Assessment of injury and mortality from rapid decompression on crucian carps

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Abstract. Knowledge of barotrauma injury mechanisms is required in the fish-friendly turbine design. Rapid decreases in pressure is one of the main reasons that cause injury and mortality to fish that pass through hydropower machines. A steel tank and related pressure regulation components were designed to study the barotrauma injury thresholds of crucian carps. Decompression from 700 kPa to 6 kPa in 0.3 s was implemented in this system with dozens of yearling and subyearling crucian carps. The fish swim bladder and other organs were observed to find the main reason for injury after the rapid decompression. The tested fish were kept for at least 48 hours to study the physiological status in different observation periods. The direct and indirect decompression induced mortality after the test was observed and analyzed.

1. Introduction

Maximize the survival of turbine-passed fish continues to be an issue in both hydropower development and environmental protection areas. Lots of fish-friendly turbine designs [1, 2] were developed to meet the electricity generation efficient while minimizing damage to fish and fish habitats. Knowledge of injury mechanisms that impact fish and fish's tolerance of these injury were required in the fish-friendly turbine design [3]. Blade strikes, rapid decreases in pressure, shear stresses, and cavitation were the main sources of injury and mortality inside the turbine passage [4], while rapid change of pressure caused fish injury was mostly studied because it's unavoidable for fish passing through turbines. The fish passing through a hydraulic turbine experiences a rapid decompression (less than 1 s) after a compression which lasts minutes in the penstock. Then the pressure gradually increased to the free stream pressure. The decompression process turned out to be more harmful than the compression process for fish according to earlier studies about the damage to fish caused by compression and decompression [5].

Several researchers have drew the conclusions that rapid decompression can lead to fish injury and mortality and fond that rapid pressure changes were possible to lead to barotraumas, most notably rupture of the swim bladder, exophthalmos, emboli and hemorrhaging [6]. The likelihood and degree of damage are related to both the pressure to which a fish is acclimated and the pressure to which it is exposed during turbine passage [7].

Few studies, however, have quantified barotrauma resulted from rapid decompression in crucian carps. A series of experiments were implemented on crucian carps to find out the decompression caused injury mechanism and fish-friendly turbine design guidelines in this research.

2. Fish culture and the rapid decompression system

Crucian carps with different size was tested to establish systematic acknowledgement of its decompression injury mechanism upon fish size, pressure change ratio and nadir pressure. The crucian

carps were held in a 24 m^3 recirculated water pond for at least two weeks before tests. The water quality in the pond was controlled by using UV lamps, biological filter and water quality monitoring system. Adding fresh water, draining dirty water, using oxygen machine were also measures to maintain water quality according to ammonia, PH, dissolved oxygen and temperature daily detection. The mean PH of the water in the pond was 7.60 ± 0.30, dissolved oxygen was 7.4 ± 0.5 mg/l and temperature was 21 ± 2℃ during the experiment period.

Crucian carps after the decompression was kept in the same pond separately for more than 48 hours for observation and histological examinations.

Figure 1. Observation tank and pressure control system

As shown in figure 1, a pressure control and fish holding system was designed to simulate the pressure history of fish passing through a hydraulic turbine. A tank with observation windows was connected to two larger tanks, one of them (not included in figure 1) connected to air compressors and the other (the left higher tank in figure 1) connected to vacuum pumps. With a volume of 100 l, the tank can hold fish equal to or shorter than 0.3 m. Water supply and drainage system was designed to keep the water inside the observation tank fresh and safe for fish. Electromagnetic valves were used to pressurize and depressurize the observation tank and ball valves were used to control the compression and decompression speed. The system can drop the pressure inside the observation tank from 700kPa to 4kPa (close to the vapor pressure of water) in 0.3 s.

3. Experiment procedures

Quality tested water was firstly poured into the tank, several crucian carps were then put into the observation tank before it was sealed. Open or close the electromagnetic valves to pressurize or depressurize the observation tank according to experiment needs. Take the crucian carps out from the tank and held them in isolated zones in the recirculated water pond for subsequent observation or histological examinations.

To make the rapid decompression process reasonable on the basis of the pressure history that fish would experience in passing through a hydro-turbine, the water inside the observation tank was firstly pressurized to a high pressure (700kPa) (Stage 1), and then depressurized to the nadir pressure (6kpa) (Stage 2), and lastly, increased to 101 kPa (Stage 3). The Stage 2 was the rapid decompression process in the whole pressure change experiment.

In order to focus on the rapid decompression introduced injury and mortality, the Stage1 and 3 were studied earlier to find the appropriate pressure change speed that introduced no obvious harm to fish. The pressure evolution in the rapid decompression experiment was shown as figure 2.

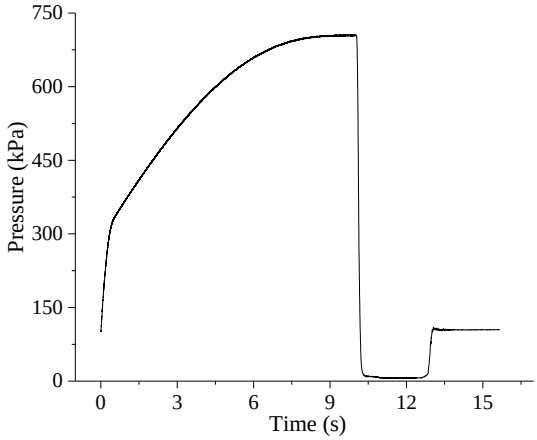

Figure 2. The pressure evolution during the rapid decompression process

4. Results and discussion

Totally 20 Size I (200g ± 20g), 33 Size II (100g ± 10g), 49 Size III (50g ± 5g) crucian carps were exposed to the rapid decompression test. After the experiment, the fish were held separately in the pond for different period observation.

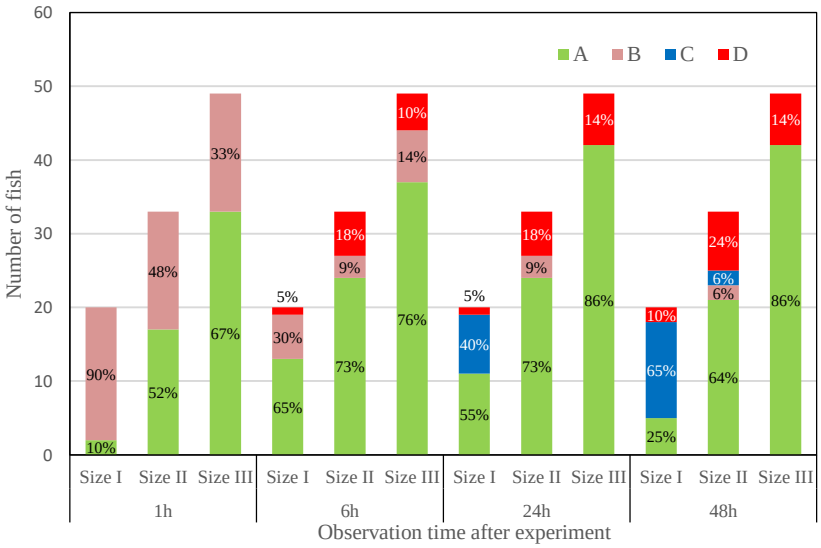

Figure 3. Fish status at different observation time after experiments
(A: Normal; B: Loss of equilibrium; C: External signs of trauma; D: Dead.)

As shown in figure 3, 90% of big size crucian carps (Size I) lost equilibrium in the first hour after experiment, while 48% and 33% of Size II and Size III failed to keep balance. Swim bladder is the main organ for fish to maintain equilibrium, crucian carps lost equilibrium was mainly caused by the injury or rupture of the swim bladder.

During the decompression process, the air in the swim bladder quickly inflated as the pressure decreased. If the pressure decreased by half, the gas in the swim bladder doubled. The elasticity of the swim bladder is limited, which means that the volume of the swim bladder will only expand as the gas inside inflated before its limit. The swim bladder may lost part of its air through fish throat if the pressure decrease speed is slow, or just rupture if the decompression speed is too fast. After the decompression, the swim bladder shrank greatly as the gas left in the swim bladder shrank quickly when pressure recovered to 101 kPa. As shown in figure 4(b), the swim bladder of the fish lost equilibrium was almost empty while the swim bladder (figure 4(a)) of normal fish was full. Bubbles come out from the gills were observed for most of the curcian carps during the rapid decompression in the experiments. The

ruptured swim bladder can only be noticed in the anatomical observation, as shown in figure 4(c). Clotted blood was found inside some of the ruptured swim bladder, as shown in figure 4(d). Hemorrhaging from caudal vein often extended into the swim bladder if it was ruptured.

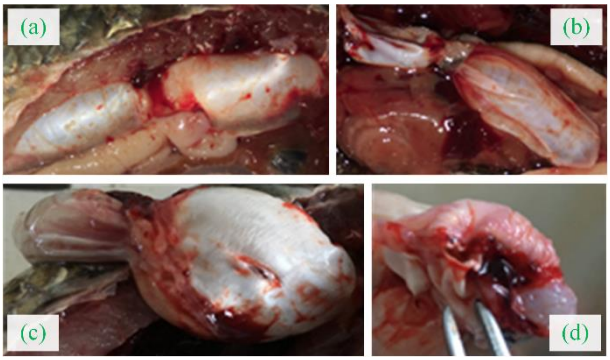

Figure 4. Swim bladders of crucian carps after rapid compression experiments

According to several times of anatomical observation, crucian carps can keep balance in water with a 25% or higher percentage inflated swim bladder. Some of the crucian carps lost less than 75% of the gas in their swim bladder during the rapid decompression, they were able to keep balance and looked normal after the experiments. Some of the crucian carps lost more than 75% of the gas in their swim bladder or got their swim bladder ruptured, they lost equilibrium after the experiment.

As shown in figure 3, in the second period (1~ 6 hours after the experiment), some of the fish lost equilibrium gradually recovered, while some of them died. The one lost gas gradually recovered by breathing in air slowly to their swim bladder, while the one got their swim bladder ruptured was not able to recover. Big size fish with larger swim bladder were easier to be influenced by the sudden decompression, however, their stronger body and regulatory capacity help themselves faster to restore balance when compared with smaller size fish. For Size I, 55% of the 20 crucian carps recovered in 6 hours, while 5% of them died.

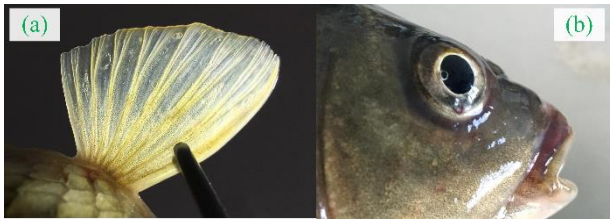

Figure 5. Bubbles in fins and fish eyes after rapid compression experiments

Swim bladder rupture, however, was not the only reason for mortality. The undissolved gas rapidly expanded not only in the swim bladder, but also inside other organs, dissolved gas become undissolved and released into tissues as the solubility of gas dissolved in blood decreases, these phenomenon can also lead to injury and mortality. Emboli in the fins and in the eyes were observed at 24th hour after experiment, as shown in figure 5.

External signs of trauma was found in the third period (7 ~ 24 hours after the experiment) as shown in figure 3, 40% of Size I fish were found carried different external signs of trauma, such as faded of fish scales, loss of surface mucus and hemorrhage in fish belly near the fins. With the increase of time, the hemorrhage area expanded continuously, as shown in figure 6.

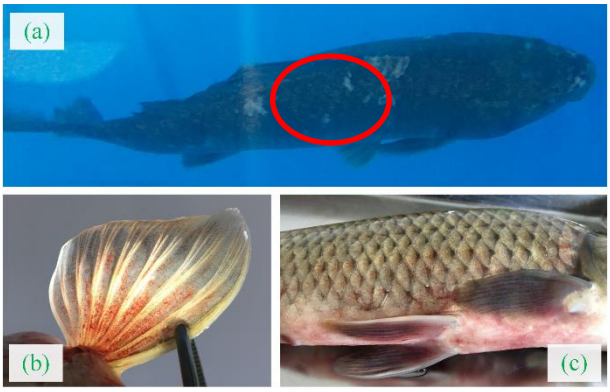

Figure 6. External signs of trauma on crucian carps

It can be seen that, Size I crucian carps were easier to be influenced with hemorrhage comparing to Size II and Size III. None of Size III crucian carps was observed with external signs of trauma. Mortality percentage increased in Size I after external signs of trauma was observed, which means that, fish died in first 6 hours were caused by rupture of swim bladders or injured organs, fish died after 24 hours were caused by hemorrhage and trauma.

Hemorrhage was leaded to by the rupture in the caudal vein. The rupture in the vein would markedly reduce blood pressure and thus fail to supply heart and other organs with adequate blood pressure. And sometimes hemorrhage in pericardial sac would also lead to mortality.

It can be seen from Figure 3 that, another 25% of 20 Size I and 6% of 33 Size II crucian carps were found external signs in the fourth period (24~48 hours after experiments), 5% of 20 Size I and 6% of 33 Size II crucian carps were dead. While the injury condition of Size III fish was the same with it in the third period. Which means that, the larger the fish size is, the easier they got trauma and hemorrhage, the greater probability of death leaded by trauma after 24 hours.

From figure 3 we can learn that fish passing through turbines would not die immediately after the rapid decompression. This doesn't mean that the simulated turbine passage is not dangerous for crucian carps, on the contrary, the loss of equilibrium just after the decompression may last 6 or more hours brought in more dangerous such as disable to dodge from high supersaturation area, high shear stress area and predators. And the mortality in the following 48 hours cannot be neglected. If the observation time was longer, such as 5 days, higher mortality rate would be observed. Since C status on fish showed a growing trend from 24 hours to 48 hours after the decompression as shown in figure 3.

5. Conclusion

A steel tank and related pressure regulation components were designed to assessment the injury and mortality of crucian carps from rapid decompression. The fish were observed for 48 hours after the experiments, and anatomical investigation were carried out to study the swim bladder and other organs injury condition.

Gas leaking from the swim bladder and rupture of the swim bladder were the main reasons for the loss of equilibrium after the decompression. Curcian carps gradually recovered by breathing in air slowly to their swim bladder if the swim bladder did not burst. While the ruptured swim bladder may be the reason for death for first 6 hours after the rapid decompression.

Dissolved gas became undissolved and released into tissues as the solubility of gas dissolved in blood decreased, this phenomenon can also lead to injury and mortality. External signs of trauma were observed on fish during 24~ 48 hours after the experiments, and these signs were highly related to mortality during this period.

Knowing the lethal nadir pressure and understanding the relationship between the speed of pressure change and severity of barotrauma injury are important ways for protecting fish populations because it can guide the turbine design and operation optimization. Study of the injury and mortality mechanism

should not focus on the equilibrium and mortality in short period of time after the experiments, but also focus on the gradually occurring lesions and incentives in a relatively long observation period.

ACKNOWLEDGMENT
The authors gratefully acknowledge the support from the IWHR Research & Development Support Program (HM0145B552016).

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