *Gradwhol's Clinical Laboratory Methods and Diagnosis,* 6th ed., C. V. Mosby, St. Louis.
6. FRASER, F. C. 1934. Report on Cetacea stranded on British coasts from 1927-1932. British Mus. Nat. Hist., 11: 1-41.
7. FRASER, F. C. 1946. Report on Cetacea stranded on British coasts from 1933-1937. British Mus. Nat. Hist., 12: 1-56.
8. FRASER, F. C. 1953. Report on Cetacea stranded on British coasts from 1938-1947. British Mus. Nat. Hist., 13: 1-48.

9. HALL, J. D. 1970. Conditioning Pacific white-striped dolphins, *Lagenorhynchus obliquidens,* for open-ocean release. Naval Undersea Center, San Diego, California, Technical Paper No. 200.

10. HARRISON, R. J., and J. D. W. TOMLINSON. 1956. Observations on the venous system in certain Pinnipedia and Cetacea. *Proc. Zool. Soc. Lond.,* 126: 205-233.

11. HENRY, R. J. 1964. *Clinical Chemistry: Principals and Techniques,* Harper and Row, New York.

12. HENRY, R. J. 1967. *Handbook of Specialized Laboratory Tests,* 7th ed., Bioscience Laboratories, Van Nuys, California.

13. NISHIWAKI, M. 1971. General biology in *Mammals of the Sea: Biology and Medicine,* S. H. Ridgway Ed., Thomas, Springfield.

14. NORRIS, K. S. (Ed.). 1966. *Whales, Dolphins and Porpoises,* Univ. of California Press, Berkeley, p. 602-606.

15. NYE, S. W., P. TANGCHAI, and S. PUNYAGUPTA. 1970. Lesions of the brain in eosinophilic meningitis. Arch. Path., 89: 9-19.

16. REYSENBACH DE HANN, F. W. 1957. Hearing in whales. Acta Oto-Laryngol. suppl. 134: 1-114.

17. RIDGWAY, S. H. 1965. Medical care of marine mammals. J. Amer. Vet. Med. Assoc., 147: 1077-1085.

18. RIDGWAY, S. H., N. J. FLANIGAN, and J. G. McCORMICK. 1966. Brain-spinal cord ratios in porpoises: possible correlations with intelligence and ecology. Psychon. Sci. 6: 491-492.

19. RIDGWAY, S. H., and D. G. JOHNSTON. 1965. Two interesting disease cases in wild cetaceans. Amer. J. Vet. Res., 26: 771-775.

20. RIDGWAY, S. H., B. L. SCRONCE, and J. KANWISHER. 1969. Respiration and deep diving in a bottlenose porpoise. Science, 166: 1651-1654.

21. WOOD, F. G., D. K. CALDWELL, and M. C. CALDWELL. 1970. Behavioral interactions between porpoises and sharks. In *Investigations on Cetacea,* G. Pilleri Ed., Waldau, Berne, Switzerland, 2: 264-279.

22. WOODARD, J. C., S. G. ZAM, D. K. CALDWELL, and M. C. CALDWELL. 1969. Path. Vet., 6: 257-272.

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