Buoyancy compensation problems in a sand tiger shark,  
(Carcharias taurus, Rafinesque, 1810)

Problèmes de flottabilité chez un requin tigre de sable,  
(Carcharias taurus, Rafinesque, 1810)

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ABSTRACT

The sand tiger shark (Carcharias taurus, Rafinesque, 1810), generally adapts well to large aquaria and has become a popular exhibit species. It is acknowledged that C. taurus is capable of ingesting air at the surface of the water, storing it in its stomach and approximating neutral buoyancy. Although the predisposing factors are unclear, buoyancy compensation problems in this species have been observed in a number of aquaria. This article reports on buoyancy problems observed in a female C. taurus held at the Madrid Zoo-Aquarium. Steps were taken to identify the cause of the condition and try to alleviate the symptoms. Air was artificially introduced into the stomach of the shark with initially positive results. Unfortunately, the buoyancy compensation problem returned, suggesting an underlying chronic health condition. Implications for the future diagnosis and clinical treatment of this condition are discussed.

RéSUMÉ

Le requin tigre (Carcharias taurus, Rafinesque, 1810), s'adapte généralement bien dans des Aquaria de grand volume et cette espèce est devenue très populaire. Il est bien connu que le requin tigre de sable peut ingérer de l'air à la surface de l'eau, le stocker dans son estomac et maintenir une flottabilité approximativement neutre. Bien que les facteurs déclencheurs soient encore inconnus, des problèmes de flottabilité chez cette espèce ont été observés en aquarium à de nombreuses reprises. Cet article rapporte un cas pathologique rencontré chez une femelle requin tigre maintenue à l'Aquarium du Zoo de Madrid. Certaines mesures furent adoptées pour identifier les causes et essayer d'atténuer les symptômes. L'insufflation artificielle d'air dans l'estomac a eu dans un premier temps des effets positifs. Malheureusement, les problèmes de flottabilité se sont renouvelés, indiquant une pathologie chronique. Les hypothèses de diagnostic et les traitements cliniques envisagés sont donc ici sujets de discussion.
INTRODUCTION

During the first ten days of April, 1999 a female sand tiger shark (Carcharias taurus Rafinesque, 1810), maintained at the Madrid Zoo-Aquarium, was observed exhibiting unusual swimming behavior indicative of an excessive negative buoyancy (Figure 1). The specimen, having a total length of 200.0 cm and weighing 130.0 kg, had been kept in a mixed-species exhibit for four years without previous observable health problems. The exhibit, 20 m in diameter and 4 m deep, contained 1.1 nurse sharks (Ginglymostoma cirratum Bonnet, 1788), 4.1 tangerine sharks (Carcharhinus plumbeus Nardo, 1827), 1.2 C. taurus and numerous species of teleost fishes. The seawater for the exhibit was synthetic (Bromine-lite coral reef Red Sea salt, Red Sea Fish Ptlm, Israel) and water treatment was provided by rapid sand filtration and sub-gravel biological filtration. Water temperature and pH were maintained within the ranges 21.5°C - 26.0°C and 8.0 - 8.2, respectively.

It is known that C. taurus will swallow air at the water surface and store it in the stomach to approximate neutral buoyancy (Hussain, 1989). This allows the shark to stay almost motionless in the gentle currents that sweep the rocky gorges of their preferred natural habitat. Although the predisposing factors are not always clear, buoyancy compensation problems in C. taurus have been observed in other aquariums (Long, 1998; 1999; Fachau, 2000; Garratt, 2000). In this case, it was believed that the observed swimming behavior was indicative of a chronic loss of air from the stomach. Attempts were therefore made to artificially re-introduce air into the stomach of the shark and treat other associated symptoms with chemico-therapeutic. This study details progress of the observed buoyancy compensation problem and steps taken to try to alleviate it.

METHODS

Oral chemico-therapeutics were administered by ‘pate’ feeding the shark with medicated food, specifically, European hake (Merluccius merluccius Linnaeus, 1758) and squid (Loligo sp.). IM (intra-muscular) chemico-therapeutics and anesthetics were injected into the dorsal suture of the shark using a syringe and a 19 gauge, 25.4 mm long needle. Blood was drawn from the ventral caudal vessel using a syringe and 18 gauge, 38.1 mm long needle. Ultrasonography was performed using an Aloe eco-camera (Echocamera Aoka SSD-500, Aoka Co. Ltd., Japan) and a 5.0 MHz and 3.5 MHz transducer on 11 November 1999 and 7 June 2000, respectively. Endoscopic examinations were performed using a Fujinon endoscope (Endoscope Fujinon Col-MP2, Fuji Photo Optica Co. Ltd., Japan) with a sound of 143.0 cm.

Air was introduced into the stomach of the shark using a modification of techniques previously adopted by Smith (1999), Garratt (2000) and McEwan (2000). Air was injected into the shark by passing a rigid 0.8 cm diameter polyethylene tube through the mouth and 50.0 cm past the oesophagus into the stomach. This was done while the mouth of the shark was held open using a 30.0 cm length of 15.0 cm diameter high-pressure PVC. The other end of the small polyethylene tube was attached to a hose and the first stage of an air-filled SCUBA tank. Air was injected into the stomach of the shark by opening and closing the valve of the SCUBA tank in four short, sharp, bursts.

RESULTS

The food intake of the affected shark was higher than that of conspecifics throughout the four year period leading up to the observed health condition. The mean food intake for all C. taurus within the exhibit was 0.91 kg/day. The mean food intake for the affected shark was 1.15 kg/day. The initial symptoms of the buoyancy condition were observed three days following the highest food intake observed for any of the C. taurus (i.e. 4.5 kg/day). Neither the ultrasound nor the endoscopic examination revealed any apparent anomalies in or around the stomach.

Anti-biotic treatments given to the affected specimen appeared to yield little improvement. The transient remissions observed on 21 June, 1999 (i.e. 14 days) and 12 March 2000 (i.e. 4 days) did not appear to be directly related to any of the treatments administered, although the first episode occurred shortly after the administration of an anti-inflammatory pharmaceutical.

Following the injection of air into the stomach of the animal (11 November, 1999), it commenced swimming normally with its caudal fin slightly higher than the horizontal plane of its head. Its swimming behavior was dramatically improved for 13 days, before reverting back to the pre-treatment pattern. This observation was repeated after the second injection of air (7 June, 2000), whereby swimming behavior reverted back to the negatively buoyant pattern 22 days after the procedure. Results from the blood analysis performed on 11 November, 1999 indicated profound leucocytosis when compared to previously recorded values for both ‘wild’ and ‘captive’ C. taurus (Muru, 1991). White
cell counts were also well in excess of results recorded for C. taurus following a long-term transportation (Tosai, 1999). These results were repeated for the blood analysis performed on 7 June, 2000. In addition, some of the erythrocytes observed on 7 June, 2000 exhibited abnormalities.

Despite every effort to improve the condition of the shark, it was found deceased on the morning of 16 August, 2000. During necropsy the following abnormalities were observed: (1) numerous ulcerations on the ventral surface of the shark; (2) clavus slightly protruded, friable and haemorrhagic; (3) stomach distended and full of clean seawater; and (4) spiral valve highly congested, necrotic, inflamed and containing large quantities of sero-haemorrhagic fluid and mucus.

Microbiological samples taken while the animal was alive yielded negative results. Bacteria isolated from the spiral valve during the necropsy included Vibrio anguillarum, Vibrio alginolyticus and Aeromonas hydrophila. Histological examinations confirmed the macroscopic finding that there was no previous damage to the tissue of any of the major organs (i.e. gut, heart, liver, kidney, spleen, pancreas, stomach, ovaries and uterus) except for the spiral valve. The spiral valve displayed advanced inflammation, severe necrosis of the mucosa and evidence of fibrin deposits. Loss of mucosal epithelium, the presence of pathogenic bacteria and large numbers of lesions within the spiral valve suggested a chronic necrotic ulcerative enteritis.

**DISCUSSION**

External symptoms observed during the onset of the health condition seemed to suggest an excess negative buoyancy as the cause of the abnormal swimming behavior in the affected shark. The most likely cause for this situation appeared to be an inability for the shark to maintain an adequate supply of air in its stomach. This condition has been observed in C. taurus previously, and can be related to a number of different causal agents. They include: (1) loss of air during the manipulation associated with capture and transportation; (2) “obturation”, or colliding with the walls of the exhibit; (3) fighting with conspecifics due to excessive specimen density; social hierarchy or competition; (4) impaired swimming behavior through poor exhibit design; and (5) an alteration of the adrenal glands causing a failure of osmo-regulation (Yaiullo, 1999; Firenza, 2000; Hoke, 2000).

The first three possibilities are generally manifest as acute problems that the shark is able to self-regulate by swimming to the surface and swallowing air. In addition, the simple act of injecting air should be able to resolve such acute problems. The fourth and fifth options also seem unlikely. None of the other C. taurus exhibited difficulties negotiating the exhibit and the necropsy did not reveal any abnormalities in the adrenal glands. It is therefore difficult to attribute the development of the buoyancy problem to any one specific factor, however two alternative hypotheses were considered:

**Hypothesis 1:** A chronic deformity of the spine and / or spinal musculature distorted the posture of the animal and prevented it from retaining air in its stomach. Isolated infectious agents may have been secondary to the cause of the condition and exacerbated it.

**Hypothesis 2:** Damage to the spiral valve by parasitic and / or bacterial infections may have compromised elimination of water from the stomach and consequently impeded the adequate storage of air. This could be caused by both disrupted peristaltic movements and changes in the equilibrium of intracellular and extra-cellular fluids. The distorted posture of the animal could then have been secondary to the initial cause of buoyancy loss.

Unfortunately, the dorsal spine was not examined on necropsy and hence hypothesis 1 could not be confirmed or refuted. However, the unexpected, short-term remissions of 21 June, 1999 and 12 March, 2000 do not appear to support this hypotheses. It seems unlikely that the animal would spontaneously recover from a spinal deformity for a period of a few days. The remission of 21 June, 1999 occurred one week after the administration of an anti-inflammatory treatment, while the remission of 12 March, 2000 followed an unprecedented period of inappetance (i.e. 8 days). This indicates the possibility that some kind of gastrointestinal ‘change’ may have been involved in the transient improvements.

The origin of the lesions in the spiral valve could have been due to the presence of a parasitic infection (e.g. metazoan parasites, protozoa, etc.) (Stokof, 1993). Isolated bacteria could therefore have represented opportunistic pathogens colonising an already compromised organ. Interestingly, no endo-parasites or protozoa were observed on necropsy. It is possible that the oral anti-helmithic treatment of 17 November, 1999 (i.e. nine months before the death of the specimen) was responsible for the absence of any parasites post-mortem.

An alternative possibility is that isolated bacteria could have altered the tissues of the spiral valve directly, through the production of necrosing toxins. This has been previously observed in teleost fishes and some shark species (Grimes et al., 1985; Fouz et al., 1995).

Regardless of the initial causative agent, as the condition progressed bacterial infection may have played a role in exacerbating or even perpetuating the buoyancy problem. Ultimately, death probably resulted from a profound systemic infection and septicaemia. The observed leucocytosis is consistent with
the suggestion of a progressive infectious disease (Stokoskof, 1993). It is also known that some bacteria of the genus Vibrio spp. produce haemolytic toxins and this could account for the unusual aspect of some of the erythrocytes and the alteration of blood values.

The following cautious conclusions can be suggested:

1. Because air injection alone was not sufficient to solve the condition, it seems likely that the problem causing air loss was chronic in nature. It is possible that the condition was in some way related to damage observed in the spiral valve. An impaired function of the spiral valve may have prevented the shark from avoiding excess fluids and retaining an adequate supply of air in its stomach to regulate buoyancy. The unusual posture of the animal may have therefore been the result of a spiral deformation caused by the resulting chronic negative buoyancy.

2. Injection of air into the stomach appeared to provide a good means of addressing an acute case of negative buoyancy in C. taurus. In this case, it seemed that the treatment was long overdue and ultimately treated a secondary symptom of a chronic causative agent. In more acute cases, where air loss is due to a transient cause, introduction of air into the stomach of the shark may be sufficient to rectify the problem (as per Smith, 1999). It is the opinion of the authors that prompter action in this case (i.e. expeditious air injection, in conjunction with an appropriate anti-biotic regime) may have prevented a chronic condition from

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Fig. 1: Sand tiger shark (Carcharodon taurus), with buoyancy compensation problems, showing unusual swimming pattern.