

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/287854740>

Respiratory and circulatory adaptations of Siberian sturgeon to environmental hypoxia

Article · January 1998

CITATIONS

4

READS

173

3 authors, including:



Valérie Maxime

Université Bretagne Sud, Vannes

25 PUBLICATIONS 974 CITATIONS

[SEE PROFILE](#)



Patrick Williot

Patrick Williot Sturgeon Consultant Company

233 PUBLICATIONS 3,604 CITATIONS

[SEE PROFILE](#)



Circulatory and respiratory effects of an hypoxic stress in the Siberian sturgeon[†]

V. Maxime^{a,*}, G. Nonnotte^b, C. Peyraud^a, P. Williot^c and J.P. Truchot^b

^aLaboratoire de Physiologie Animale, Université de Bretagne Occidentale, Brest, France

^bLaboratoire de Neurobiologie et Physiologie Comparées, CNRS URA 1126 et Université de Bordeaux I, Arcachon, France

^cCEMAGREF, Division Aquaculture et Pêche, Gazinet, France

(Accepted 2 January 1995)

Abstract

Changes in circulatory, ventilatory and acid-base variables were studied in Siberian sturgeon (*Acipenser baeri*) exposed to acute and severe hypoxia ($Pw_{O_2} = 10$ torr), followed by a rapid return to normoxia. This treatment caused a significant stress, revealed by the high levels of plasma catecholamines and cortisol. The moderate circulatory changes firstly observed would represent the effects of increased plasma catecholamine levels together with an increased adrenergic nervous tone on the cardiovascular system. Then, these effects were masked by a possible vagal reflex resulting in bradycardia. Deep hypoxia induced a ventilatory alkalosis combined with a moderate metabolic acidosis. The latter amplified concomitantly with a massive flush of lactate into the blood stream. The initial hyperventilation was followed by a deep ventilatory depression. During return to normoxia, hyperventilation resumed consistent with the repayment of an oxygen debt. Thus, the sturgeon, although considered as an archaic fish, developed the same adaptive responses as teleosts submitted to comparable hypoxic conditions.

Key words: Acid base, severe hypoxia; Fish, sturgeon (*Acipenser baeri*); Hypoxia, severe environmental; Mediators, catecholamines, cortisol; Ventilation, severe hypoxia

1. Introduction

The frequent occurrence of hypoxia in aquatic environment has entailed many studies carried out in

fish, primarily rainbow trout. They have dealt with various aspects of respiratory and circulatory responses to a decrease in ambient Pw_{O_2} (Tetens and Lykkeboe, 1985; Boutilier et al., 1988). Environmental hypoxia initiates immediate ventilatory and cardiovascular reflexes, allowing the maintenance of arterial oxygen saturation to its optimum level despite a reduced transbranchial oxygen gradient (reviewed by Fritzsche and Nilsson, 1993). The typical response of a fish to hypoxia consists in increasing its ventilation rate and amplitude and bradycardia. The nature of the mechanisms that regulate these processes has raised large interest. In particular, cir-

* Corresponding author. Tel.: (33) 98 01 65 37, Fax: (33) 98 01 63 11.

[†] This article is dedicated to Professor Claude Peyraud, whose recent death has deeply saddened us. The experiments that gave rise to the results reported here were instigated by him and have been successfully carried out and interpreted thanks to his continual encouragement and sound advice. The scientific community has lost a man endowed with high intelligence and unique humane qualities. We respectfully pay tribute to his memory.

culatory and ventilatory controls by catecholamines have been extensively studied. Changing levels of circulating catecholamines result in numerous physiological effects, both direct and indirect; all of them lead to either increasing or maintaining energy turnover and oxygen supply (for review, see Randall and Perry, 1992).

Our previous work (Nonnotte et al. 1993), showed that in spite of the chondrostei being among the most ancient of vertebrate groups, sturgeon (*Acipenser baeri*), like most other fish, exhibit typical O_2 regulatory behavior when subjected to graduate and moderate hypoxia. Besides, a slight lactic acidosis developed below a critical level of 40 torr Pw_{O_2} and became more marked during return to normoxia. In the face of these major results and by comparison with the response to hypoxia of another species of sturgeons, *Acipenser naccarii* (Randall et al., 1992) and of other studied fishes, the sturgeons seem to present a high ability to cope with the lack of ambient oxygen. Considering that the time course and severity of hypoxia greatly influence the degree of physiological responses of fish (Boutilier et al., 1988), the purpose of the present work was an attempt to determine the limits of this ability by measuring respiratory, acid-base and circulatory responses in the Siberian sturgeon when subjected to an acute and severe hypoxia followed by a rapid return to normoxic conditions. In order to test whether environmental hypoxia is a potent stimulus for catecholamines and cortisol release into sturgeon blood plasma as in teleosts, this study was completed by the measurements of plasma catecholamines and cortisol concentrations to assess the magnitude of the stress generated by hypoxia.

2. Materials and methods

Fish and water

Three-year old sturgeon (*Acipenser baeri*), each weighing about 1.5 kg were obtained from the experimental hatchery of CEMAGREF (St Seurin/l'Isle, France). Prior to experimentation, they were maintained for three weeks in a large outdoor circular tank supplied with well-aerated running tap water at seasonal temperature. They were fed once a day (1% of live weight) with a commercial diet

(Aqualim, France) until 48 h before the experiments.

The experiments were performed successively on 8 fishes, each fitted with a dorsal aortic cannula for arterial blood sampling and recording of circulatory variables and a cleithral cannula for recording hydrostatic pressure changes in the branchial cavity. Anaesthesia, surgical and post-operative procedures and the control of water acid-base balance were previously described (Nonnotte et al., 1993).

Each fish was placed in a respirometer supplied in closed circuit with normoxic or hypoxic water from two thermostatted (18 °C) tanks (120 L each). By operating a three-way tap, normoxic or hypoxic water was pumped at a constant flow rate (about 3 L·min⁻¹) to reduce as much as possible the time required to entirely replace the experimental medium used in the respirometer.

Experimental protocol

By using the set-up described above, the imposed deep hypoxia level (10 torr) was reached in 30 min. The average time course of O_2 partial pressure in water (Pw_{O_2}) during acute deep hypoxia and return to normoxia is depicted in Fig. 1.

Blood samplings and recordings of ventilatory and circulatory variables were performed at 10 min (Pw_{O_2} = 30 torr) and 30 min during the Pw_{O_2} decrease; then, at 2h and 5h30 from the beginning of return to normoxic conditions. In order to avoid possible disturbances created by blood sampling, recordings were always performed at first. Averaged values were obtained from continuous recordings during 10-min periods.

Blood samples (about 1.7 ml) were slowly withdrawn from the dorsal aortic cannula into pre-heparinized syringes. Oxygen partial pressure in arterial blood (Pa_{O_2}), extracellular acid-base characteristics and haematocrit were immediately determined from aliquots of whole blood (200, 240 and 25 μ l, respectively). The remaining blood was centrifuged (3000 rpm for 15 min) for subsequent measurements of adrenaline and noradrenaline (700 μ l), cortisol (150 μ l), main ions (150 μ l), lactate (50 μ l) and glucose (10 μ l) concentrations in the plasma.

Measurements of plasma catecholamines and cortisol

Plasma adrenaline and noradrenaline were extracted with boric acid gel (Affigel 601; Biorad)

activated by the Maruta method (Maruta et al., 1984) slightly modified. The efficiency of this extraction method, determined by an internal standard procedure was about 90%. The catecholamines eluted from the gel by 0.75 M acetic acid were separated by High Performance Liquid Chromatography and detected by means of an electrochemical device (Bio Analytical Systems).

Cortisol assay was performed by radioimmunoassay (CIS Bio International, ref: SB-CORT).

Measurements and calculation of circulatory variables

Dorsal aortic pressure was recorded by connecting the dorsal aortic cannula to a pressure gauge (Honeywell 156PC). The mean pressure (P_{DA}) and the difference between the diastolic and systolic blood pressures (P_{Puls}) were calculated according to the following relations:

$$P_{DA} = 1/3 [\text{systolic pressure} + (2 \times \text{diastolic pressure})]$$

$$P_{Puls} = \text{systolic pressure} - \text{diastolic pressure}$$

The heart rate (HR) was determined from the pulse rate on the P_{DA} record.

Measurements of respiratory variables

Frequency (VF) and amplitude (VA) of ventilatory movements were obtained by recording hydrostatic pressure changes in the branchial cavity, measured by connecting the catheter inserted through the cleithrum bone to a Honeywell 156P pressure transducer. The maximal change of pressure observed during each breathing cycle was used as an estimation of ventilatory amplitude. An average value of this variable measured for each fish in steady state during a 10-min period before the exposure to hypoxia was used as an arbitrary unit to estimate the ventilatory amplitude changes which occurred during hypoxia.

P_{aO_2} was measured by using a thermostatted P_{O_2} measuring cell (Radiometer E5046). After each measurement, the blood was returned to the fish.

Measurements of blood acid-base characteristics

Arterial blood pH (pHa) was measured by using a Radiometer microelectrode G222A calibrated with Radiometer precision buffer solutions type S1500

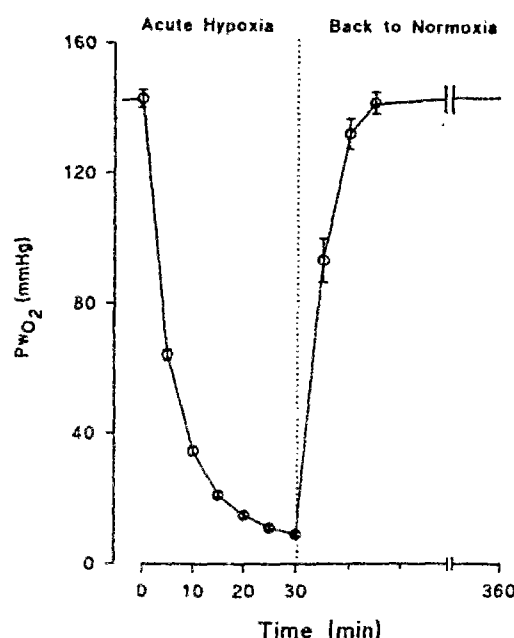


Fig. 1. Average time course of water oxygen tension (P_{wO_2}) within the respirometer during acute hypoxia and rapid return to normoxia.

and S1510 and connected to a Radiometer PHM72 pHmeter. The partial pressure of carbon dioxide in arterial blood (P_{CO_2}) was determined by the Astrup method. The bicarbonate concentration in arterial blood was calculated from the Henderson-Hasselbalch equation, using a CO_2 solubility coefficient and an operational pK' from Boutillier et al. (1985).

Measurements of plasma ions and blood glucose concentrations

Chloride concentration ($[Cl^-]$) was measured with a chloride titrator Radiometer CMT10. Sodium ($[Na^+]$) and potassium ($[K^+]$) concentrations were obtained with a flame photometer (Instrumentation Laboratory 243). Lactate ($[Lact^-]$) and glucose concentrations were respectively measured by enzymatic methods, Boehringer-Mannheim, kit 139084 and Sigma-Diagnostics, Procedure n° 16-UV.

Statistical analysis

The hypoxic and post-hypoxic responses were compared to control measurements by paired t-test.

3. Results

The non-significant decrease in haematocrit throughout the experimental period (first sampling: 24.0 ± 1.8 ; last sampling: 19.4 ± 1.5), indicated a limited impact of the numerous and large blood samplings which represented 3 to 5% of total blood volume. This limited impact was due to the big size of the fish.

Hypoxia

Resting levels of plasma adrenaline (A) and noradrenaline (NA) obtained in *Acipenser baeri* are a little lower than those reported by Randall et al. (1992) in *Acipenser naccarii*. In *Acipenser baeri*, acute exposure to deep hypoxia ($Pw_{O_2} = 10$ torr) promotes a considerable elevation in both A ($\times 77$) and NA ($\times 159$) (Fig. 2). By the end of the 30-min exposure to deep hypoxia, the mean values of circulating adrenaline and noradrenaline respectively reached

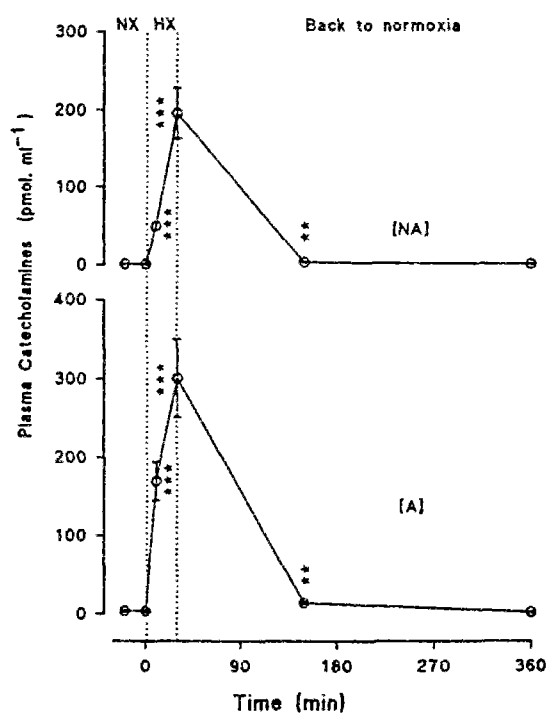


Fig. 2. Effects of acute deep hypoxia (HX) and rapid return to normoxia (NX) on plasma adrenaline (A) and noradrenaline (NA) concentrations. Stars indicate significant differences from normoxic value (** $P < 0.01$, *** $P < 0.005$, paired t-test).

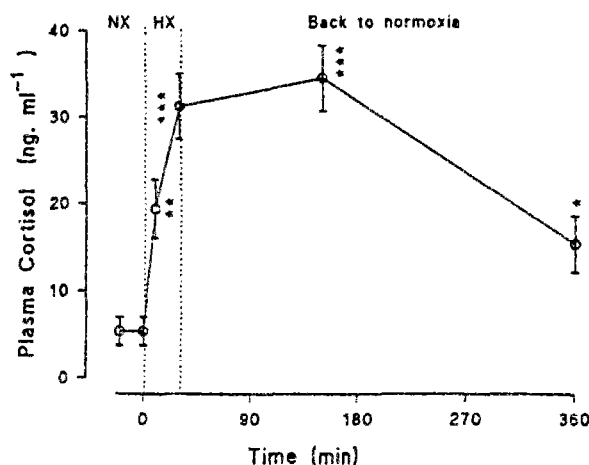


Fig. 3. Effects of acute deep hypoxia (HX) and rapid return to normoxia (NX) on plasma cortisol concentration. Stars indicate significant differences from normoxic value (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.005$, paired t-test).

301 ± 50 and 196 ± 33 pmol·ml⁻¹ (Fig. 2). Both catecholamines, especially adrenaline, rapidly increased at the onset of hypoxia, but the highest plasma levels corresponded to the lowest Pw_{O_2} value. In parallel, the abrupt Pw_{O_2} decrease induced a 6-fold increase in plasma cortisol concentration (Fig. 3), and a 54% rise in blood glucose (Fig. 4).

Deep hypoxia induced a moderate increase in

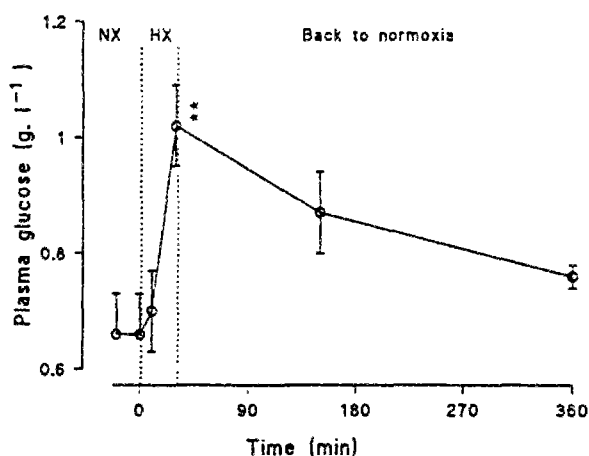


Fig. 4. Effects of acute deep hypoxia (HX) and rapid return to normoxia (NX) on plasma glucose concentration. Stars indicate significant differences from normoxic value (** $P < 0.01$, paired t-test).

heart rate at 10 min, followed by a marked bradycardia (from 52 ± 2 beats·min⁻¹ at 10 min to 17 ± 1 beats·min⁻¹ at 30 min – Fig. 5). In parallel, mean dorsal aortic pressure approximately increased 1.5-fold during the first 10 minutes (Fig. 5). This hypertensive response was followed by an acute fall down to a value corresponding to the two thirds of the control. Mean dorsal aortic pressure changes were associated with an approximate 2-fold increase in difference between the systolic and diastolic pressures.

As shown by Nonnotte et al. (1993), sturgeon is an intermittent breather under normoxic conditions.

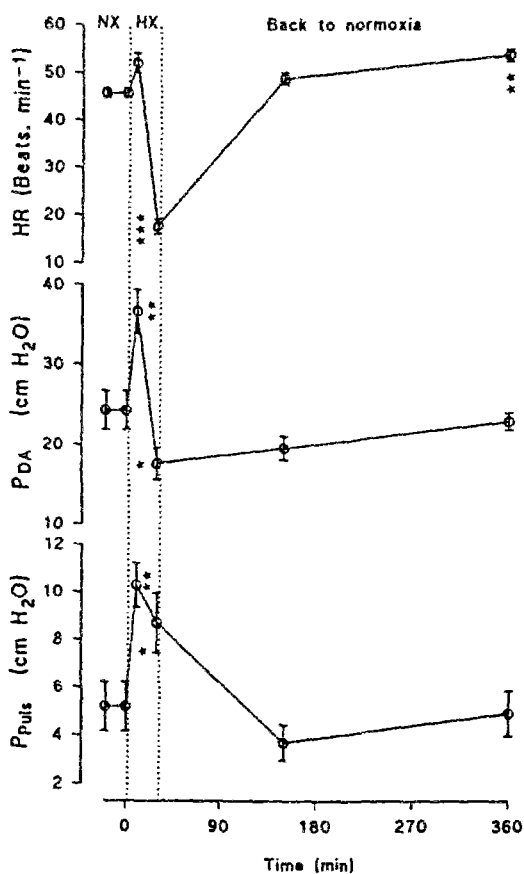


Fig. 5. Effects of acute deep hypoxia (HX) and rapid return to normoxia (NX) on heart rate (HR), mean dorsal aortic blood pressure (P_{DA}) and difference between systolic and diastolic pressures (P_{Puls}) measured in dorsal aorta. Stars indicate significant differences from normoxic value (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.005$, paired t-test).

The time course of changes in both ventilatory amplitude and frequency presented a biphasic aspect during the hypoxic test (Fig. 6). The first 10-min of hypoxia provoked a shift from a spontaneously interrupted to a continuous ventilatory pattern and a marked hyperventilation resulting from an increase in amplitude (+ 260%) and frequency (+ 124%) of pressure changes in the branchial cavity. In a second stage (below $Pw_{O_2} = 30$ torr), sturgeons presented a ventilatory depletion characterized by the reoccurrence of frequent short apneas. Overall ventilatory frequency was therefore lower than the control value by 38%, while amplitude did not differ from normoxic level. Oxygen tension in arterial blood (Pa_{O_2}) decreased dramatically throughout the hypoxic test, reaching 2.2 ± 0.4 torr within 30 min (Fig. 7).

The carbon dioxide wash out caused by the initial hyperventilation resulted in a marked hypocapnia

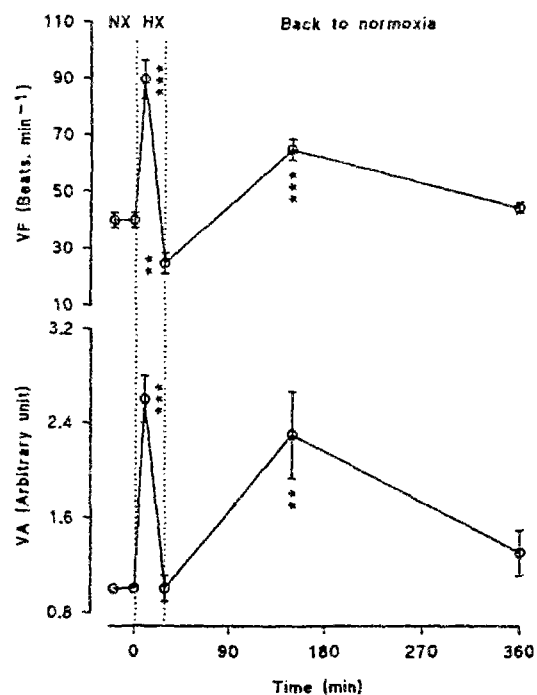


Fig. 6. Effects of acute deep hypoxia (HX) and rapid return to normoxia (NX) on ventilatory frequency (VF) and amplitude (VA). Normoxic ventilatory frequency is calculated as an overall frequency, i.e. in reference to the total time including apneas. Stars indicate significant differences from normoxic value (** $P < 0.01$, *** $P < 0.005$, paired t-test).

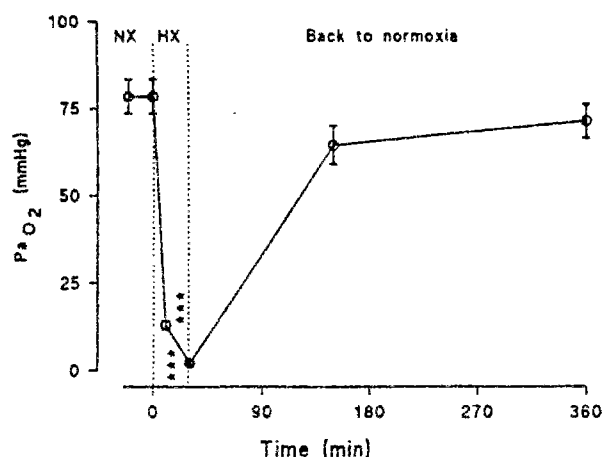


Fig. 7. Effects of acute deep hypoxia (HX) and rapid return to normoxia (NX) on oxygen tension in arterial blood (P_{aO_2}). Stars indicate significant differences from normoxic value (***) $P < 0.005$, paired t-test).

which was coupled to a moderate metabolic acidosis (Fig. 8) due to a small increase in plasma lactate concentration (from 0.57 ± 0.10 in normoxia to 1.44 ± 0.19 mEq·L⁻¹ at $P_{wO_2} = 30$ torr) (Fig. 9). So, arterial pH ($= 7.85$) was not different from control

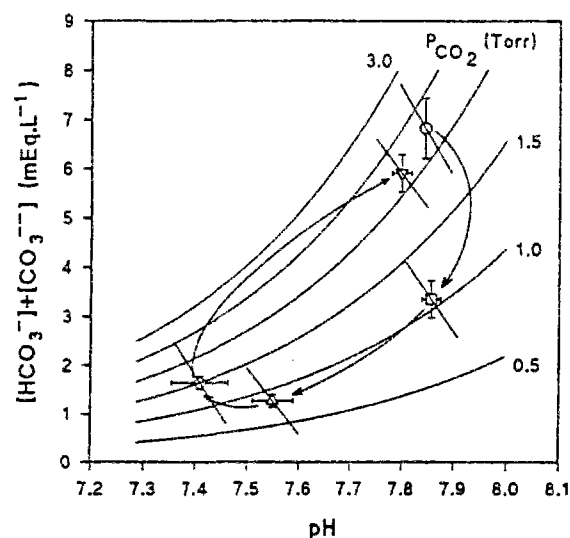


Fig. 8. Davenport diagram illustrating acid-base changes in arterial blood resulting from acute deep hypoxia and rapid return to normoxia. Normoxia (○). Hypoxia: 10 min (□), 30 min (△). Back to normoxia: 2h (◇), 5h30 (▽).

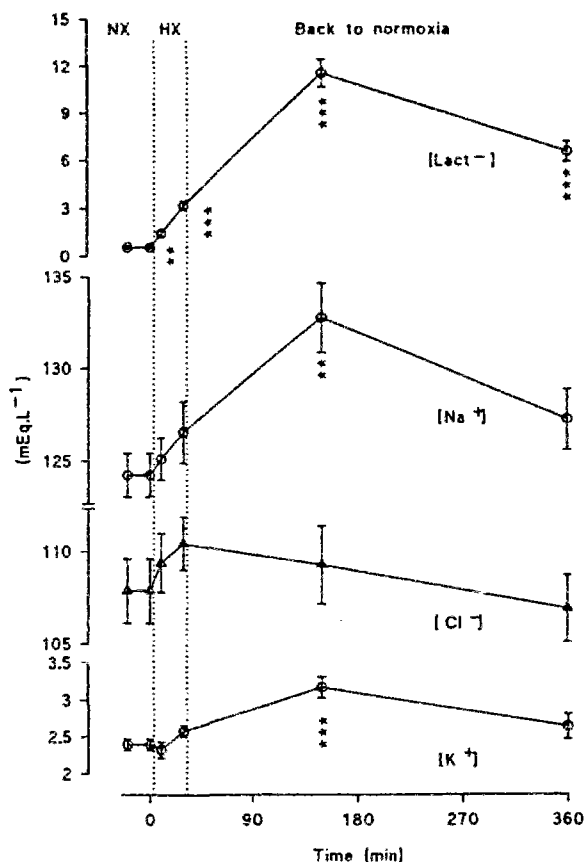


Fig. 9. Effects of acute deep hypoxia (NX) and rapid return to normoxia (NX) on plasma ions concentrations (Sodium [Na⁺], Potassium [K⁺], Chloride [Cl⁻], Lactate [Lact⁻]). Stars indicate significant differences from normoxic value (** $P < 0.01$, *** $P < 0.005$, paired t-test).

value during the first 10 min of hypoxia. Then, the intensification of anaerobic metabolism induced an important decrease in pH_a (from 7.85 ± 0.01 in normoxia to 7.55 ± 0.04 at $P_{wO_2} = 10$ torr). At that time, the carbon dioxide tension was paradoxically maintained at a low level despite a ventilatory depression (Fig. 8). Plasma Na⁺, K⁺ and Cl⁻ concentrations remained unchanged throughout hypoxic exposure (Fig. 9).

Back to normoxia

At the end of stress, the plasma levels of catecholamines rapidly declined to control value (Fig. 2). By contrast, plasma cortisol level remained

at its highest level during the first 2 h of return to normoxia, before decreasing (Fig. 3). 5h30 after the end of hypoxia, its concentration was still 3 times higher than the control value. Plasma glucose decreased progressively throughout the post-hypoxic period (Fig. 4).

The marked bradycardia observed at the lowest level of Pw_{O_2} disappeared quickly (Fig. 5). Moreover, 5h30 after the end of hypoxia, heart rate was significantly higher than control value. A rapid return to normoxic conditions induced a progressive increase in mean dorsal aortic pressure from its lowest hypoxic level; so that no significant difference could be noted between control and post hypoxic value by 5h30 (Fig. 5). In the same way, the difference between systolic and diastolic pressures decreased swiftly to the normoxic value (Fig. 5).

During rapid return to normoxia, the hypoxia-induced apneas vanished quickly. The frequency and amplitude of pressure changes in the branchial cavity increased again. Two hours after the end of hypoxia, this post-hypoxic hyperventilatory effect, affecting especially amplitude, began to decrease. Finally, post-hypoxic VA and VF values were not significantly different from the normoxic reference values (Fig. 6). Moreover, a return to normoxia caused an immediate increase in Pa_{O_2} (Fig. 7).

The metabolic acidosis was emphasized during the first 2 hours of the posthypoxic period. However, this increase was moderate ($\Delta pH_a = 0.14$) in spite of an important release of lactate into the blood stream up to $12.98 \pm 0.81 \text{ mEq}\cdot\text{l}^{-1}$ (Fig. 9). Then, carbon dioxide tension and pH_a progressively recovered their reference values (Fig. 8). Plasma Cl^- level remained unchanged throughout the post-hypoxic period. In parallel, Na^+ and K^+ concentrations rose markedly to their maximum levels, respectively 7% and 32% higher than normoxic levels, before decreasing (Fig. 9).

4. Discussion

Catecholamines and cortisol release

An almost invariable neuro-endocrine response of fish to environmental stress is the release of catecholamines and corticosteroids into the blood stream (for review, see Mazeaud and Mazeaud,

1981; Donaldson, 1981). Interrenal tissue has been described in sturgeon (Matty, 1985). But, there is a lack of information about the localization of chromaffin tissue in this fish.

Levels of catecholamines as high as those reported in the present paper were observed by Metcalfe and Butler (1989) in *Scyliorhinus canicula* submitted to moderate hypoxia (55 torr). In the same way, Thomas et al. (1991) showed in rainbow trout an increase in plasma adrenaline concentration to about $300 \text{ pmol}\cdot\text{ml}^{-1}$ at the midpoint of a 48 h chronic exposure to moderate hypoxia (50–70 torr). Because of the rapid clearance of catecholamines from the blood by the combined effects of tissue accumulation and metabolism, peak levels decrease within a few minutes after the end of stress (Fig. 2).

Resting levels of cortisol (Fig. 3) are of the same order of magnitude as those reported for goldfish (Venkatesh et al., 1989) and rainbow trout (Pickering et al., 1991). There is not many information on the effects of hypoxic stress on plasma cortisol in fish. Only effects of hypoxia initiated by deep anaesthesia can be mentioned. Mazeaud et al., (1977) reported in coho salmon a moderate elevation of corticosteroids in males, but not in females. Surprisingly, Iwama et al. (1989), observed in rainbow trout, a progressive decline of cortisol concentrations. Corticosteroid stress response in sturgeon is stronger and more persistent compared to these data.

An analogy with known effects of catecholamines and corticosteroids in teleosts submitted to a similar stress could be drawn to envisage the role of adrenaline, noradrenaline and cortisol in sturgeon submitted to extreme hypoxia. The main sites of action of catecholamines are the cardiovascular and respiratory systems (see further discussion). The catecholamines also act directly on the liver to stimulate glycogenolysis (β effect). Cortisol, like catecholamines, is responsible for the mobilization of energy reserves by activating liver glycogenolysis and inhibiting glycolysis (reviewed by Donaldson, 1981). These effects result in a moderate increase in plasma glucose concentration (Fig. 4). So, during the post-hypoxic period, cortisol appears to reinforce and prolong the immediate catecholamine response to hypoxic stress. However, the relative contributions of corticosteroid and adrenergic responses to the

increased plasma glucose concentrations usually observed in fish are still unclear (Mazeaud and Mazeaud, 1981). Blood glucose levels vary considerably among species, within a species and over time, even within an individual (McDonald and Milligan, 1992). The relatively low resting level measured in sturgeon and the moderate maximum increase observed at the lowest level of $P_{W_{O_2}}$ might be partly due to starvation during the experiments.

Circulatory responses

Early circulatory changes observed during the first 10 min of the abrupt decrease in $P_{W_{O_2}}$ (Fig. 5) could be due to an increased adrenergic nervous stimulation of the heart and vasculature together with the adrenergic influence of catecholamines released from the chromaffin tissue (Fritsche and Nilsson, 1993). The initial moderate rise in HR associated with a relatively more acute increase in P_{Puls} represent positive chronotropic and inotropic β effects on the heart (Wood and Shelton, 1980). In addition, catecholamines cause an α -adrenoceptor-mediated vasoconstriction of the systemic vasculature (Wood and Shelton, 1975) which increases systemic resistance and results in a moderate elevation in P_{DA} . However, the disproportion between the small magnitude of these circulatory effects and the high corresponding levels of circulatory catecholamines must be underlined. This could indicate a low sensitivity of sturgeon to catecholamines resulting either from a density of adrenoceptors lower than in teleosts, or from fundamental differences with teleosts in the way that target tissue is stimulated consequently to receptors activation (Perry and Reid, 1992).

At the deepest level of $P_{W_{O_2}}$, the effects of catecholamines are masked by specific effects of hypoxia. The important bradycardia, occurring in spite of the highest plasma catecholamines concentration (Fig. 5), could be regarded as a classical vagal reflex response in fish to severe hypoxia. But, further investigations using atropine to block any vagal tone on the heart will be necessary to test this hypothesis. This bradycardia results in a pronounced decrease in P_{DA} . The $P_{a_{O_2}}$ threshold at which the gills O_2 -sensitive chemoreceptors were stimulated is very low. Indeed, *Acipenser baeri* submitted to moderate progressive hypoxia ($P_{W_{O_2}} = 20$ torr) does not present any change in HR (unpublished data).

Bradycardia, as suggested by Hughes (1973), may be regarded as an adaptative response to hypoxia, because the increased residence time of blood in the gills can help the fish to maintain the effectiveness of O_2 transfer. The absence of a compensatory increased P_{Puls} during bradycardia, together with the decrease in P_{DA} below its control normoxic value (Fig. 5), may be considered as caused by a lowered contractile strength of the myocardium resulting from a deficient O_2 supply ($P_{a_{O_2}} = 2$ torr – Fig. 7).

Respiratory effects

The hyperventilatory response to hypoxia in fish is largely driven by stimulation of gills chemoreceptors due to oxygen lack. However, the location and afferent pathways for these receptors are not well known. Burleson and Smatresk (1990) suggested that gill ventilation in channel catfish is interactively controlled by separate internally and externally oriented receptors, the latter eliciting also bradycardia. Moreover, some additional action of catecholamines in extreme hypoxia could be envisaged. Indeed, Peyraud-Waitzenegger et al. (1979) demonstrated that circulatory catecholamines pass the blood-brain barrier. So, as suggested by Peyraud-Waitzenegger et al. (1980), catecholamines may be capable of directly stimulating the β -adrenoceptors of the brain centers, thus increasing the spontaneous rate of firing of the ventilatory neurons.

It may be assumed that the ventilatory depression, observed after 10 min of hypoxic stress, results from a decrease in chemosensitivity and a depression of respiratory centers activity due to extremely low values of $P_{a_{O_2}}$. To support a part of this hypothesis, it was reported that chemoreceptor output declined in extreme hypoxia (Randall and Perry, 1992). Then, the post-hypoxic period is characterized by the resumption of high ventilatory levels which allows the repayment of an oxygen debt. As suggested by Nonnotte et al. (1993), this hyperventilatory response may partly result from the blood lactic acidosis (Fig. 8). Indeed, during the post-hypoxic period, $P_{a_{O_2}}$ swiftly reaches high normoxic levels (Fig. 7) which cannot explain such a high ventilation rate.

At the deepest hypoxia level (10 torr), the lack of increase in PCO_2 in spite of hypoventilation can be interpreted as a consequence of a reduction in carbon dioxide production resulting from a deficiency of

O₂ supply to tissue. The return to normoxia is characterized by a more massive flush of lactate. However, the pH fall is not so important as expected from this effect; this exhibits the development of some adjustment to maintain the pH level as normal as possible. Indeed, the elevation of blood lactate is accompanied by a marked increase in plasma sodium concentration (Fig. 9) suggesting a metabolic compensation of developed acidosis. Another hypothesis might be a possible excretion in external medium of protons issued from lactic acid and balanced by an influx of sodium at the gills level. These exchanges could also explain the slight acidosis and the increase in plasma sodium concentration.

The increase in catecholamines and cortisol plasma concentrations, the circulatory and ventilatory responses and the changes in acid-base status of arterial blood induced in sturgeon by acute deep hypoxia and after the return to normoxia provided evidences to explain both the strong resistance of this fish to oxygen depletion and the ecological success of sturgeon in hypoxic habitats. The hypoxia-induced adjustments, reported here, were similar to those observed in teleosts submitted to identical conditions. Thus, it might be assumed that sturgeon was able to survive during the evolution because it early acquired the same adaptative processes as the most advanced fish.

Acknowledgement

The plasma cortisol and glucose concentrations were respectively carried out by Mrs J. Nicolas and Mr C. Veillard whom we are thankful to. The authors also want to express their thanks to Drs J. Caroff and C. Cann-Moisan for their kind help in the course of plasma catecholamines assay.

References

- Boutilier R.G., G.K. Iwama, T.A. Heming and D.J. Randall (1985). The apparent pK of carbon acid in rainbow trout blood plasma between 5 and 15 °C. *Respir. Physiol.* 61: 237–254.
- Boutilier R.G., G. Dobson, U. Hoeger and D.J. Randall (1988). Acute exposure to graded levels of hypoxia in rainbow trout (*Salmo gairdneri*): metabolic and respiratory adaptations. *Respir. Physiol.* 71: 69–82.
- Burleson M.L. and N.J. Smatresk (1990). Evidence for two oxygen-sensitive chemoreceptor loci in channel catfish, *Ictalurus punctatus*. *Physiol. Zool.* 63: 208–221.
- Donaldson E.M. (1981). The pituitary-interrenal axis as an indicator of stress in fish. In: *Stress in fish*, edited by A.D. Pickering New York, Academic press: 11–47.
- Fritsche R. and S. Nilsson (1993). Cardiovascular and ventilatory control during hypoxia. In *Fish Ecophysiology*, edited by J.C. Rankin and F.B. Jensen, London, Chapman & Hall: 180–206.
- Hughes G.M. (1973). Respiratory response to hypoxia in fish. *Am. Zool.* 13: 475–489.
- Iwama G.K., J.C. McGeer and M.P. Pawluk (1989). The effects of five fish anaesthetics on acid-base balance, haematocrit, blood gases, cortisol and adrenaline in rainbow trout. *Can. J. Zool.* 67: 2065–2073.
- Maruta K., K. Fugita, S. Ito and J. Nagatsu (1984). Liquid chromatography of plasma catecholamines, with electrochemical detection, after treatment with boric acid gel. *Clin. Chem.* 30: 529–548.
- Matty A.J. (1985). The 'adrenal' and the kidney hormones. In: *Fish Endocrinology*, London, Sidney, Croom Helm publ.: 112–137.
- Mazeaud M.M., F. Mazeaud and E.M. Donaldson (1977). Primary and secondary effects of stress in fish: some new data with general review. *Trans. Am. Fish. Soc.* 106: 201–212.
- Mazeaud M.M. and F. Mazeaud (1981). Adrenergic responses in fish. In: *Stress in fish*, edited by A.D. Pickering New York, Academic press: 49–75.
- McDonald D.G. and C.L. Milligan (1992). Chemical properties of the blood. In: *Fish Physiol.* Vol XII, part B. Edited by W.S. Hoar, D.J. Randall, and A.P. Farrell, Academic Press: San Diego, 55–133.
- Metcalf J.D. and P.J. Butler (1989). The use of alpha-methyl-p-tyrosine to control circulating catecholamines in the dogfish *Scyliorhinus canicula*: the effects on gas exchange in normoxia and hypoxia. *J. Exp. Biol.* 141: 21–32.
- Nonnotte G., V. Maxime, J.P. Truchot, P. Williot and C. Peyraud (1993). Respiratory responses to progressive ambient hypoxia in the sturgeon, *Acipenser baeri*. *Respir. Physiol.* 91: 71–82.
- Perry S.F. and S.D. Reid (1992). The relationship between beta-adrenoceptors and adrenergic responsiveness in trout (*Oncorhynchus mykiss*) and eel (*Anguilla rostrata*) erythrocytes. *J. Exp. Biol.* 167: 235–250.
- Peyraud-Waitzenegger M., A. Savina, J. Laparra and R. Morfin (1979). Blood-brain barrier for epinephrine in the eel (*Anguilla anguilla* L.). *Comp. Biochem. Physiol.* 23(1): 35–38.
- Peyraud-Waitzenegger M., L. Barthelemy and C. Peyraud (1980). Cardiovascular and ventilatory effects of catecholamines in unrestrained eels (*Anguilla anguilla* L.). *J. Comp. Physiol.* 138: 367–375.
- Pickering A.D., T.G. Pottinger, J.P. Sumpter, J.F. Carragher and P.Y. Le Bail (1991). Effects of acute and chronic stress on the levels of circulating growth hormone in the rainbow trout, *Oncorhynchus mykiss*. *Gen. Comp. Endocrinol.* 83: 86–93.
- Randall D.J., D.J. McKenzie, G. Abrami, G.P. Bondiolotti, F. Natiello, L. Bolis and E. Agradi (1992). Effect of diet on re-

- sponses to hypoxia in sturgeon (*Acipenser naccarii*). *J. Exp. Biol.* 170: 113–125.
- Randall D.J. and S.F. Perry (1992). Catecholamines. In: *Fish Physiology*, Vol. XII, part B, edited by W.S. Hoar, D.J. Randall, and A.P. Farrell, San Diego, Academic Press: 255–300.
- Tetens V. and G. Lykkeboe (1985). Acute exposure of rainbow trout to mild and deep hypoxia: O₂ affinity and O₂ capacitance of arterial blood. *Respir. Physiol.* 61: 221–235.
- Thomas S., R. Kinkead, P.J. Walsh, C.M. Wood and S.F. Perry (1991). Desensitization of adrenaline-induced red blood cell H⁺ extrusion in vitro after chronic exposure of rainbow trout to moderate environmental hypoxia. *J. Exp. Biol.* 156: 233–248.
- Venkatesh B., C.H. Tan and T.J. Lam (1989). Blood steroids in the goldfish: measurement of six ovarian steroids in small volumes of serum by reverse-phase high performance liquid chromatography and radioimmunoassay. *Gen. Comp. Endocrinol.* 76: 397–407.
- Wood C.M. and G. Shelton (1975). Physical and adrenergic factors affecting systemic vascular resistance in the rainbow trout: a comparison with branchial vascular resistance. *J. Exp. Biol.* 63: 505–523.
- Wood C.M. and G. Shelton (1980). Cardiovascular dynamics and adrenergic responses of the rainbow trout in vivo. *J. Exp. Biol.* 87: 247–270.