CLINICAL AND PATHOLOGICAL FINDINGS IN DOLPHINS IN 1976

A. G. Greenwood MA, VetMB, MRCVS., and D. C. Taylor BVMS, FRCVS.
Hainsworth House, Damems Lane, Keighley, U. K.

The purpose of this communication is to describe and analyse the clinical and pathological findings in captive small cetaceans which died in Europe and South Africa during 1976. At present we are aware of 24 deaths occurring last year, comprising 3 South Atlantic Bottlenosed dolphins *(Tursiops truncatus),* 2 Indian Ocean Bottlenosed dolphins *(Tursiops aduncus),* 2 Cape Dusky dolphins *(Lagenorhynchus obscurus)* and 17 North Atlantic Bottlenosed dolphins *(Tursiops truncatus).* Of the latter *T. truncatus* one was captured in the Adriatic, 4 off the Bahamas and the remaining 12 off Florida or in the Gulf of Mexico. Two of them were infants born and conceived in captivity. The Adriatic dolphin and the two Duskys were newly captured.

No clinical or pathological data is available for 3 *T. aduncus* reported lost in one pool in Africa. An adult male and a 3-week old infant *T. truncatus* were dissected post-mortem by Prof. R. J. Harrison, but no clinical information is available. These animals are therefore not included in the analysis. Of the remaining 19 animals two were not under our care at any time, although we have been asked to comment on the clinical and autopsy data provided ; one animal was not submitted for autopsy ; eight animals were autopsied by well qualified university pathologists at our request, and eight animals were autopsied by us.

The death of 22 adult animals (excluding the two infants) in the twelve month period of 1976 represents 15% of the estimated total number of small cetaceans in captivity in Europe and Africa during that year, although there may have been other deaths not reported to us. Those deaths which occurred in Britain comprise 6.25% of the animals maintained there. The ownership and location of animals discussed in this report will not be individually identified .

Clinical diagnosis and survival.

This analysis does not include the many sick animals successfully treated, but only those which finally came to autopsy. As indicated by the success of diagnosis in life of animals finally coming to autopsy (Table 1), despite great advances in diagnostic

TABLE 1

DIAGNOSIS IN LIFE IN 16 DOLPHINS

Correct	4	25%
Correct but incomplete	5	31.25%
Incorrect	4	25%
Not examined	3	18.75%

procedures (Sweeney & Ridgway 1975, Greenwood 1976) much treatment is still rather empirical, and an analysis of surviving clinical cases would not be very satisfactory. Of the 16 dolphins on which we have full data, 25% had a completely correct clinical diagnosis made in life, but treatment was unsuccessful or only palliative (as in a case of cor pulmonale). These include acute hepatitis with extensive gastric ulceration, pulmonary abscess with secondary renal lesions, tension pneumothorax following severe lungworm infestation, and progressive pulmonary fibrosis with heart failure following a treated lungworm infection. A further 31.25% (5 animals) had a correct but incomplete diagnosis, in that some important underlying disease or lesion was not recognised. Thus myocardial dystrophy was not recognised in two animals (one with enteritis/hepatitis and one with renal necrosis), chronic infective lung disease was not recognised in two animals (one with chronic allergic bronchitis and one with enteritis/gastric ulcer), and thyroid atrophy and pancreatic duct carcinoma were not recognised in one animal presenting with pyelonephritis and enteritis. Thus in over 50% of cases diagnosis was correct or sufficient for some appropriate therapy to be applied.

In 4 cases (25%) the diagnosis was substantially incorrect or was not made before death. These comprised one case of lymphosarcoma, one case with combined thyroid and adrenal atrophy, and two cases of pulmonary abscessation with no detectable clinical or laboratory abnormalities.
Three animals were not clinically examined whilst ill, two because they died suddenly and one which the owners refused to restrain for examination, although it had very obvious severe skin lesions and scoliosis.

TABLE 2

SURVIVAL TIMES IN 17 DOLPHINS

< 1 week	3	17.6%
1-4 weeks	8	47%
1-6 months	1	6%
> 6 months	5	29.4%

The survival time of one dolphin from the first recognition of illness is not known, as it was obviously sold whilst ill, but the survival times of the other 17 which were autopsied are recorded in Table 2. A considerable proportion (29.4%) of the animals lost in 1976 had been recognised as chronically ill for more than 6 months. One had chronic skin lesions and scoliosis and could not be examined or treated, one had a persistent pulmonary abscess, and three were Bahamas *T. truncatus* with pulmonary fibrosis (recognised), lymphosarcoma and thyroid/adrenal atrophy (unrecognised). All the three Bahamas dolphins were maintained empirically on low dose corticosteroids for several months and all eventually died quite suddenly with acute complications. These represent three of the very few indications for long-term corticosteroid therapy in dolphins, and then only after exhaustive laboratory testing had eliminated any infectious process. All three animals were mature females and it is possible that steroid therapy had considerably altered their reproductive cycles, as judged by examination of their ovaries at death (Prof. R. J. Harrison pers.comm.). The survival of 8 animals for periods approaching one month was responsible for a high incidence of secondary lesions attributed to their debilitated and toxic states (Table 3). Undoubtedly some survival times represent near-successes with treatment.

TABLE 3

AETIOLOGY OF LESIONS IN 18 DOLPHINS

Viral	2(?)	11.1%
Bacterial	13	72%
Parasitic	5	27.8%
Fungal	3	16.7%
Neoplastic	2	11.1%
Degenerative	7	38.9%
Toxic	2(?)	11.1%
Secondary	7	38.9%

Pathological findings.

The aetiology and location of the lesions found in 18 dolphins are presented in Tables 3 and 4. As usual bacterial infections predominate (in 72% of animals), and these are further identified in Table 5. The two possible viral lesions were generalised skin diseases, one heavily super-infected with *Candida,* and the other showing as multiple focal accumulations of lymphocytes in the dermis. In neither case could virus or virus material be certainly identified.

TABLE 4

AFFECTED ORGAN SYSTEMS IN 18 DOLPHINS

Respiratory	11	61%
Gastro intestinal	9	50%
Liver	9	50%
Cardiovascular	8	44.5%
Urinary	5	27.8%
Endocrine	3	16.7%
Skin (severe lesions)	3	16.7%
Lymphoreticular	2	11.1%
Musculo/skeletal	1	5.5%

TABLE 5

SIGNIFICANT MICRO-ORGANISMS ISOLATED FROM 18 DOLPHINS

Pasteurella multocida	2
Staphylococcus pyogenes	5
Haemolytic streptococci	3
Escherischia coli	4
Proteus sp.	2
Aeromonas sp.	1
Candida sp.	3
Geotrichum sp.	1

Only one severe case of parasitism was seen - an Adriatic Bottlenosed dolphin with parasitic pneumonia due to *Stenurus ovatus* lungworms. Incidental parasitism was mainly degenerated trematodes in the gastric mucosa. In two cases, however, allergic-type lung disease was thought to be secondary to parasitism of the respiratory tract (*Halocercus lagenorhynchi* lungworm, and *Nasitrema* trematodes in the nasal sacs) which had been successfully eliminated by anthelmintics. Similar lung disease has been associated with *Nasitrema* infection previously (Kumar et.al. 1975), although this infestation has been rarely seen in Europe. The fungal lesions were skin and blowhole infections with *Candida* sp., of which only one was serious. In addition *Geotrichum* sp. was isolated from a case of enteritis, but its significance is unknown.

The two neoplastic lesions, a lymphosarcoma of the spleen, and a pancreatic duct carcinoma, are the only malignant tumours we have seen in dolphins, and are both previously unrecorded. The lesions possibly due to toxins occurred in two dolphins together, presenting as severe and acute hepatic necrosis. No certain toxin could be identified, but the very poor quality fish was highly suspect.

TABLE 6

SPECIFIC LESIONS IN 18 DOLPHINS

Enteritis	4
Oesophageal and gastric ulcers	4
Oesophageal and gastric candidiasis	3
Pancreatic disease	3
Hepatitis	4
Hepatic degeneration/necrosis	4
Tonsilitis	1
Acute pneumonia	3
Chronic pneumonia	5
Pulmonary fibrosis	1
Parasitic pneumonia	1
Chronic Blowhole infection	2
Pneumothorax	1
Pericarditis	2
Myocarditis	1
Myocardial degeneration	4
Heart failure	1
Uraemia	3
Renal necrosis	3
Membranous glomerulonephritis	1
Thyroid disease	3
Adrenal atrophy	1
? Viral skin lesions	2
Infected skin wounds	1
Septicaemia	2
Abscess	1
Incidental parasitism	4
Carcinoma	2
Scoliosis	1

Of particular interest are the high incidence of degenerative and secondary lesions, some of which probably killed animals which would have been expected to survive their acute infectious conditions. An outstanding example is of a young, apparently healthy animal which contracted a bacterial entiritis along with two other animals in the same pool. The others responded well to treatment, but the individual which died had severe myocardial and hepatic degeneration. This might well have been associated with an inadequate intake of vitamins B1 and E, which were much lower than those we generally recommend. Hepatic and myocardial degeneration occurred with high frequency in this series (Table 6). The secondary lesions seen were mainly ulcerative degeneration and fungal infection of the upper gastro-intestinal tract, which is common in dolphins which are severely ill for prolonged periods (Greenwood et al. 1976), and renal lesions such as membranous glomerulonephritis and renal tubular necrosis, which may result from the chronic intoxication associated with bacterial abscessation.
The organ systems affected (Table 4) followed the usual distribution (Sweeney & Ridgway 1975a) with lungs, gastro-intestinal tract and liver disease occurring in 50% of more of the animals. Heart lesions were surprisingly common, and in all cases undiagnosed. In most cases more than one system was diseased, and in one case of *Pasteurella septicaemia* almost every organ was affected.

Staphylococci, streptococci, and coliform bacteria were the most common species isolated, representing 65% of the total significant organisms cultured. Most isolates were sensitive to the more common antibiotics in vitro, although one strain of *E.coli* isolated from a case of pyelonephritis was sensitive to cephalosporins, polymixin B and nitrofurantoins only. No *Pseudomonas* were isolated from dead animals, in marked contrast to previous years. Those which were isolated from clinical cases in 1976 were all resistant to most antibiotics, with the exception of gentamicin and tobramycin. The two *Candida* isolates tested were resistant to miconazole and 5-fluorocytosine, but sensitive to amphotericin. One was resistant to clotrimazole. *Pasteurella multocida* was isolated from two cases of septicaemia (Table 5).

The occurrence of specific lesions is tabulated in Table 6.

TABLE 7

FACTORS LEADING TO HIGH MORTALITY

Inadequate monitoring
Slow initiation of diagnosis and treatment
Importation of new animals
Chronic disease

Discussion

The mortality rate among captive cetaceans in Europe is still regrettably high, despite undoubted improvement in husbrandy and medical care.
The high rate of correct diagnosis, among those animals fully examined, when coupled

with the unusual diseases seen in some undiagnosed animals is reassuring, although there are still many problems in therapy. Not the least of these are the practical problems of handling and treating dolphins in some establishments. The diagnosis and successful treatment of the chronic bacterial lung infections are frequently unsatisfactory. Ideally these infections should be recognised in their early stages, before they become established and the toxic effects suppress the normal inflammatory responses of the animal. Early diagnosis can only be achieved by early aggressive investigation, after recognition of important signs by trainers. A surprising number of inapparent infections are identified by routine blood sampling, and if the acute phases of disease are short-lived frequent sampling will be necessary to intercept transient changes. There are always a proportion of chronically ill animals in any sizeable population, and eventually we may hope that therapeutic advances will provide permanent cures for most of them. Meanwhile, such animals must be very carefully monitored, they are always likely to succumb to some other minor stress or disease. Newly captured specimens are always at risk, especially at present when capture operations are rather amateur, and when the scarcity of animals means that any captive animal is taken for display, instead of selecting the best and rejecting the less perfect. It is especially important that animals are closely examined before transportation, as the stress of this will often cause the breakdown of hidden disease.

Some of the factors which maintain this high mortality rate are outlined in Table 7. It is our opinion that poor monitoring of animals, and slow and inadequate reaction to abnormal signs from the animals, are still serving to negate some of the diagnostic and therapeutic advances that are being made.

Acknowledgements

We are extremely grateful to those clinical and pathological laboratories which have provided us with facilities around the world, and to pathologists who have carried out some of the autopsies and histopathology. We particularly wish to thank D. Bostock MRCVS, Prof. Dr. H. Stunzi, Dr. A. F. Heydorn, and the laboratory staff of the Cambridge Veterinary School and Airedale General Hospital.

References

GREENWOOD A. G. (1976). Recent advances in clinical and diagnostic techniques in marine mammals. European Association for Aquatic Mammals, 4th Annual Symposium, Mallorca, Spain.

GREENWOOD A. G., D. C. TAYLOR and D. WILD (1976). Diseases of the upper gastro-intestinal tract of dolphins. American Association of Zoo Veterinarians, Annual Conference, St. Louis, Missouri. October 1976.

KUMAR V., J. VERCRUYSSE, P. KAGERUKA and J. MORTELMANS (1975). *Nasitrema attenuata* (Trematoda) infection of *Tursiops truncatus* and its potentialities as an aetiological agent of chronic pulmonary lesions. J. Helminthol. 49: 289-292.

SWEENEY J. C. and S. H. RIDGWAY (1975). Procedures for the clinical management of small cetaceans. J. Amer. Vet. Med. Ass. 167:540-545.

SWEENEY J. C. and S. H. RIDGWAY (1975a). Common diseases of small cetaceans. J. Amer. Vet. Med. Ass. 167:533-539.

— — —