CLINICAL AND PATHOLOGICAL FINDINGS IN DOLPHINS IN 1977.

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In contrast to our analysis of the deaths of captive cetaceans in European dolphinaria for 1976 (Greenwood & Taylor 1977), 1977 saw a reduction in the loss of established animals, but a considerable loss amongst newly captured specimens. There was a net influx into the captive population of about 30 specimens, including killer whales (*Orcinus orca*), Guiana dolphins (*Sotalia guianensis*) and Bottlenosed dolphins (*Tursiops truncatus*), after considerable losses amongst the newly captured Bottlenosed dolphins, both from the Adriatic and from Mexico. There were no losses amongst killer whales, although one etablished specimen was exported to the United States. The immediate loss of three *Sotalia* with post-transport lung disease has been reported (Bössenecker 1978).

For this analysis, we have recorded the deaths of 19 established *Tursiops truncatus*, one captive-bred infant *T. truncatus* and one established *Sotalia guianensis* (more than one month in Europe), a total of 21 animals. We have no clinical or pathological information about the loss of one further *T. truncatus.* This estimated total of 21 established animals (excluding the infant) represents about 12% of the European captive population, rather lower than 1976.

Nine dolphins were autopsied by us and nine by other veterinarians or pathologists. From some of these, only limited data are available but these have been included. Three animals were not submitted for autopsy.

Clinical diagnosis and survival

Findings in dolphins surviving episodes of disease are not included, although some of the most important are discussed later. Of the 18 animals autopsied, only 12 had received full clinical examinations, although some had been treated on previous occasions and their history was known. Three animals died suddenly. Diagnosis was confirmed as substantially correct (i.e. including all important disease processes) in 8 cases (44%) and substantially incorrect or not completed before death in 4 (22%) (Table 1).

TABLE 1
DIAGNOSIS IN LIFE IN 18 DOLPHINS

Correct	8	44%
Incorrect	4	22%
Not examined	6	33%

Correct diagnosis, for which therapy was ineffective or impossible, included parasitic bronchitis, pyelonephritis, chronic pulmonary abscessation, pulmonary fibrosis, liver disease, gastric ulceration and multiple organ collapse. The latter occurred in a dolphin which contracted pneumonia, from which it recovered, but then developed gastric ulceration and candidiasis, thyroid, liver and renal failure, all of which were monitored before death. Diagnosis was incorrect or not made in cases of *Pseudomonas* pneumonia (believed to be candidiasis), fatty degeneration of the liver, possible poisoning and pregnancy toxaemia with arteriolar necrosis.

The survival times of the eighteen animals autopsied, from the initial recognition of illness, are recorded in table 2. The two animals with a history of disease for more than six months were a case of chronic pulmonary fibrosis maintained on corticosteroids, which animal collapsed after being attacked by its companions, and one case of chronic pleuro-pneumonia.

TABLE 2
SURVIVAL TIMES IN 18 DOLPHINS

< 1 week	4	22%
1 - 4 weeks	8	44%
1 - 6 months	4	22%
>6 months	2	11%

Pathological findings

The aetiology and location of lesions found in 18 dolphins are represented in tables 3 and 4. As usual, bacterial and fungal infections predominate and these are identified, where possible, in table 5. No virus-like conditions were seen. Parasitism occurred only in Adriatic Bottlenosed dolphins and included *Anisakis simplex* and *Pholeter gastrophilus* in the stomach and *Halocercus lagenorhynchi* in the bronchial tree. The case of chronic pulmonary fibrosis, in a Bahamas *T. truncatus*, was thought to be the result of a previous lungworm infestation. The

TABLE 3
AETIOLOGY OF LESIONS IN 18 DOLPHINS

Viral	0	
Bacterial	8	44 %
Parasitic	5	27.8%
Fungal	4	22 %
Degenerative	6	33 %
Toxic	3	16.7%
Traumatic	1	0.5%
Secondary	3	16.7%

TABLE 4
AFFECTED ORGAN SYSTEMS IN 18 DOLPHINS

Respiratory	7	38.9%
Gastro intestinal	7	38.9%
Liver	5	27.8%
Cardiovascular	5	27.8%
Genito urinary	3	16.7%
Skin (severe lesions)	3	16.7%
Lymphoreticular	2	11 %

TABLE 5
SIGNIFICANT MICRO-ORGANISMS ISOLATED FROM 18 DOLPHINS

Staphylococcus pyogenes	2
Haemolytic streptococci	2
Klebsiella rhinoscleromatis	1
Haemolytic E.coli	1
Proteus morgani	2
Pseudomonas	1
Candida sp.	4
Morganella	1

fungal lesions were skin and oesophageal infections with *Candida sp.*, in one case identified as *Candida parapsilosis.* No tumours were seen. The toxic lesions were due to severe external damage with sodium hypochlorite, pregnancy toxaemia with intra-uterine foetal death and a possible heavy metal poisoning. The degenerative lesions mainly included fatty infiltration and cirrhosis of the liver, myocardial fibrosis and medial arteriolar necrosis. Gastric ulceration occurred secondarily in most chronically sick animals.

Amongst the organ systems affected, lungs, liver and gastro-intestinal tract predominate as usual. Three animals showed arteriolar necrosis and one had myocardial fibrosis. The usual range of pathogenic organisms was isolated, with a high number of severe *Candida* skin infections. *Klebsiella rhinoscleromatis* represents a rare isolate in pure culture from a case of chronic exudative pleurisy. Quite a number of obvious bacterial lesions were not cultured.

The occurrence of specific lesions is recorded in Table 6. The presence of 37 serious lesions in 18 dolphins, even though some were clearly directly connected (e.g. lung abscess and lymphadenitis) demonstrates the complexity of disease processes in these mammals, and the necessity for full investigation of sick animals. At present, tehniques of the investigation of

TABLE 6

SPECIFIC LESIONS IN 18 DOLPHINS

Enteritis	1
Gastro-intestinal parasitism	2
Ulceration	3
Gastro-intestinal mycosis	1
Hepatic degeneration	5
Chronic pneumonia	3
Pleurisy	1
Pulmonary fibrosis	1
Parasitic bronchitis	2
Lymphadenitis	2
Pericarditis	1
Myocardial fibrosis	1
Arteriolar necrosis	3
Pyelonephritis	2
Foetal death	1
Dermatitis	3
Abscess	3
Septicaemia	2

circulatory diseases, particularly of the blood vessels, are poor and the possibilities for curative treatment are very limited. The same constraints apply to degenerative liver desease which may not cause any alteration in blood chemistry, although the technique of liver biopsy (Sweeney & Ridgway 1975) deserves wider use. On the other hand, chronic pneumonia and lung abscess are quite readily diagnosed or strongly suspected but unless such infections are identified at routine blood and clinical examinations, staff may notice no significant abnormality until large areas of tissue are destroyed and abscesses well established. At this stage, antibiotic therapy can only be palliative. Although treatment will often suppress symptoms, too often chronic foci of infection merely become walled-off from the circulation, to break out again when therapy is discontinued. The effects of chronic toxaemia or massive destruction of tissue may finally bring about death through degenerative liver and kidney disease or congestive heart failure. It is often surprising how little viable lung tissue a dolphin appears to need to live and work apparently normally in captivity.

Clinical problems

The major clinical problem occurring in 1977 has been lungworm infestation in newly captured dolphins from the Adriatic sea. The deaths of only two of these animals have been recorded in this analysis because very few became established in captivity. Parasitic bronchitis has been extensively recorded in Adriatic and Black sea dolphins (Delyamure 1968, Hörning et.al. 1970) and, in some species, infection has been thought to be a limiting factor on populations

(Kleinenberg 1956). The situation described in the Black sea common dolphin (*Delphinus delphis*) infected with *Skrjabinalius cryptocephalus* by Russian authors, would appear to obtain in the northern Adriatic sea in *Tursiops truncatus* infected with *Stenurus ovatus* and *Halocercus lagenorhynchi.* A very large proportion of the animals seem to carry very heavy infestations with lungworms and some of the older specimens have severe, permanent lung damage, including emphysema and chronic bronchitis. It is difficult to understand how some of the animals can survive life at sea. Needless to say, when the stresses of captivity and transport are superimposed on such chronic lung disease, the chances of survival are poor, unless treatment can be carried out.

The success of treatment for active lungworm infestation depends on the existing degree of lung damage, the degree of infestation and, apparently, on the species of lungworm present. The *Stenuridae* are short, straight nematodes some 15 - 25 mm. long, which lie essentially free in the bronchial three and can be blown out of the respiratory tract if they die. The *Halocercidae*, on the other hand, are often over 100 mm. long and show various adaptations for firm attachment to the host. The tails of these nematodes may be threaded through the lung tissue itself and coiled into large knots, so that they are difficult to remove even by dissection. This attachment means that the removal of dead worms and debris from the lung is difficult, making a servere allergic reaction to nematode proteins more likely. In practice, this proved to be the case and the more common *Halocercus* were very difficult to treat. Unfortunately, there are no criteria for distinguishing the larvae of these species, which can be readily observed in blowhole smears from infected animals. The most useful treatment regime seemed to be to protect the animal with broad spectrum antibiotics and antihistamines through a long period of repeated dosage with levamisole hydrochloride (5 mg/kg once weekly by mouth) which is effective against the adult lungworms. This appeared to prolong the time to eliminate the appearance of live larvae at the blowhole, but minimised the chances of anaphyllaxis or secondary bacterial pneumonia.

Discussion

The indications continue to be that bacterial disease and the long term sequelae of chronic infections are the major killers of established captive dolphins. *Candida* infections regularly give problems, especially in debilitated animals. Their occurrence is undoubtedly due to a gradual build-up of organisms in closed water systems, until the animals either become secondarily infected during the course of some other illness, or become completely overwhelmed by the challenge and develop primary candidiasis. Whichever situation obtains, it is essential that safe and effective methods to reduce or eliminate yeasts in water systems be developed.

The loss of an animal during her second pregnancy (the first produced an infant which survived 14 months), as a result of pregnancy toxaemia, may be a warning that pregnancy is a significant source of risk to captive dolphins. The development of hormonal methods of early pregnancy diagnosis is vital so that each pregnant animal can be carefully observed right through the period of risk and foetal viability monitored, if under suspicion, by ultrasound scanning. The early diagnosis of intra-uterine foetal death and its prevention, by minimising handling of pregnant animals, is of great significance to any breeding programme and to the survival of the mothers.

High death rates among newly imported dolphins are now extremely rare. They are usually the result of inadequate acclimitisation and incompetent shipment of animals, or of accidents. It is unusual that, as in the case of Adriatic Bottlenosed dolphins, such losses should be, in large part, due to the state of health of the wild stock. A small scale live capture operation may be contemplated in an area if animals are present in suitable waters or if a commercial fishery already exists. It is essential that the health of the animals be examined before the operation begins, either by autopsy of commercially killed specimens or by careful clinical examination of a few animals in holding facilities, so that dolphins are not lost because they are unusually unfitted for the stresses of transition to captivity.

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