CC 186

Daniel F. Cowan, M. D.
Professor of Pathology
The University of Texas Medical Branch
dcowan@utmb.edu

CC 186 was a 145 cm male bottlenose dolphin, *Tursiops truncatus*, estimated to be about 2 years old. He was found stranded April 7, 2002 near the 33 mile marker, Padre Island National Seashore (Kenedy County) by USGS personnel. It was reported that he had stranded earlier at the 44 mile marker, but had been pushed back into the water by observers. At the time of retrieval, he weighed 65 kg. He had several wounds attributed to a boat propeller, as well as small shark bites. He was taken to the Texas State Aquarium for care and rehabilitation.

He was named ‘Corky’. Initial blood tests suggested infection as well as dehydration, and he was put on antibiotics and vitamins, as well as fluid replacement. He appeared to improve rapidly over the next several weeks with good weight gain and growth. Serological testing for morbillivirus, the cause of dolphin distemper, was negative. His progress was such that discussions were begun with National Marine Fisheries to plan for his release. To all observers he was healthy and active, and seemed able to cope with life in the wild. Then rather abruptly his condition began to deteriorate, and after just a few days of overt illness, he died on June 4. At the time of necropsy on June 5, he weighed 87.8 kg, and measured 190 cm.

On external examination, his wounds were nearly completely healed. His lungs and airways contained foam, suggesting heart failure, and the smaller airways were full of mucus and tissue debris. Several areas of inflammation typical of mild bronchopneumonia were present. The first approximately 10 feet of the intestine were contracted, and a further segment was dilated, discolored and thinned. The wall of the intestine was in part ulcerated, and very thinned, almost

perforated. The appearance was consistent with severe impairment of the local blood supply. The muscle fibers of the stomach, small intestine and urinary bladder were in spasm. The pancreas showed evidence of mild chronic inflammation and scarring. Several lymph nodes in the chest had microabscesses indicating bacterial infection. The most striking findings were chronic meningitis, with enlargement of the normally thin ventricles of the brain (hydrocephalus). A few parasites were found in the lungs and in the stomach.

I think that the evidence points to several things; first, meningitis with complicating meningeal fibrosis and hydrocephalus. This process would take weeks to evolve, and I suspect that meningitis is what brought him ashore. Second, the chronic inflammation and accumulation of mucus in the lungs is most likely related to chronic aspiration of water over a period of days or weeks. Third, the intestine is on the verge of becoming necrotic, and would certainly have been seeding the blood with bacteria. The microabscesses are at a stage of evolution that suggests that infection has been going on for at least a period of days. What is going on, and can this all be put into a coherent picture? I think it can. Whatever his behavior might have suggested, Corky had a serious, progressive neurological problem. His functional impairment was marginal; he could get around well enough, and feed, but his reflexes were slowed just enough that he snuffled in water occasionally. The water is not sterile, and irritation and inflammation resulted. The muscle contraction of the stomach, intestine and bladder are very common, and I think are a disordered diving response. When dolphins dive, they shunt blood away form the internal organs to the brain, with just enough blood going to them to keep them alive. Although I have no experimental evidence for it, I suspect that the abnormal response in the gut is linked to abnormal brain function. The normal reflexes become disordered, and may be extreme in some segments of the intestine, causing tissue to get too little blood to maintain them. This would allow bacteria to penetrate tissues and to get into the bloodstream.

One remaining mystery is how he managed to appear to be so well, even to experienced observers, when he had a progressive and fatal neurological disease, and several other major problems. In its normal environment, a sick dolphin has nowhere to ‘den up’ and recuperate in a relatively protected place. It must give no sign of weakness or disability, or else it will attract predators. It must be able to stay with its fellows, who may offer some protection. It seems that as part of their adaptation to the aquatic environment, dolphins have developed the ability to hide or mask illness or weakness. They act normal until they crash. This is something we have seen several times. Animals that, by every external criterion are well, and suitable for release suddenly go sour and die. This has been true of recent animals with meningitis.

This has some serious implications for rehabilitation workers. Unlike familiar domestic animals, who when sick tend to look sick, dolphins can be sick with a fatal illness, including infections, yet look and behave ‘normal’. This can lull rehabilitators into thinking the animal is well and safe to be around. Experience is teaching us that strandings, whose clinical history is not known, should always be treated as an infection hazard no matter how well they may seem, and that contact with them and with the water they swim in should be minimized. Contact should always be seen as a risk to be accepted only when necessary. Weeks of observation are not enough to assure that an apparently healthy animal is in fact healthy. Even an animal feeding well, growing, and putting on weight as Corky was is not necessarily free of disease.