Is your fish "bent" and will it survive?

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Fish, like humans, can get "bent" when exposed to rapid changes in pressure during capture. The bends, or decompression sickness, is a syndrome associated with a rapid and extensive reduction in environmental barometric pressure (Philp 1974). Because the bends is caused by the application of basic physics to living organisms, it is reasonable to expect that fish suffer bends in a manner similar to humans. Bends has been studied in humans involved in deep-sea diving, high altitude aviation, and underground engineering projects since the beginning of last century. Indeed, the early theories of Haldane and associates (Boycott et al. 1908) are still used today for modelling decompression schedules. Most of our limited understanding of the effects of the bends in fish is based on our knowledge of the bends in humans.

Barotrauma is defined here as all the physical effects of rapid and extensive reduction in barometric pressure. Of the barotraumas, the bends is the most well known, but there are other types of barotraumas that affect both humans and fish. Although most of the barotrauma damage occurs internally and thus is invisible, there are some external symptoms.

"Bent" fish are most likely widespread in the live reef fish trade, as most of the species that have been examined were found to suffer symptoms of decompression sickness after capture from shallow depths of 10 to 15 metres (m) (see Histopathological studies, below). To increase your understanding and awareness of "bent" fish, I will explain how fish get "bent", describe other barotraumas in fish, and discuss some useful methods to both prevent pressure-related mortality and increase survival of fish suffering barotraumas.

Physical laws of nature: Boyle's Law

Laws of physics govern barotraumas in animals. Gases are highly compressible, and at constant temperature the pressure of a given volume of gas varies inversely to its volume (Boyle's Law). Sea pressure increases one atmosphere (atm) every 10 m of water from the surface. Thus, pressure increases will halve the volume of air in a balloon at 10 m, reduce it to one-third its volume at 20 m, and so on (Fig. 1). The balloon represents the swim bladder in a fish, which must be kept at a constant volume to maintain neutral buoyancy (Pelster 1997). As the ambient pressure changes, gas is moved in or out of the swim bladder, so when the fish swims deeper, gas is taken into the swim bladder from the bloodstream. The reverse occurs when the fish swims upwards in the water column. As the greatest change of pressure occurs in the top 10 m, most of the damage to captured fish occurs during the last part of their ascent.

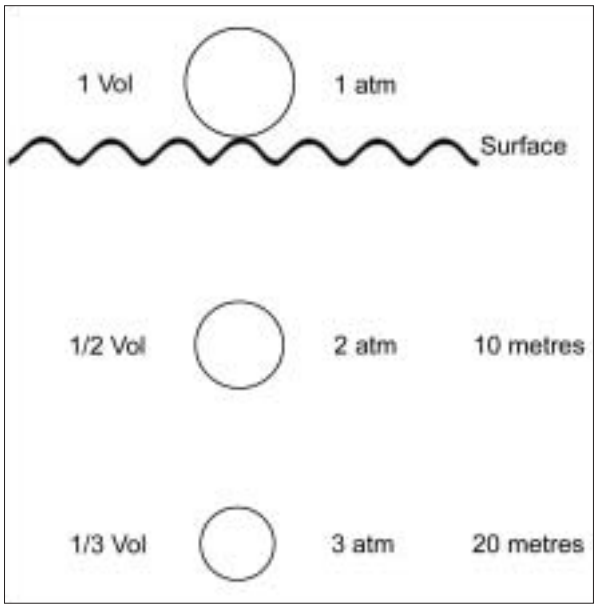

Figure 1. A demonstration of Boyle's Law: the effects on the volume of gas in a balloon as pressure increases with depth.

Partial pressures

As a fish descends, the pressure of air in the swim bladder equalises with the ambient water pressure, and the partial pressures of the individual gas components of the air increase. Air is made up of 79 per cent nitrogen and 21 per cent oxygen. Also, seawater, regardless of depth, is saturated (normal atmosphere as a reference point) in dissolved nitrogen

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(Saunders 1953). Nitrogen is present at a partial pressure of 0.79 atm at all depths in ocean seawater and it is the most common of the inert gases. Decompression sickness is caused by the formation of inert gas bubbles in the blood and tissues as the result of a sudden lowering of the ambient pressure. As the most common inert gas, nitrogen usually causes decompression sickness. All inert gases dissolve in the blood and tissue according to Henry's Law, where the dissolved concentration for any given gas equals its absorption constant multiplied by its partial pressure.

At any given depth a fish's body will absorb nitrogen gas through the gills and into the blood stream until equilibrium is reached and no more gas is absorbed (saturation). A decrease in absolute pressure results in a decrease in the maximum nitrogen that can be stored and nitrogen must be removed from supersaturated tissues according to Henry's Law. If decompression is slow, the excess nitrogen can be removed via the blood to the gills. During capture of the fish, however, rapid decompression saturates the rate of nitrogen elimination. Just like the formation of bubbles associated with opening a soft drink bottle, the nitrogen leaves solution from the blood and tissues as bubbles, until the concentration of dissolved nitrogen re-establishes equilibrium with the reduced partial pressure in the swim bladder. The formation of intravascular bubbles obstructs the blood stream, slowing the blood flow to the gills and further reducing the rate of nitrogen elimination. The stationary bubbles that accumulate in the bloodstream and tissues lead to the symptoms of decompression sickness.

The function of the swim bladder

In terms of Boyle's Law, swim bladders in fish are analogous to lungs in humans. Although some fish (physostomes) can release expanding air from their swim bladder through a pneumatic duct directly into the gut (Sauders 1953), other fish (physoclists) cannot. These physoclist fish have a well-developed capillary mesh that supplies blood to the swim bladder (Ferguson 1989) and is the interface for gaseous exchange. Thus, when ambient pressure reduces, the expanding gas in the swim bladder must be removed via the bloodstream.

All benthic reef fish can be assumed to have closed swim bladders, as physostomes are generally shallow freshwater species (such as carp and trout) that swallow surface air for buoyancy. Marine finfish exceptions are herring-type fishes, such as Atlantic mackerel (*Scomber scombrus*), which require greater depth flexibility and speed in moving through columns of water (Schmidt-Nielson 1997).

The effects of rapid depressurisation on the swim bladder

In many fish, especially benthic dwellers that do not usually swim up and down the water column, the drastic increase in the volume of gas during depressurisation at capture will inflate the swim bladder. The size of the inflated swim bladder at the surface increases with depth of capture and the swim bladder will rupture when the volume of gas becomes too great.

Symptoms of gas bubbles

During the rapid depressurisation at capture, gas bubbles formed from two sources (from gas exchange from expanding air in the swim bladder and from dissolved nitrogen in the body tissues) are released into the bloodstream. These intravascular gas bubbles can cause air embolism, blocking the flow of blood, and thus oxygen, to the tissues. As the blood supply from the capillary mesh of the swim bladder leads directly to the heart, large bubbles can cause a "heart attack" (Feathers and Knable 1983). Bubble formation in the tissues also leads to rupturing of cells, haemorrhaging and clotting, as well as other haemotological changes (Kulshrestha and Mandal 1982). If bubble pressure is great enough, the blood vessels can rupture, resulting in the haemorrhaging of blood into body tissue and the formation of clots at the damaged site.

Mortality studies

Most studies on the effect of barotraumas on both marine and freshwater fish have examined rates of mortality at differing depths because any release mortality reduces the effectiveness of legal minimum lengths as a management tool. Release mortality is an important component of fishing mortality in stock assessments. The general conclusion of research into the mortality of a range of fish species is that there is an inverse relationship between survival and capture depth (e.g. largemouth bass (*Micropterus salmoides*), Feathers and Knable 1983; red snapper (*Lutjanus campechanus*), Gitschlag and Renaud, 1994; snapper (*Pagrus auratus*), St John and Moran 2001). Thus, capture depth plays a critical role in the survival of released reef fish in both freshwater and marine environments due to barotraumas caused by depressurisation.

In one study, a hyperbaric chamber was used to simulate capture to study the effects of rapid decompression on largemouth bass (Feathers and Knable 1983). Fish depressurised from all depths showed some signs of barotraumas such as bloating or external haemorrhaging. Severe internal haemorrhaging and formation of gas bubbles in

the blood occurred in fish decompressed from depths greater than 18.3 m. Mortality of largemouth bass was 40 per cent at the pressure corresponding to 18.2 m, and 47 per cent at 27 m. Survival was strongly correlated to the degree of internal damage although swim bladder inflation was not always correlated to internal damage. Total mortality was directly related to the magnitude of decompression.

In salmonoids, both the effects of the bends and the period between onset of signs of stress and death were considerably longer for larger fish than for small fish (Beyer et al. 1976). Bubbles were visible in the fins and tail of small salmonoids but not in the larger fish. Beyer et al. (1976) attributed their findings to the relationship between bubble size and critical blood vessel size. Although gas bubbles range in size in all fishes, a bubble of a given size is more likely to create an embolism or blockage in a small fish — with its narrower blood vessels — than in a large fish. Other evidence suggests that older and larger fish could be more susceptible to decompression sickness. For example, increasing age and body weight increases the susceptibility to decompression sickness in man (Shilling et al. 1976). The amount of nitrogen released can be expected to be proportional to the size of the fish and its proportion of fat, as nitrogen is more soluble in fat. The solubility coefficient for nitrogen gas in fat tissue (0.067, for biological fluids at 37° C) is five times larger than in lean tissue (0.012) or blood (0. 013, Shilling et al. 1976). In spite of these factors, size was found not to significantly affect the degree of barotraumas in largemouth bass (Feathers and Knable 1983).

In general, benthic dwelling fish that never swim near the surface are more susceptible to barotraumas as they do not appear to have the physiology to dump gas from swim bladders quickly and cope with intravascular gas bubbles. Whether or not other species that are more mobile within the water column have better systems to cope with rapid changes in pressure remains to be discovered.

Histopathological studies

In Western Australia, a release mortality experiment and histopathology studies have been done on two species of physoclist fish, snapper (*Pagrus auratus*) and Western Australian (WA) dhufish (*Glaucosoma hebraicum*) (Ashby 1996; Longbottom 2000; St John and Moran 2000; St John unpublished data). Caging experiments on release mortality of both species found that approximately 70 per cent of fish died at depths greater than 45 m (St John and Moran 2001, St John unpublished data). The pattern of mortality, however, differed between the two species at depths less than 45 m, as snapper were found to be more robust to barotraumas, with higher survivorship.

The histopathological studies examined acute damage to organs such as the heart, kidney, liver, spleen, and gills by assessing the degree of bubble formation, clotting, and haemorrhaging in these tissues (Ashby 1996; Longbottom 2000). WA dhufish were caught from two depths, below and above 20 m (range = 9 to 73 m), whereas snapper were caught from three depths shallower than 35 m: 10–15 m, 20–25 m, and 30–35 m. Bubbles, clotting and haemorrhaging were found in all WA dhufish from both depth categories, though damage was greater in the deep-water fish (Ashby 1996). Exopthalmia (haemorrhaging, swelling, and in extreme cases the rupture of the cornea) and rupture of the swim bladder also occurred in some deep-water fish.

Organ damage (including haemorrhaging, formation of small bubbles, and tissue displacement) occurred in snapper at all depths sampled (Longbottom 2000). The percentage of damage caused by haemorrhaging and tissue displacement, however, was significantly higher at depths greater than 25 m (Longbottom 2000). As mortality of snapper was relatively low at depths less than 35 m (St John and Moran 2001), many snapper survive a relatively high level of barotrauma. The degree of barotraumas in snapper was not related to fish size (length or weight) (Longbottom 2000). Between 10 and 40 m, mortality in WA dhufish was found to be higher than in snapper, and thus this species appears to be more susceptible to mortality from barotraumas.

Although there was no clear correlation between external symptoms of barotrauma and internal damage in red snapper (Gitschlag and Renaud 1994), swim bladder inflation was a significant indicator of internal organ damage in snapper (Longbottom 2000).

Condition, liveliness, and fish stress patterns of a fish on the deck of a boat after capture have not been found to be related to the degree of internal damage or survival (Neilson et al. 1989; personal observation).

Effects of inflated swim bladders

Overinflated swim bladders damage other internal organs inside the fish. Fish with severely overinflated swim bladders will be stressed in holding tanks because they are positively buoyant. They float upside down on the surface until the gases within their swim bladder have equalised to the surface pressure, which may take several hours.

Floating fish are more susceptible to mortality from heat stress or exhaustion from trying to swim normally. Venting or piercing the swim bladder with a sharp tube releases the gas and allows the fish to swim normally (see box on "how to vent fish" and Figs. 2 and 3).

Decompression of fish may be better than venting the swim bladder. For example, a study comparing the survival of yellow perch (*Perca flavescens*) between the two methods found that survival was higher in fish immediately returned to depth in cages (Keniry et al. 1996). Fish species that regularly show signs of severe barotraumas after capture may require decompression stops. Decompression stops can be done during or after capture (see box on decompression stops for "bent" fish). When losses to sharks or other predators are a problem during capture, decompression after capture is the best option for "bent" fish.

Summary

Although much more research into the problems of barotraumas in captured fish is required, the results of the research to date provide useful information for fishers in the live reef fish trade:

1. Most fish captured from depths greater than 10 m will suffer barotraumas.
2. Both the severity of barotraumas and mortality will increase with depth of capture.
3. The degree of barotraumas and rates of mortality for any given depth varies among species.
4. Generally, the extent of external symptoms of barotraumas reflect internal damage, but the condition or stress pattern of a fish on deck is not always related to mortality.
5. The size of the fish may affect the degree of barotauma.

As methods to cope with barotraumas vary among species and fishing methods, there will be no one method to treat "bent" fish. The following four points may help you develop the best practical procedures to treat "bent" fish.

1. Look for symptoms of barotraumas in all fish and note their severity (see Figure 4 and Table 1). *Tip: Keep a record of depth, handling methods, and degree of barotraumas for each fish to determine patterns of barotraumas in your target species.*
2. Decide on treatment: Venting is best for mild cases; decompression is best for more severe barotraumas. Kill severely "bent" fish.
3. Practice quick, sterile venting of swim bladders.
4. Keep records of treatment and outcomes of each fish to develop and refine decompression schedules for your target species.

How to vent fish

Venting is best done using a sterile, low-gauge, hypodermic needle (e.g. 1.2 x 38 mm). It can be inserted at a 45° angle under a scale below the lateral line near the tip of the pectoral fin (Fig. 2). After insertion, apply gentle pressure to the ventral surface of the fish to remove as much air as possible. Clear and sterilise the needle before re-use. Remember to keep fish out of direct sunlight during any procedure on deck and handle them carefully using wet towels and hands.

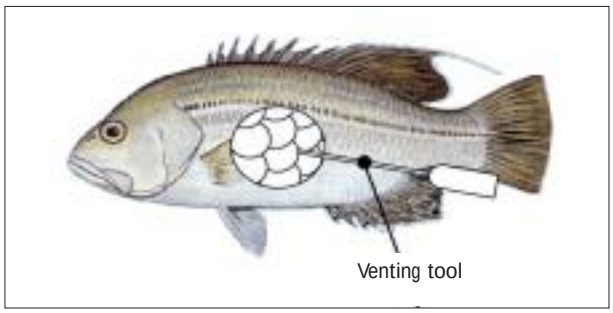

Figure 2. **How to vent a Western Australian dhufish.**

Figure 3. **Venting an undersize Western Australian dhufish.**

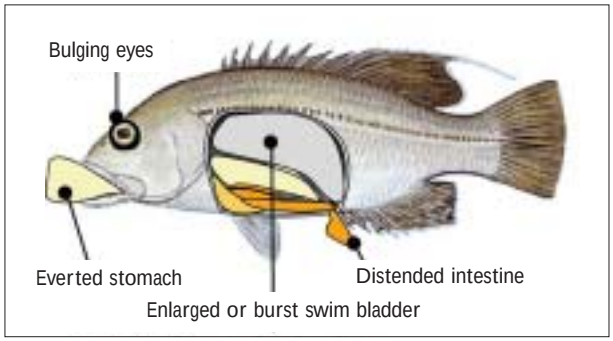

Figure 4. **Symptoms of barotraumas in Western Australian dhufish.**

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Decompression stops for "bent" fish

During capture, decompression stops may involve leaving a fish swimming on the line at 3 or 5 m below the surface for a few minutes while preparing a wet deck for capture. This method depends on the type of fish caught, as some species may tire and stress more the longer they stay on the end of the line.

Alternatively, a decompression stop or stops can be done after the fish has been brought on deck. This method may only be suitable on relatively stationary boats.

Although bent fish should be decompressed as soon as possible after capture, decompression at any stage should improve the health of bent fish. The exact decompression schedule would vary depending on fish species, depth of capture, and handling methods. Fish to be decompressed after capture could be put in a dark ventilated container and returned to specific depths for various times in weighted baskets or containers. These containers may need to be shark proof: made from metal and totally enclosed.

Table 1. External symptoms of barotraumas, graded from mild to severe.

	External Symptoms	
Degree of barotrauma	Associated with swim bladder	Associated with gas bubble formation
Mild	Inflated tight stomach	Small bubbles visible at the gills, fins, and eyes
Medium	Very inflated abdomen, distortion of scales	Some haemorrhaging at the gills and fins
Severe	Stomach everted from mouth, protruding eyes, distended intestine	Large bubbles and/or haemorrhaging at the gills, fins, and eyes