

**CARDIOVASCULAR RESPONSES OF WHITE STURGEON, *ACIPENSER*
TRANSMONTANUS, TO ACUTE HYPOXIA: PRELIMINARY RESULTS**

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INTRODUCTION

The white sturgeon, *Acipenser transmontanus*, is a large benthic semi-anadromous, oxyconforming fish (high critical PO_2) found in estuarine and coastal marine habitats of the northeastern Pacific basin. Burggren and Randall (1978) suggested that juvenile white sturgeon (0.9 Kg) at 15° C enter an energy-conserving, hypometabolic state when exposed to hypoxia. Recent studies on juvenile white sturgeon indicate that with acute exposure to hypoxia (60-90 torr PO_2) the hypometabolism manifests itself primarily as significant reductions in oxygen consumption rate and swimming activity (Crocker and Cech 1993).

If, during hypoxic exposure, oxygen consumption rate is reduced and there are no biochemical (e.g. changes in hemoglobin- O_2 binding), ventilatory (e.g. increase in ventilatory stroke volume), or circulatory adjustments (e.g. lamellar recruitment) to increase the oxygenation of the blood at the gills (Holeton 1980), systemic hypoxia may ensue. In many fish species, systemic hypoxia is an oxygen-limiting condition which threatens proper cardiac performance (Driedzic 1992).

Consistent with the observation that swimming activity and oxygen consumption are reduced during hypoxic exposure is the occurrence of hypoxic bradycardia; an energy-conserving strategy encountered among several vertebrate classes (Hill and Wyse 1989). A reduced heart rate during hypoxia presumably increases cardiac O_2 extraction by increasing blood residence time in the heart. Hypoxic bradycardia is typically accompanied by an increase in blood pressure (BP) and cardiac stroke volume (SV) (Holeton 1980). While the increase in SV may facilitate the perfusion of the trabeculated ventricular myocardium, the increase in BP increases ventricular stroke work (Berne and Levy 1986). If the transition from normoxia (aerobic conditions) to systemic hypoxia (oxygen-limiting conditions) is not accompanied by similar compensatory increases in glycolytic ATP production, which could fuel the increased cardiac power demand, then cardiac performance will be impaired (Driedzic 1992).

We are investigating the cardiac responses to acute, progressive hypoxia in juvenile white sturgeon at 23° C. Our primary objectives are: (1) to determine what PO_2 causes a significant reduction in HR, (2) to assess the time course for the development and

maintenance of bradycardia. If hypoxic bradycardia is a marshalled response to environmental hypoxia, then it should be concomitant with the reduced oxygen consumption rate and activity.

METHODS AND MATERIALS

Juvenile (1 year old, 550 - 700 g wet weight range) white sturgeon, *Acipenser transmontanus* were maintained at the University of California Davis Wildlife and Fisheries Biology fish holding facility. They were held in a large (1.5 m, diameter), circular tank supplied with a continuous flow of air-equilibrated water at $22 \pm 1^\circ \text{C}$. The fish were fed (Silvercup trout pellets) and fecal material and excess food were siphoned out of the tank daily.

The fish were individually selected and placed into a canvas surgery sling fitted with a continuous gill irrigation system (Crocker and Cech 1993). They were anesthetized with 0.1% tricane methane-sulfonate (MS-222; Sigma Chemical Co.) and then non-occlusively cannulated (PE-50) in the dorsal aorta using the caudal approach. The cannula was anchored (3.0-silk suture) at the point of entry and at one other point (lateral body) for extra support. The cannula was filled with heparinized (100 IU/ml) saline solution and the distal end plugged.

Each fish was recovered from the anesthesia and placed into a 14 L acrylic cylinder with a continuous flow of air-equilibrated water for ≥ 24 h. Cannulae were flushed periodically to prevent clotting. Immediately before experimentation, cannulae were connected to a Statham physiological pressure transducer (Model P23BB), which was connected to a calibrated Gilson IC-MP module/physiological recorder for BP and the HR measurements.

Heart rate was determined by counting systolic peaks on the strip record and then dividing by the appropriate time interval. The mean arterial pressure (MAP) was calculated for each PO_2 exposure regime using the following equation:

$$\text{MAP} = P_d + 1/3 (P_s - P_d)$$

where: P_d is diastolic pressure (mmHg) and P_s is systolic pressure (mmHg) (Berne and Levy 1986).

After the normoxic recording period, the water PO_2 was progressively decreased over 45 min by bubbling nitrogen gas through a multichambered gas stripping column, leading to the fish chamber and exposing fish water at 40-45 torr PO_2 . Dissolved oxygen tension was monitored with an electrometric oxygen analyzer system (Radiometer Model PHM 71/D616/E5046).

RESULTS

All yearling white sturgeon showed a marked bradycardia during severe hypoxia, with no change in MAP. The mean MAP during normoxia was 20.5 mm Hg ($n = 3$) and the mean HR was 66 beats min^{-1} (Fig. 1). Exposure to hypoxic water (40-45 torr PO_2) resulted in a marked decrease in HR (30 beats min^{-1}), but no significant change in MAP (22 mm Hg, Fig. 2). After the hypoxic period, the nitrogen flow was replaced with air and the water PO_2 was returned to air-saturation levels. The heart rate and MAP immediately returned to normoxic levels.

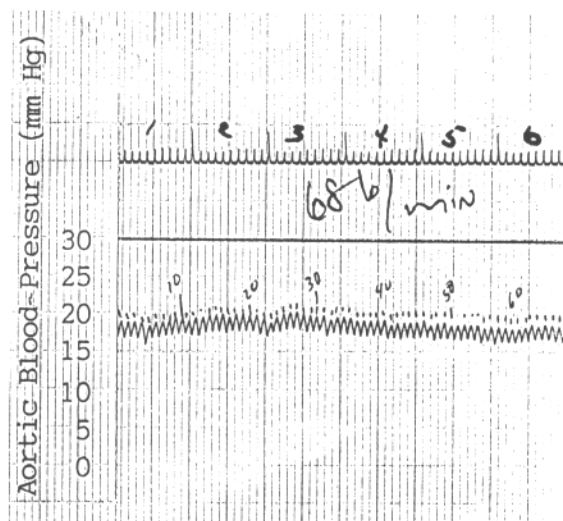


Figure 1. Strip recording of white sturgeon dorsal aortic pressure during normoxia (155 torr PO_2). Paper speed was 10sec/cm. The calibration limits (0 to 20 mm Hg) are indicated on the left of each trace.

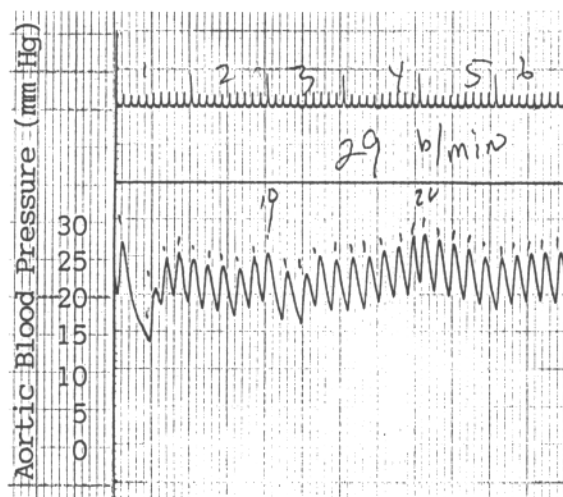


Figure 2. Strip recording of the pressure pulse for white sturgeon during hypoxia (40 torr PO_2). Paper speed was 10sec/cm. The calibration limits (0 to 20 mm Hg) are indicated on the left of each trace.

DISCUSSION

The cardiovascular responses of white sturgeon, *Acipenser transmontanus*, to severe hypoxia were investigated. Hypoxia-induced bradycardia was observed when the ambient PO_2 was \leq 40 torr. The bradycardia was of rapid onset and persisted throughout the hypoxic bout. During hypoxia, there was no appreciable change in MAP. These preliminary data suggest that white sturgeon may employ bradycardia as a strategy for energy conservation during certain environmental conditions. Our previous studies on metabolism (oxygen consumption rate) and swimming activity clearly indicate that white sturgeon do marshall hypometabolism when exposed to acute hypoxia (Crocker and Cech 1993). Bradycardia appears to accompany severe hypoxia (\leq 40 torr PO_2) exposure in this chondrosteian fish. Ongoing

studies will better define PO₂ thresholds of cardiovascular (e.g. HR) and metabolic (e.g. critical PO₂) responses.

LITERATURE CITED

Berne R.M. and Levy M.N. (1986) Cardiovascular physiology. 5th Edition. C.V. Mosby Company, St. Louis.

Burggren W.W. and Randall D.J. (1978) Oxygen uptake and transport during hypoxic exposure in the sturgeon *Acipenser transmontanus*. *Respiration Physiology* 34: 171-183

Crocker C. E. and Cech J.J. Jr. (1983) Effects of acute hypoxic exposure on oxygen consumption rate and activity in juvenile white sturgeon, *Acipenser transmontanus*. Proceedings of the 123rd Annual Meeting of the American Fisheries Society: Shared responsibility for shared resources. Portland Oregon, 1993.

Driedzic W. R. (1992) Cardiac energy metabolism. pp. 219-266 in Hoar W.S. and Randall D.J. (eds.) *Fish Physiology*, Volume XII, Part A, The cardiovascular system. Academic Press Inc., San Diego.

Hill R. W. and Wyse G.A. (1989) *Animal physiology*. 2nd Edition. Harper and Row, Publishers, New York.

Holeton G.F. (1980) Oxygen as an environmental factor in fishes. pp. 7-32 in Ali M.A. (ed.) *Environmental physiology of fishes*. Plenum Press, New York.