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Neonatal mortality in two Indian Ocean bottlenose dolphins bred in captivity

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Summary

Two Indian Ocean bottlenose dolphins (*Tursiops aduncus*) bred in captivity through the same father were born within a week of each other. The first dolphin died within five hours of birth as a result of high speed collision with the tank wall. The second dolphin died 93 hours after birth whilst suffering from jaundice. The history, clinical syptoms, clinical pathology, autopsy examinations and possible causes and prevention are reported and discussed.

Introduction

The capture of wild dolphins for the purpose of research and display is drawing increasing criticism from conservationists and sentimentalists alike. Due to the increased pressure from these groups, oceanaria and dolphinaria are turning their attention to establishing breeding colonies within their centres in order to supplement the dolphin populations in captivity. Although knowledge of reproductive physiology and neonatal care has advanced rapidly over the past few years, neonatal fatalities are, however, still high (Sweeney 1978). This paper reports on neonatal fatalities in two Indian Ocean bottlenose dolphins (*Tursiops aduncus*). Both animals shared a common father, the dams cohabiting the same pool as the male.

History and Case Report

All three adult parents (all *T. aduncus*) were captured within a week of each other in the Indian Ocean, not further than 20 km from Durban, Natal. One of the females and the male appeared to be of similar age, while the other female seemed to be older, based on size and the wearing of the teeth.

Initially the younger pair of dolphins were kept together but, at a later date, the older female was allowed to join them, as incompatibility developed between her and her selected mate, a large *Tursiops truncatus*. The three animals accepted each other and during the four years that they were together, the male was frequently observed to mate with either of the females. After an exceptionally prolonged and heavy period of mating with both females during November 1983, the male developed a septicaemia of unknown aetiology and died in December 1983.

Over the next few months both females gained weight and showed enlargement of the mammary glands. A spontaneous secretion of colostrum, following tactile stimulation, was observed in the older female in July 1984. Based on these observations, as well as the finding of markedly elevated serum progesterone levels above 20 000 pg/ml and 40 000 pg/ml respectively, both animals were diagnosed as being pregnant.

The first calf, a female, was born in posterior presentation to the older mother on 22 October 1984. Calving took 3 hrs 3 min. The afterbirth was expelled within two minutes of partus having been completed. Effective suckling was first observed 3 hr 37 min after partus. The frequency of suckling thereafter was exceedingly variable (2–22 min), but the average time spent suckling remained constant at 3–5 seconds (Peddemors, in prep.).

Five hours after partus a violent fight between the two females ensued and the neonate, in an attempt to escape, collided at high speed with the wall of the tank. The neonate rapidly lost consciousness and, although retrieved and supported on the surface by its mother, soon died. The fight started when the other female tried to claim the neonate, probably due to a strong maternal instinct present as a result of her own imminent parturition. An autopsy performed on the calf revealed extensive meningeal haemorrhage, which was particularly severe over the cerebellum, and extended cranially over the cerebrum and caudally along the dura mater of the spinal canal to the region of C_7–T_1. Compression fractures of cervical vertebrae between C_5 & C_6 and C_6 & C_7 were also found. The remaining organs appeared to be normal.

The birth of the second dolphin, a male, took place two days later. Parturition, by posterior presentation,

was shorter and took only 1 hr 51 min. The afterbirth was expelled within 30 minutes. In order to prevent a repetition of the accident described above, fine-meshed nets were installed around the perimeter of the tank, extending from top to bottom, suspended away from the wall, so as to provide a cushioning effect in the event of a collision. The two females had also been separated before the second parturition so as to avoid any fighting.

The respiratory interval of this calf initially averaged 13.6 sec, but lengthened progressively to an average of 29.1 sec over the next few days (Peddemors, in prep.). Effective suckling occurred four hours after parturition but, unlike the first calf, the frequency of suckling was very high and the period of time attempting to suckle was variable but longer than that observed in the first calf.

On the second, third and fourth days post partum, the newborn dolphin appeared to be doing well. Defaecation was observed on the second day, the exreta being a normal brown colour. Suckling remained erratic, while the respiratory interval remained between 24.7 to 34.2 sec (Peddemors, in prep.).

Towards the end of the fourth day, the mother displayed a sequence of unusual behaviour patterns which intensified the following day. She started by pushing the neonate around with her beak which progressed to throwing the baby out of the water, using the tip of her beak. She would repeatedly take the baby to the bottom of the tank, pinning it down, or would aggressively beat it with her tail. At times, however, she would become more compassionate and gently support the calf on the surface.

By 04h00 on the morning of the fifth day following partus, the neonate's eyes remained tightly closed for most of the time and he uttered clearly audible squeaks when assaulted by his mother. The respiratory interval had shortened to 18.8 sec. The calf last suckled at 04h59 and thereafter he aimlessly searched for a teat in the ano-genital region. By 09h00 his swimming speed had slowed considerably and by 11h39 he began to perform 'rocking-horse' motions which were assumed to be a form of convulsion. By 12h00 the calf would remain motionless at times for periods of 6–8 sec, either on the water surface, or on the bottom of the tank. The mother persistently displayed abnormal patterns of behaviour, frequently pushing the baby to the bottom of the tank, rather than supporting him on the surface. The respiratory interval at this stage had increased dramatically to 5.9 sec. Soon after this, the condition of the calf rapidly deteriorated and he died at 12h55, i.e. 93 hours post partum.

Clinical Pathology

Blood was taken from the second calf via cardiac puncture approximately 20 minutes after clinical death and was collected into plain test tubes as well as tubes containing the anticoagulant EDTA (Venoject, Terumo).

Table 1. Blood results obtained from the second calf via Cardiac puncture

			Adult Normals (undetermined for neonates)
a) Haematology			
B–WBC($\times 10^9$/l)	4.5		7.3
B–RBC($\times 10^{12}$/l)	5.1		4.22
B–Hb(q/l)	177		171
B–Ht(l/l)	0.56		0.51
E–MCV(fl)	115.0		120
Differential count	B– Neutrophils	0.00	0.44
	B–Lymphocytes	0.38	0.23
	B–Monocytes	0.60	0.3
	B–Eosinophils	0.02	0.29
Monocytes showed gross toxic changes.			
Red cell morphology –	Anisocytosis +		
	Polychromasia +		
	Howell-Jolly bodies +		
	Reticulocytes 2.4%		
Platelets–fibrin clots.			
b) Biochemistry			*Adult Normals*
Serum alkaline phosphatase	1950 iu/ml		600–800 iu/ml*
Total bilirubin	85 µmol/l		5–9 µmol/l**
Osmolality	382 mmol/l		310–350 mmol/l***

*Method: 'Optimized standard method' conforming to the recommendations of the Deutsche Gesellschaft für Klinische Chemie done at 30°C.
**Method: Jendrassik, L. *et al.* (1938). *Biochem.*Z.297: 81.
***Analysed by depression of freezing point by Roebling automatic osmometer.

Unfortunately, due to either postmortal haemolysis, or perhaps severe antemortal haemolysis, serum enzyme and electrolyte levels could not be accurately determined and are, therefore, not included in this report. However, serum alkaline phosphatase, total bilirubin and osmolality were measured with reasonable accuracy.

Comments:

Infant dolphins are recognized as being rather delicate and succumb rapidly to 'capture shock' if handled before six months of age (Sweeney, 1978). For this reason comparative normal haematological values are not available but, based on observations in human and other animals, one could assume that the values for red blood cell count and haemoglobin would be higher in the neonate than in the adult (Dacie & Lewis, 1984). The values obtained for these parameters in the neonate would be regarded as

being within normal limits in an adult animal, whereas in the neonate both may be regarded as being on the low side indicating an anaemia. This was further supported on necropsy of the animal where a distinct paleness was observed in certain organs and in the musculature which, in the dolphin, is normally a deep red colour (as was observed on autopsy in the first neonate). The presence of reticulocytes would support haemolysis and regenerative anaemia as this is not a normal finding in an adult dolphin, but again this may be regarded as normal in a neonate. Of considerable interest was the lack of mature neutrophils on the smear. This could be due to exhaustion and/or phagocytosis or perhaps misidentification of immature forms. Serum alkaline phosphatase, although occurring in other tissues, has been reported to be present in large amounts in the liver and large increases in circulating SAP, could therefore be indicative of liver disease (Sweeney 1978; Feldman 1980). Total bilirubin was markedly elevated but, unfortunately, levels of conjugated and unconjugated bilirubin were not determined. The increased osmolality indicates dehydration. A Coombs test (Ortho Diagnostic Systems Inc.) incorporating ortho antihuman serum (rabbit) for the Direct Antiglobulin Test was used to test for the presence of red blood cell antibody. This test was negative.

The serum was also screened (using the Abbott radioimmunoassay for the detection of IqM antibody to Hepatitis A (Havab–M) and the Abbott radioimmunoassay for the detection of antibody to Hepatitis B (Austria II). Both tests were negative.

Morphological Pathology

Macropatholgy

An autopsy on the second neonate was conducted within two hours of its death. The general condition of the animal was good with an adequate muscle development and fat covering. Externally no lesions were evident apart from a discolouration of the skin on the ventral surface of the body, giving the normally creamy-white skin a muddy-yellow tinge. On examination of both the oral mucosa and the occular mucous membranes, a pronounced icterus was visible. This jaundiced condition was generalized but was particularly evident in the blubber, aorta and walls of the other large arteries and in the fat. The muscles were very pale red. The liver showed moderate hepatomegaly and had a light tan colour. A slightly gritty texture was noticed on transection. The intra- and extra-hepatic bile ducts were examined and found to be patent. Subcapsular petechiae and ecchymoses were found on examination of the spleen. A moderate splenomegaly was also present.

Severe bilateral pulmonary oedema with focal areas of congestion and ecchymoses were observed together with copious amounts of froth in the trachea and bronchi. The stomach was empty but the intestines contained a moderate amount of digested milk which was slighly mucoid in consistency and markedly bile stained. The kidneys were pale brown in colour and moderate congestion was evident at the cortico-medullary junction. The urine was clear and had a dark yellow-brown colour. The urine bilirubin level as well as the haemoglobin level were both abnormally elevated; recorded test strip readings were '4-plus' (Combur[9] Test strips, Boehringer Mannheim Pharma).

Micropathology

Histological examination of the liver showed most of the hepatocytes swollen and vacuolated, indicating both cloudy swelling and hydropic degeneration. The nuclei were, however, normal. Random single cells and small groups of hepatocytes had a shrunken appearance with increased cytoplasmic eosinophilia (i.e. the cyctoplasm stained pinker than normal), and moderately pycnotic nuclei. This change probably represents single-cell necrosis. As the general appearance of the liver was mostly that of degenerate cells, a pathological anatomical diagnosis of a moderate to severe hepatosis was made. No sign of inflammation was present, thereby excluding hepatitis.

The kidneys revealed a cholaemic nephrosis. All the proximal tubular epithelial cells were markedly swollen, with vesicular nuclei and vacuolated cytoplasm which contained large amounts of bile pigment. The cortico-medullary junction showed moderate congestion.

The spleen showed hyperlasia of the red pulp characterized by an increase in the macrophages and reticular cells. There appeared to be an abnormally active erythrophagocytosis; even more than one would normally expect in a neonate. Large amounts of bile pigments and some haemosiderin was observed in the macrophages.

The lungs revealed moderate congestion with only a small percentage of the alveoli containing pink laminated fibrillar proteinaceous material. This completely filled the affected alveoli.

Numerous sections of the skeletal muscle were examined histologically, but no abnormalities were found.

Bacteriology

Bacterial isolates were taken from cardiac blood, lung, liver and stomach contents. The liver yielded a *Moraxella lacunata* and from the lung *Pseudomonas aeruginosa* was isolated. Both organisms isolated from autopsy material were regarded as commensuals or contaminates, as no heavy growth was reported and also because both were isolated only from single organs.

Discussion

The cause of death of the first neonate is obvious. Trauma, through high speed collision with the tank wall, caused severe meningeal and cerebral haemorrhage with spinal cord damage in the region of C_5 to C_7. This resulted in a rapid loss of consciousness and death. In order to prevent a similar incident, nets were installed and secured around the tank perimeter so as to provide a cushioning effect following the birth of the second neonate.

It was not possible to make an aetiological diagnosis in the case of the second neonate. Jaundice was the outstanding clinical sign in this dolphin. Post hepatic (obstructive) icterus was excluded on the patency of the bile ducts and the finding of large amounts of bile in the intestine.

Although serum alkaline phosphatase levels were highly elevated, possibly indicating liver involvement, hepatocellular jaundice would appear to be unlikely as the histopathology revealed a hepatosis rather than a hepatitis. Elevated serum alkaline phosphatase levels however may be normally present in the dolphin neonate as observed in terrestial animals. Serological testing for the presence of viral hepatitis antibodies was negative. However, in acute viral hepatitis, an elevation in the antibody levels could well be delayed. No attempt to culture a virus was made. By exclusion, it would appear, therefore, that the icterus observed was probably prehepatic (haemolytic) in origin. Prehepatic jaundice in animals and man is accompanied by severe anaemia (Smith *et al.* 1972). In this case, however, the red blood cell count and haemoglobin levels indicated only a moderate anaemia. This is assuming that the values for RBC and haemoglobin are higher in the newborn dolphin than in the adult, as observed in man. The haemoglobinuria recorded on the urine dipstick could possibly be due to contamination by blood on incising the bladder wall and, therefore, cannot be used as a definite indication of haemolysis. If severe haemolysis had occurred, one would have expected to find evidence of haemoglobinuric nephrosis, i.e. haemoglobin pigment in the cytoplasm of the proximal convoluted cells and perhaps haemoglobin cases in the tubule lumens. Neither was present however. The haemolysis observed in the collected serum could have occurred antemortally, but again, may have resulted through postmortal autolysis.

The aggressive behaviour of the mother towards her newborn cannot be ignored. This behaviour was initiated at the same time that the condition of the neonate started to deteriorate. Whether this abnormal behaviour was a primary cause of the death of the animal is debatable. One would then have to assume that the jaundiced condition and high bilirubin level were not severe enough to result in death and were merely incidental findings. However, the mother did display very strong maternal instincts both towards the other neonate and, initially, in her own baby and, therefore, it would be surprising to interpret her behaviour as being malicious or hysterical. Another possible explanation of her behaviour is somewhat conjectural, and assumes that she may have deliberately hastened the death of the neonate when she realized that it had a terminal illness.

Despite the absence of a positive diagnosis, the finding of high serum levels of bilirubin is significant. In the human neonate bilirubin may cross the blood-brain barrier and cause death (so-called kernicterus of human infants with, for example, erythoblastosis foetalis (Smith *et al.* 1972)).

It remains a possibility that the raised bilirubin levels in this dolphin resulted from an isoimmune haemolytic syndrome similar to erythroblastosis foetalis of humans. The fact that the parents had been observed to mate frequently over a few years, could have resulted in one or more resorptions or early abortions. These miscarriages, in turn, may have precipitated the formation of large amounts of isoantibody and led to haemolytic icterus and death in the newborn dolphin. Bearing this possibility in mind, it would be advisable, in future, to consider cross-matching the blood of breeding pairs in order to be in a position to prevent the occurrence of such a syndrome.

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