GA 810

Daniel F. Cowan, M. D.
Professor of Pathology
The University of Texas Medical Branch

This animal (GA 810), a female *Stenella attenuata* weighing 64.7 Kg and measuring 191 cm long, was recovered alive 3.5 miles west of San Luis Pass, Brazoria county on April 21, 1996. She died in truck on the way to the laboratory at about 2:30 PM.

External examination: In general, external appearance was normal, with no obvious weight loss. There were several cookie cutter shark bites, some old and scarred, and some recent and raw. There was a notch in the dorsal fin, and another in the dorsal ridge of the tail stock, both well healed. 12 *Xenobalanus* were attached to the flukes.

Internal examination: The air sinuses contained a relatively small number of flukes, typical of *Nasitrema*. Many small and very large (up to 3 x 3 x 7 cm) parasitic cysts were present in the abdomen under the peritoneum, especially deep in the pelvic recess, and very many in the blubber near the genital area. One was present in the chest, near the heart.

There were many small nodules of the kind produced by nematode lung worm in the lungs, along with patchy hemorrhage and a small abscess in the left lung. The remaining organs were all normal. The uterus and ovaries were small with no ovarian corpora, suggesting that this was an immature animal.

The brain was swollen, with flattening of the folds of the surface of the brain, and grooving of the surface by bony ridges of the skull. Since the skull is a closed box, even a relatively small amount of swelling causes compression of the brain,

with impairment of circulation through it. In the right cerebrum, where the superior and medial surfaces meet, and along the medial surface, the gray matter was slightly brownish, with a focus of softening. The meninges were a little cloudy especially along the tracks of the vessels, suggesting non-suppurative (non-bacterial) meningitis. This change was caused by a focus of necrosis, producing collapse of the brain with gliosis, the brain equivalent of a scar. Deep in the cerebellum was a focus of hemorrhage, in a peculiar pattern of twisted tracts, with larger areas of bleeding. Microscopic examination revealed worm ova in these areas of hemorrhage. While no adult worms were identified in the cerebellum, the lesion and the ova are typical of *Nasitrema*. We attributed stranding to this brain damage; a complication of parasitism of the air sinuses.

Comment: Some years ago we reported a study of 50- odd beach stranded dolphins from southern California. In contrast to our experience here, the majority of those animals were *Delphinus*, and most had brain lesions identical to those we found in this animal. We have examined the brains of more than 50 beached dolphins here, nearly all *Tursiops*, none of which had brain parasitism. However, the three *Stenellas* in our local group all had these brain lesions. Certainly our bottlenose dolphins do not lack *Nasitrema*. Nearly all have at least a few, and some have very many. How can we explain the presence of brain involvement in some animals, but not others? This is an interesting question, and we might speculate that the different dolphin species have different species of *Nasitrema*, one of which likes to burrow in brain, and one that doesn't. No one in our group is expert enough to speciate marine mammal flukes, so we can't answer that question. Another explanation might be that the worm is the same, but that features of the dolphin determine brain involvement. We can't answer that one either. What we do know is that in *Delphinus* caught in the open Pacific Ocean (studies done on by-catch animals in the fishing industry) we almost always found worms in the sinus of *Delphinus*, but never in the brain. So there is a difference in the in-shore animals that are likely to strand, and the off-shore

animals of the same species. Some of the lesions are in functionally "silent" areas of the brain, and are survivable, while others are in vital areas, as in the animal presented here. Why didn't off-shore *Delphinus* have old lesions? That is on our long list of things we speculate about, but don't have an answer for. One thing we do know is that it is not in the worm's best interest to get into the brain. It is a dead-end. The ova die. It is wasted effort on the part of the worm, an abnormal self-destructive act. Infestation of the brain doesn't correlate well with the density of the worms in the sinuses. It is not "population pressure" causing migration. I think the poor worm gets confused, and perhaps stimulated by an urge to deposit eggs, begins to wander. It creeps along the acoustic nerve from the ear bone, which is in communication with the air sinus, into the fluid-filled space (subarachnoid space) around the brain. It picks a likely spot on the surface, and burrows in deep, twisting, turning and laying eggs as it goes. This tears the brain, producing those contorted blood filled galleries in the acute lesion, and necrosis, collapse and gliosis in the old ones. The other thing we know is that the California dolphins have some of the heaviest loads of pesticide recorded from animal tissues. Perhaps the toxin makes the worm sick, and in its confusion it migrates into the brain. That doesn't tell us why our Gulf *Stenella* have brain involvement, but the *Tursiops* don't.

I would appreciate any ideas or comment on this most interesting problem in biology. I can be reached by E-mail at dcowan@UTMB.edu or by snail mail at Department of Pathology, UTMB, Galveston 77555-0588.