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ADRENERGIC CONTROL OF OXYGEN TRANSFER IN PERFUSED GILLS OF THE COD, *GADUS MORHUA*

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SUMMARY

A perfused and ventilated gill preparation is described in which the pO_2 of the perfusion medium and the irrigating water is controlled. Dorsal aorta effluent pO_2 and flow were measured together with the branchial vascular resistance.

Decreasing pO_2 in the perfusion fluid caused increased branchial vascular resistance, probably by constriction of efferent lamellar arterioles. α-adrenoceptor stimulation caused constriction of arteriovenous connections and of efferent lamellar arterioles, and enhanced oxygenation of the perfusion fluid. β-adrenoceptor stimulation also increased O_2 transfer, but to a lesser extent.

It is suggested that both hypoxia and α-adrenoceptor stimulation improved O_2 transfer via constriction of efferent lamellar arterioles. Both stimuli may also increase systemic blood flow by constriction of the arteriovenous connections, although such an effect of hypoxia has not been clearly shown. β-stimulation probably increased O_2 transfer by dilation of afferent lamellar arterioles, thereby causing recruitment of unperfused lamellae.

INTRODUCTION

Since Krawkow (1913) reported that vasodilation is the main response to adrenaline of the branchial vascular bed a number of studies on the control of the branchial vasculature have been performed (see Wood, 1975; Smith, 1977). The response to catecholamines is now known to be biphasic, consisting of a β-adrenergic dilation and an α-adrenergic constriction in perfused gill preparations (Reite, 1969; Belaud, Peyraud-Waitzenegger & Peyraud, 1971; Bergman, Olson & Fromm, 1974; Wood, 1975; Dunel & Laurent, 1977; Payan & Girard, 1977; Claiborne & Evans, 1980; Wahlqvist, 1980, 1981; Nilsson & Pettersson, 1981). A decrease in branchial vascular resistance following catecholamine injections *in vivo* has been reported [e.g. in the cod, *Gadus morhua*, (Pettersson & Nilsson, 1980), and the lingcod, *Ophiodon elongatus* (Farrell, 1981)] while Wood & Shelton (1980) have demonstrated that adrenaline causes either dilation or constriction of the branchial vasculature in the rainbow trout, *Salmo gairdneri*.

It has also been shown that adrenaline increases arterial pO_2 in the eel, *Anguilla anguilla*, *in vivo* (Steen & Kruysse, 1964; Peyraud-Waitzenegger, 1979) and oxygen transfer in perfused gill preparations (Pettersson & Johansen, 1982; Pärt, Tuurala &

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Soivio, 1982) and in totally perfused rainbow trout (Wood, McMahon & MacDonald, 1978). The aim of this study was to investigate the nature of the adrenergic receptors which control oxygen transfer.

MATERIALS AND METHODS

Atlantic cod, *Gadus morhua*, of both sexes, weighing between 600 and 1000 g, were used in this study. After capture they were kept in re-circulated, aerated sea water at 10 °C.

Fish were stunned by a blow on the head, and approximately 1500 i.u. of heparin was injected into the caudal blood vessels. After 5 min the head was removed from the body, and a slow perfusion of the gills was made through a catheter (PE90) inserted into the bulbus arteriosus. The effluent from the perfused head was collected from another catheter (PE90) in the dorsal aorta. The coeliaco-mesenteric artery was ligated and the head was immersed in sea water. The gills were irrigated with sea water at approx. 1200 ml min^{-1} with an Eheim pump through a Y-tube in the mouth.

To record ventral aortic pressure (PVA) the ventral aortic catheter was connected to a Statham P23 pressure transducer via a T-piece. The dorsal aortic effluent was passed through a Radiometer E 5046 O_2 electrode and a Grass photoelectric drop counter. The O_2 electrode was connected to a radiometer PHM 71 or 73 and a Goerz Servogor potentiometric recorder displaying pO_2 of the dorsal aortic effluent, $p_{da}O_2$. The drop counter was connected to a Grass mod. 7 polygraph, and a tachograph (Grass mod. 7P4 or 7P44) converted the signal to drops min^{-1}. The pressure transducer was also connected to the Grass polygraph.

The gills were perfused with approx. 8–10 ml min^{-1} kg bw^{-1} by a Gilson peristaltic pump with a filtered Ringer's solution (House & Greene, 1964) containing glucose at a concentration of 1 g l^{-1}. Its pO_2 was adjusted to the desired level with a Wösthoff mod. 301 gas mixing pump. No CO_2 was dissolved in the Ringer's solution, and its pH therefore increased from approx. 7·3 at the start to approx. 8·0 at termination of the experiments. This increase had no effect on the measured variables.

Johansen & Pettersson (1981) showed that O_2 consumption of the gill tissue is high. The O_2 utilized is partly taken directly from the irrigating water to the O_2 consuming cells, but also from the blood stream. Control experiments to ensure that it was oxygenation of the perfusion fluid that was studied, and not the O_2 utilization by the branchial tissue, were therefore performed.

In *series A* pO_2 of both the irrigating water (p_iO_2) and the perfusion medium reaching the gills ($p_\bar{v}O_2$) was equal (approx. 157 mmHg; air saturation). Oxygen extraction from the perfusion fluid could be studied in this series, and whether the drugs themselves affected the extraction of O_2 from the perfusion fluid in this 'non-respiratory' preparation was also investigated.

In *series B* p_iO_2 was still kept at air saturation, but $p_\bar{v}O_2$ was decreased to 23 ± 2 mmHg (S.E.M.), and a 'natural' O_2 diffusion gradient was thus created. If the preparation is functional O_2 will diffuse to the perfusion fluid and elevate $p_{da}O_2$. Effects of drugs on $p_{da}O_2$, other than those seen in series A can thus be ascribed to effects on O_2 transfer.

Further control experiments were performed in *series C*. $p_\bar{v}O_2$ was elevated

approx. 290 ± 8 mmHg (S.E.M.), with p_iO_2 still kept at air saturation. If the effect on O_2 transfer of the drugs seen in series B was reversed it seems reasonable to believe that O_2 transfer, and not O_2 utilization, was measured.

All experiments were performed at 10 °C. The drugs used were dissolved in the Ringer's solution prior to use. Wahlqvist & Nilsson (1981) reported that the catecholamine concentration in cod plasma can exceed 3×10^{-7} M in stress, and a concentration of 10^{-6} M of the agonists (adrenaline and isoprenaline) was therefore used. Complete blockade of the α-adrenoceptor responses of the agonists in the concentration used is obtained with phentolamine 10^{-5} M, and blockade of β-adrenoceptors can be made with 3×10^{-6} M propranolol (Nilsson & Pettersson, 1981). These concentrations of antagonists were therefore used in the present experiments.

Statistical treatment according to the Wilcoxon matched-pair signed-ranks test was performed when n exceeded 6.

RESULTS

The dorsal aorta catheter and the thermostatted cuvette of the O_2 electrode added some resistance to flow through the perfusion system. By lowering the tip of the outflow catheter slightly below the water surface compensation was obtained for this resistance. After this adjustment dorsal aorta flow (FDA) averaged 26·5 % of the total perfusion in series A. Although slightly lower in series B, flow was not significantly different from series A (Table 1). The venous drainage of the gills and the cephalic circulation were not ligated, and this flow together with the leakage from the cut ends of the systemic vessels explain the low flow through the dorsal aorta.

The perfusion rate was 8–10 ml min^{-1} kg bw^{-1} and resulted in a perfusion pressure of 3·9 ± 0·54 kPa in series A and 4·7 ± 0·23 kPa in series B, which is significantly higher ($P < 0{\cdot}01$, $n = 9$).

In series B PVA and FDA increased in response to adrenaline. The increase in FDA was significantly smaller than in series A (Table 1, Fig. 1). $p_{da}O_2$ increased from 61 ± 11 mmHg (S.E.M.) to 80 ± 12 mmHg (S.E.M.) (Table 1, Fig. 1).

The effect of adrenaline administration after β-blockade with propranolol showed that the increase in PVA and FDA were due to α-adrenoceptor stimulation (Table 1, Fig. 1). $p_{da}O_2$ increased as much as prior to β-blockade (Table 1, Fig. 1). β-stimulation (isoprenaline or adrenaline following α-blockade with phentolamine) decreased PVA and left FDA unchanged (Table 1, Fig. 1). $p_{da}O_2$ increased by approx. 6 mmHg, a response that was only about 30 % of that elicited by adrenaline itself or by adrenaline in the presence of propranolol (Fig. 1).

In series C $p_{da}O_2$ was 184 ± 4 mmHg. A decrease was found following both α- and β-stimulation, showing that, in this case, catecholamines induced an increase in O_2 loss across the gills. PVA and FDA increased due to α-stimulation. PVA did not decrease when p_vO_2 was increased above air saturation. The changes in FDA and PVA were not more marked in this series than in series A, suggesting that above a certain level branchial smooth muscle does not respond to changes in pO_2. The results in series C have not been statistically evaluated due to the small number of experiments ($n = 4$).

Table 1. *The results of adrenaline (10^{-6} M) and selective* α- [*adrenaline (10^{-6} M) following propranolol (3×10^{-6} M)*] *and* β- [*isoprenaline (10^{-6} M) or adrenaline (10^{-6} M) following phentolamine (10^{-5} M)*] *-adrenoceptor stimulation on the measured variables*

In series A $p_eO_2 = p_iO_2$ = air saturation, in series B $p_eO_2 = 23 \pm 2$ mmHg and p_iO_2 = air saturation. The value recorded immediately before agonist administration served as control. x: $P < 0.05$; xx: $P < 0.02$; xxx: $P < 0.01$. n = number of experiments.

Series	Treatment	PVA (kPa) Control	PVA (kPa) Stimulated	FDA (dpm) Control	FDA (dpm) Stimulated	$P_{da}O_2$ (mmHg) Control	$P_{da}O_2$ (mmHg) Stimulated	n
A	Adr	3·93 ± 0·54[1]	4·44 ± 0·59[xx]	43·6 ± 2·2	48·3 ± 3·2[xxx, 2]	128·3 ± 3·2	127·1 ± 3·0	8
	Adr after Prop	4·05 ± 0·27	5·25 ± 0·15	44·5 ± 0·5	51·0 ± 4·0	127·5 ± 12·5	130·5 ± 9·5	4
	Iso	3·08 ± 0·35	2·70 ± 0·28	43·0 ± 2·9	42·8 ± 2·8	129·0 ± 3·7	127·4 ± 2·9	4
	Adr after Phent	5·10 ± 1·35	4·28 ± 1·28	45·0 ± 0·5	45·0 ± 0·5	142·0 ± 1·9	142·5 ± 5·5	4
B	Adr	4·68 ± 0·23[1]	5·07 ± 0·26	39·9 ± 3·5	41·6 ± 3·9[x, 2]	60·9 ± 10·7	79·7 ± 11·9[xx]	9
	Adr after Prop	4·63 ± 0·27	5·96 ± 0·34	37·8 ± 4·3	40·3 ± 6·2	70·5 ± 13·9	93·5 ± 10·0	4
	Iso	4·33 ± 0·42	3·28 ± 0·25	37·5 ± 3·9	37·0 ± 3·9	66·3 ± 11·5	75·3 ± 15	4
	Adr after Phent	5·45 ± 1·33	4·50 ± 1·20	36·5 ± 7·2	35·9 ± 7·5	36·8 ± 11·0	42·5 ± 15·1	4

[1] In series B PVA was significantly higher than in series A ($P < 0.05$)
[2] The increase in FDA in series A was higher than in series B ($P < 0.05$)
Abbreviations: Adr = adrenaline; FDA = dorsal aorta flow; Iso = isoprenaline; $p_{da}O_2$ = dorsal aorta effluent pO_2; Phent = phentolamine; Prop = propranolol; PVA = ventral aorta pressure.

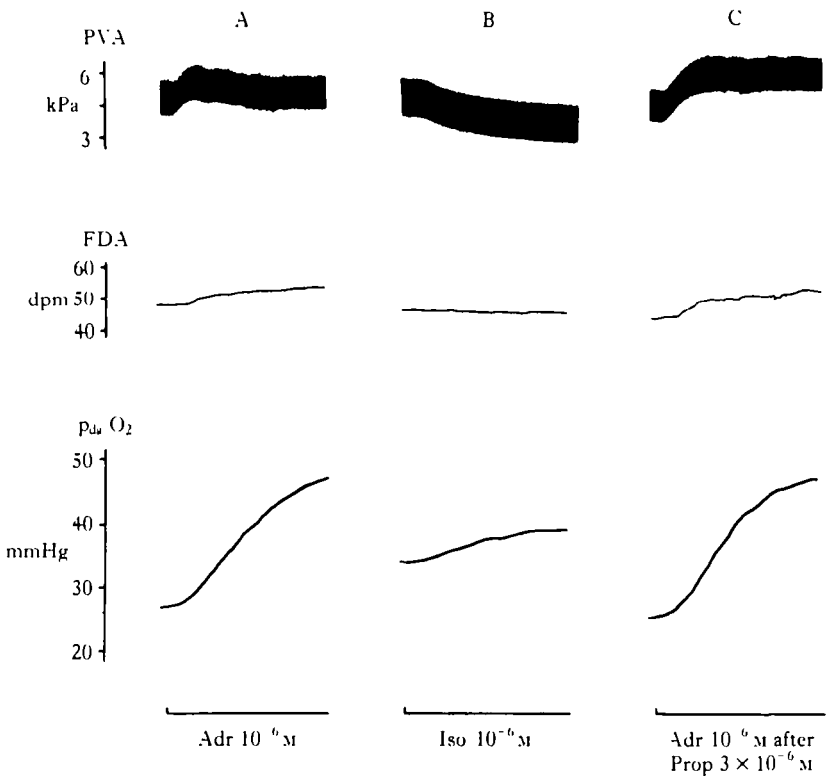

Fig. 1. Original tracings that show responses to adrenaline (A), isoprenaline (B) and adrenaline after application of propranolol (C). All traces are from one fish. Upper traces: ventral aorta pressure (PVA); middle traces: dorsal aorta flow (FDA); lower traces: pO_2 of dorsal aorta effluent ($p_{da}O_2$). Adr = adrenaline; Iso = isoprenaline; Prop = propranolol.

DISCUSSION

From the results of the series A experiments it is evident that extraction of O_2 from the perfusion medium by the branchial tissue occurs as the fluid passes along the vascular channels, confirming the results of Johansen & Pettersson (1981) and Pettersson & Johansen (1982). This metabolic O_2 utilization produced a decrease in pO_2 from 157 to 128 mmHg in the perfusion fluid in series A. Nevertheless, in series B $p_{da}O_2$ was about 60 mmHg when $p_{\bar{v}}O_2$ was only 23 mmHg, and as the Ringer solution passed the gills, pO_2 decreased from 290 to 184 mmHg in series C. These results show that an exchange of O_2 with the irrigating water was evident in both series B and C. The arterio-venous pO_2 differences ($p_{da}O_2 - p_{\bar{v}}O_2$) in series B and C were 67 and -77 mmHg, respectively, after compensation for the O_2 extraction by the branchial tissue found in series A ($157-128 = 29$ mmHg). The minus sign indicates a reversed direction of O_2 transport in series C. The preparation is thus an efficient gas exchanger.

Knowledge of microcirculation in fish gills has developed rapidly with the use of modern vascular casting techniques. O_2 and CO_2 are exchanged with the environment in the secondary lamellae which receive venous blood via the afferent branchial (ABA), afferent filamental (AFA) arteries and the afferent lamellar (ALA) arterioles.

The drainage consists of efferent lamellar (ELA) arterioles, efferent filamental (EFA) and efferent branchial (EBA) arteries. Numerous vessels also arise from the EFA and EBA, and they are drained via the venous compartment of the gills (Gannon, Campbell & Randall, 1973; Vogel, Vogel & Schlote, 1974; Laurent & Dunel, 1976; Vogel, Vogel & Pfautsch, 1976; Cooke & Campbell, 1979; Vogel, 1979). The entire cardiac output does not therefore reach the systemic circulation. Connections between the afferent arterial system and the venous compartment have been described in the eel (Steen & Kruysse, 1964; Laurent & Dunel, 1976) and in the Channel catfish, *Ictalurus punctatus*, (Boland & Olson, 1979). A preliminary investigation in the cod showed no such connections (Nilsson & Pettersson, 1981). Fig. 2 shows the principal pathways for blood flow through the cod gills. All connections to the venous compartment have been grouped together as 'arterio-venous connections' (AVCs), since they could not be separated in this study.

Possible sites of action of the various stimuli used are also shown in Fig. 2. 'Hypoxic' vasoconstriction occurs at, or distal to, the secondary lamellae, but proximal to the AVCs (Pettersson & Johansen, 1982). The pillar cells are reported to contain contractile elements (Bettex-Galland & Hughes, 1973; Smith & Chamley-Campbell, 1981). Constriction in these cells would, however, restrict flow to the marginal channels of the secondary lamellae, which is unlikely to be advantageous in gas exchange. If, however, the ELAs constrict, pressure will increase in the secondary lamellae and in the afferent arterial system. The increased PVA may cause the recruitment of unperfused lamellae found after hypoxia in the rainbow trout (Booth, 1979), and may also affect gas transfer by altering the flow pattern within the secondary lamellae (Rankin & Maetz, 1971).

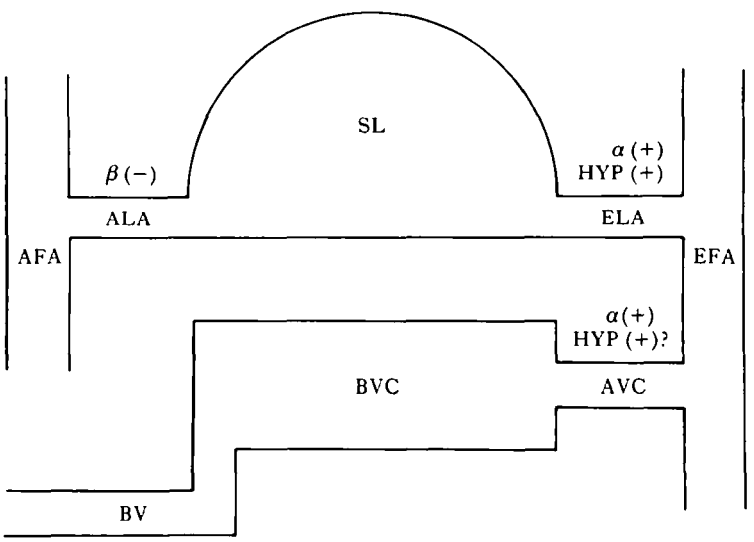

Fig. 2. The principal pathways for blood flow in cod gills. The proposed effector sites of the stimuli used in the study are indicated. Dilatory β-receptors are indicated as β (−); constrictory α-receptors as α (+). HYP (+) indicates smooth muscle that contracts at low pO_2. AFA = afferent filamental artery; ALA = afferent lamellar arteriole; AVC = arterio-venous connections; BV = branchial vein; BVC = branchial venous compartment; EFA = efferent filamental artery; ELA = efferent lamellar arteriole; SL = secondary lamella.

The increase in FDA following adrenaline administration is concluded to be due to α-stimulation of the AVCs (Fig. 2), supporting earlier findings (Dunel & Laurent, 1977; Payan & Girard, 1977; Claiborne & Evans, 1980; Nilsson & Pettersson, 1981). In a few preparations, where adrenaline increased PVA, no change in FDA was seen. This implies that an action also occurs proximal to the AVCs.

Lamellar recruitment occurs after an injection of adrenaline in the rainbow trout (Booth, 1979), a finding that has been confirmed by Holbert, Boland & Olson (1979) in the Channel catfish. A constriction of afferent arterial vessels is not likely to be consistent with lamellar recruitment. Nor is it likely to explain the pronounced increase in oxygen transfer (Table 1, Fig. 1). Constriction of the ELAs is, on the other hand, consistent with both these findings, and constriction of the ELAs is therefore suggested as response to α-adrenoceptor stimulation. Constriction of the ELAs also followed the hypoxic stimulus (see above), which suggests that oxygen transfer is improved by this myogenic mechanism although it cannot be demonstrated in experiments of this type.

The increase in FDA caused by adrenaline, due to α-stimulation of the AVCs, is less pronounced in series B than in series A (Table 1). This suggests that the AVCs also respond to low pO_2. Such an effect would increase the systemic blood flow in adverse circumstances (e.g. environmental hypoxia) and would thus be advantageous to the fish. The reduction in flow through the branchial venous compartment in the cod gills during hypoxia as reported by Pettersson & Johansen (1982) was, however, not significant. Ristori & Laurent (1977) also failed to detect any constriction in the arterio-venous pathway during hypoxia in perfused rainbow trout gills. This matter obviously requires further investigation.

β-adrenergic stimulation must have decreased PVA due to an action proximal to the AVCs, because FDA remained almost constant (Table 1, Fig. 1). A relaxation of the pillar cells would decrease PVA and increase the functional surface area of the secondary lamellae, and thus explain the increased O_2 transfer seen (Table 1). A relaxation of the ALAs would also explain these findings and appears to be a more likely interpretation since it could also explain the lamellar recruitment by adrenaline (Booth, 1979; Holbert, Boland & Olson, 1979).

Although the general response to adrenaline is a mixed α-constrictory and β-dilatory response, the net increase in PVA seen in this study is in conflict with earlier findings in other species (Reite, 1969; Belaud *et al.* 1971; Bergman *et al.* 1974; Wood, 1975; Dunel & Laurent, 1977; Payan & Girard, 1977; Claiborne & Evans, 1980) and in the cod (Wahlqvist, 1981; Nilsson & Pettersson, 1981; Pettersson & Johansen, 1982). In the investigations of the latter authors the prevailing perfusion pressure was higher prior to adrenaline administration, and the relative influence of the α- and β-responses may simply be due to the conditions prior to administration of the drug.

The increased O_2 transfer induced by adrenaline may not only be due to circulatory adjustments. Haywood, Isaia & Maetz (1977) and Isaia, Maetz & Haywood (1978) presented evidence for an increased permeability for small lipophilic and water soluble substances (such as oxygen) of the secondary lamellae membrane following adrenaline injections. This effect would also explain increased O_2 transfer.

In conclusion, the results presented above demonstrate that the resistance of the branchial vascular bed increases at low $p_{\bar{v}}O_2$, and probably leads to adjustments in

flow through the secondary lamellae. Furthermore, α-adrenoceptor stimulation strongly augments O_2 exchange. Stimulation of the β-adrenoceptor also increases O_2 exchange, but to a lesser extent. These effects, together with changes in ventilation and perfusion, are mechanisms to optimize O_2 uptake during reduced ambient O_2 availability.

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