J. exp. Biol. (1982), **97**, 263–272
With 4 figures
Printed in Great Britain

HYPOXIC VASOCONSTRICTION AND THE EFFECTS OF ADRENALINE ON GAS EXCHANGE EFFICIENCY IN FISH GILLS

BY KNUT PETTERSSON* AND KJELL JOHANSEN
The Department of Zoophysiology, University of Aarhus, DK-8000 Aarhus C., Denmark

(*Received* 27 *May* 1981)

SUMMARY

A gill perfusion technique allowing control of flow and p_{O_2} in the ventral aorta and in the irrigating water is described. The procedure includes measurements of flow and p_{O_2} in the dorsal aorta and the inferior jugular vein. Pressure recordings were made in the ventral and dorsal aortas.

Lowering the perfusion fluid p_{O_2} and/or the irrigating water p_{O_2} increased the branchial vascular resistance, without altering flow distribution. This response is probably released by vasoconstriction proximal to the arterio-venous anastomoses in the gill filaments.

Adrenaline acted on receptors both proximal and distal to the arterio-venous anastomoses: branchial vascular resistance decreased, dorsal aortic flow increased and oxygenation of the perfusion fluid increased.

It is suggested that a combination of a direct myogenic response to hypoxia and release of adrenaline serve to increase O_2 uptake efficiency when fish are exposed to hypoxic stress.

INTRODUCTION

Acute ambient hypoxia is a stress situation calling for compensatory adjustments in exchange and transport of respiratory gases. Adrenergic activity will probably increase in hypoxic stress as in other types of stress.

It is known from perfusion experiments that deoxygenation of perfusion fluid causes gill vascular constriction (Ristori & Laurent, 1977). Fish gills typically have no arterio-arterial by-pass vessels (Laurent & Dunel, 1976; Vogel, Vogel & Kremers, 1973; Vogel, Vogel & Schlote, 1974; Vogel, Vogel & Pfautsch, 1976; Gannon, Campbell & Randall, 1973; Cooke & Campbell, 1980). A preliminary study of the cod, *Gadus morhua*, has confirmed that no secondary lamellar by-pass shunts are present (S. Nilsson & K. Pettersson, unpublished observations). Exceptions to this generality have been reported for the eel, *Anguilla anguilla* (Steen & Kruysse, 1964; Laurent & Dunel, 1976), and for the Channel catfish, *Ictalurus punctatus* (Boland & Olson, 1979). With no by-pass route past the secondary lamellae, hypoxic gill

* Present address: Department of Zoophysiology, University of Göteborg, Box 250 59, S-400 31 Göteborg, Sweden.

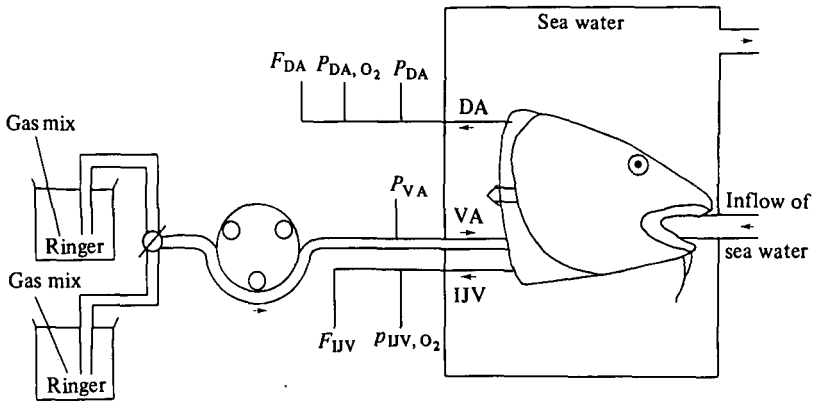

Fig. 1. Schematic representation of the perfusion arrangement. Details are given in the text. DA, Dorsal aorta; F_{DA}, dorsal aorta flow; F_{IJV}, inferior jugular vein flow; IJV, inferior jugular vein; P_{DA}, dorsal aorta pressure; P_{DA,O_2}, dorsal aorta effluent p_{O_2}; P_{IJV,O_2}, inferior jugular vein effluent; p_{O_2}, P_{VA}, ventral aorta pressure; VA, ventral aorta.

vasoconstriction might therefore be an inappropriate response to a call for increased exchange of O_2, unless the vasoconstriction diverts blood to larger and/or better ventilated areas of the gas exchange surfaces or, alternatively, exerts an influence on the blood to water diffusion barrier.

Adrenaline is known to increase arterial compared to venous outflow from perfused gills (Dunel & Laurent, 1977; Payan & Girard, 1977; Pettersson & Nilsson, 1979*a*, *b*; Rowing, Taylor & Rankin, 1979; Claiborne & Evans, 1980). Adrenaline has also been shown to increase arterial p_{O_2} in the eel (Steen & Kruysse, 1964; Peyraud-Waitzenegger, 1979).

The objective of the present study was to elucidate, by perfusion techniques, how gill hypoxic vasoconstriction is mediated and whether the response is elicited by blood-borne hypoxia and/or ambient water-oxygen lack, i.e. to elucidate the sites of the gill vascular system that are influenced by hypoxia. Additionally we have tried to resolve the nature and location of an eventual effect of adrenaline on the gill vascular bed during hypoxia.

MATERIALS AND METHODS

The Atlantic cod, *Gadus morhua*, was used in this study. The material comprised both sexes, weighing between 400 and 1600 g. The fish were kept in recirculated aerated sea water at 15 °C until used.

Preparing for an experiment a fish was stunned with a blow on the head, followed by an injection of *c.* 1000 i.u. of heparin into the caudal vein. Approximately 5 min later the head was removed from the body and a catheter (PE 90) was inserted into the bulbus arteriosus and a slow perfusion was started. We used a filtered saline, containing 1 g glucose l^{-1} for the perfusion (House & Greene, 1965). Another catheter was placed in the dorsal aorta, while the carotid and coeliaco-mesenteric arteries were ligated. The right side inferior jugular vein was catheterized with

PE 50 catheter. The left-side inferior jugular vein was ligated. The head was immersed in saline during the preparation.

Microfil injections through the right-side inferior jugular vein show the gills on both sides to be filled with the cast. Skeletal muscle vessels in 'the tongue region' were poorly filled, indicating that vascularization is poor (S. Nilsson & K. Pettersson, unpublished observations). We therefore consider the perfusion fluid collected in the inferior jugular vein to be predominantly branchial venous effluent.

Fig. 1 shows the experimental arrangement used. The gills were ventilated with sea water at a rate of about 700 ml min^{-1}, using an Eheim pump. p_{O_2} of the 'inspired' water was adjusted by means of a Wösthoff model 301 gas mixing pump. The gills were perfused with saline using a peristaltic pump (Harvard apparatus 6). p_{O_2} of the inflow perfusion fluid was adjusted to the desired level by means of Wösthoff gas mixing pumps.

Perfusion pressure in the ventral aorta (PVA) and the dorsal aorta (PDA) were recorded by Statham P23 pressure transducers connected to a Beckman 66A polygraph. Outflow from the dorsal aorta ($\dot{Q}_a$) was monitored using a 1 mm cannulating probe and a Statham SP 2202 electromagnetic flowmeter. Outflow from the inferior jugular vein ($\dot{Q}_v$) was recorded by means of a Grass photoelectric transducer (drop flow counter) connected to a Gould–Bush Biotach device. Both flow rates were monitored on a Beckman 66A polygraph. O_2 tensions of both effluents were also continually recorded by two O_2-electrodes (Radiometer E 5046) mounted in flow-through cuvettes and connected to Radiometer PHM 71, Mk 2 s, and displayed on a Hewlett-Packard 7132A two-channel potentiometric recorder.

The gills were perfused with a flow rate of about 18 ml kg^{-1} min^{-1}, a flow within the range described to occur *in vivo* (Johansen, 1962; Jones *et al.* 1974; Pettersson & Nilsson, 1980), resulting in a perfusion pressure (PVA) similar to that described *in vivo* (Helgason & Nilsson, 1973; Jones *et al.* 1974; Wahlqvist & Nilsson, 1977; Pettersson & Nilsson, 1980). The perfusion pressure remained stable for the first 3 h of perfusion, or increased very slightly. No temporal variations in the response to hypoxia and/or adrenaline were observed.

In some fish head vascular perfusions described earlier (Payan & Girard, 1977; Claiborne & Evans, 1980), the cephalic circulation has not been separated from the venous drainage of the gills, making it impractical to perfuse against an efferent arterial resistance similar to physiological systemic pressure, since this would also expose the venous system to this pressure, and probably cause severe damage to the preparation (Smith, 1977). In our experiments the dorsal aorta outflow catheter was raised until PDA was at least 1 kPa. In the preparation used, it proved impossible to ligate the mandibular, hypobranchial and orbital arteries, originating from the efferent gill arteries of the 1st and 2nd gill arches, and many small arteries originating from the suprabranchial artery, resulting in a leakage from the perfusion circuit. If dorsal aorta pressure was kept at zero this leakage was restricted, but at 1 kPa it increased markedly. This unavoidable and (in the adrenaline experiments) variable leakage represents an obvious drawback of the preparation and the results are carefully interpreted with this deficiency in mind. All earlier perfusions of fish heads or preparations consisting of more than one gill arch have, however, been affected by the same problem. Johansen & Pettersson (1981) have presented evidence

Table 1. *Effects of lowering P_{VA,O_2} from air-saturation to 23·6 mmHg (mean)*

(FDA and FIJV are expressed as percent of total perfusion gained in the catheters. The decrease in FDA depicts an increased leakage, since FIJV is not changed. Mean values ± S.E.M. When indicated, statistical treatment was performed between the groups. NS, not significant; * $P < 0.05$; ** $P < 0.01$. $n = 10$.)

p_{VA,O_2} (mmHg)	$p_{DA\ O_2}$ (mmHg)	p_{IJV,O_2} (mmHg)	$p_{DA,O_2}-p_{IJV,O_2}$ (mmHg)	F_{DA} (% $\dot{Q}$)	F_{IJV} (% $\dot{Q}$)	P_{VA} (kPa)
156	126 ± 4·8	59 ± 6·7	67 ± 6·0	20·3 ± 1·42 **	4·6 ± 0·47 NS	5·63 ± 0·49 **
23·6	34 ± 5·7	10 ± 1·7	28 ± 4·6	19·2 ± 1·45	4·6 ± 0·52	6·57 ± 0·45

F_{DA}, Dorsal aorta flow; F_{IJV}, inferior jugular vein flow; P_{DA,O_2}, dorsal aorta effluent p_{O_2}; $P_{IJV\ O_2}$, inferior jugular vein effluent p_{O_2}; P_{VA}, ventral aorta pressure; P_{VA,O_2}, perfusion medium p_{O_2}.

suggesting that leakage will occur mainly from the efferent arterial system. They compared gill tissue O_2 uptake from perfused 'leak free' single gill arches with head preparations like the one described here, and concluded that leakage occurs mainly from distal arterial vessels of little or no consequence in gill gas exchange and O_2 uptake.

The experiments were performed at 10 °C. The drug adrenaline bitartrate was obtained from Sigma. It was dissolved in saline to a final concentration of 10^{-6} M. The experiments were performed in two series. In the first series (A) both P_{i,O_2} and P_{VA,O_2} were air-saturated and showed if adrenaline caused any effects on P_{DA,O_2} when no oxygen tension gradient from irrigating water to perfusion fluid existed. This series served as control to series B, in which P_{VA,O_2} was decreased to *c.* 22 mmHg (Table 2). Wahlqvist & Nilsson (1980) have reported that plasma adrenaline concentration from severely stressed cod can exceed 3×10^{-7} M. Statistical treatment was performed according to the Wilcoxon matched-pair signed-ranks test.

RESULTS

In the experiments involving the effects of hypoxia on the gill vascular system, perfusions were started using air-saturated perfusion fluid and sea water for irrigation of the gills. After stable conditions were established, the p_{O_2} of the perfusion fluid was instanteously decreased from *c.* 155 to 23 mmHg by shifting to another reservoir. In three experiments the p_{O_2} of the irrigating sea water was also decreased (to *c.* 60 mmHg), but always after initiation of 'internal' hypoxia. The results are summarized in Table 1, and a typical response is illustrated in Fig. 2. A small but significant decrease in dorsal aorta outflow occurred, but no change was apparent in venous outflow rate (Table 1). There was also a significant increase in branchial vascular resistance expressed as an increase in ventral aortic pressure (perfusion flow was kept constant). The successive increments in ventral aortic perfusion pressure with initiation of 'internal' hypoxia and secondly 'external' hypoxia (as was the case in all experiments performed), testify that the hypoxic vasoconstrictor response can be released by both types of hypoxic stimuli (Fig. 2).

Adrenaline (10^{-6} M) did not significantly affect dorsal aorta p_{O_2} when no oxygen tension gradient existed between ambient water and perfusion fluid (Table 2, Fig. 3*a*)

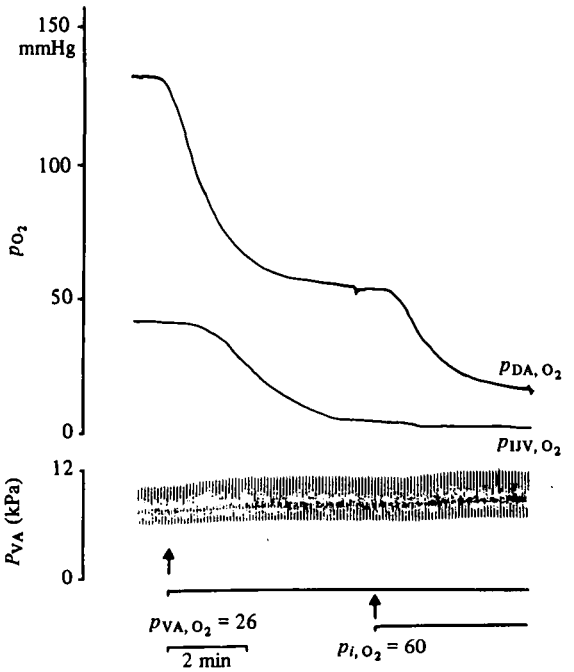

Fig. 2. Direct recordings of the responses to hypoxia by decreasing P_{VA,O_2} and subsequently P_{i,O_2}, at times indicated by arrows. Upper trace, P_{DA,O_2}; middle trace, P_{IJV,O_2}; lower trace, P_{VA}. Note the increases in P_{VA}. Legend: P_{DA,O_2}: dorsal aorta effluent p_{O_2}; P_{IJV,O_2}: inferior jugular vein effluent p_{O_2}; P_{i,O_2}: irrigating water p_{O_2}; P_{VA}: ventral aorta pressure; P_{VA,O_2}: perfusion medium p_{O_2}.

Table 2. *Effects of adrenaline* (10^{-6} M) *when* P_{VA,O_2} *as well as* P_{i,O_2} *were air-saturated (series A), and when* P_{VA,O_2} *was about 22·4 mmHg (mean) and* P_{i,O_2} *air-saturated (series B)*

(Note the increase in FDA without a simultaneous decrease in F_{IJV}, indicating a constriction of the leaking vessels. Also note the increase in P_{DA,O_2} in series B, an effect not seen in series A, and therefore showing increased efficiency in O_2 exchange between water and perfusion fluid. Mean values ± S.E.M. $n = 7$ in both series. Legend: F_{DA}, dorsal aorta flow; F_{IJV}, inferior jugular vein flow; P_{DA,O_2}, dorsal aorta effluent P_{O_2}; P_{IJV,O_2}, inferior jugular vein effluent p_{O_2}; PVA, ventral aorta pressure.)

Series	Treatment	p_{DA,O_2} (mmHg)	p_{IJV,O_2} (mmHg)	$p_{DA,O_2}-p_{IJV,O_2}$ (mmHg)	F_{DA} (% $\dot{Q}$)	F_{IJV} (% $\dot{Q}$)	P_{VA} (kPa)
A	Control	133 ± 3·3 NS	58 ± 8·6 *	75 ± 7·0 *	19·9 ± 1·27 *	4·3 ± 0·50 NS	5·93 ± 0·48 *
	Adrenaline	136 ± 4·0	15 ± 3·5	121 ± 4·0	33·0 ± 3·25	3·3 ± 0·70	5·26 ± 0·59
B	Control	37 ± 6·6 *	6·6 ± 2·0 *	30 ± 5·5 *	20·3 ± 1·35 *	4·7 ± 0·54 NS	6·23 ± 0·45 *
	Adrenaline	53 ± 8·9	3·9 ± 1·5	49 ± 8·6	28·5 ± 2·7	4·7 ± 0·80	4·81 ± 0·15

Outflow through the dorsal aorta increased, while the perfusion pressure markedly decreased (Fig. 3*a*). In another series of experiments when adrenaline was administered while a p_{O_2} gradient of about 135 mmHg (from water to perfusion fluid) was present, a marked increase in both dorsal aortic outflow rate and O_2 tension occurred. There was no change in venous outflow rate. Ventral aortic pressure fell more markedly than in the first series (Table 2, Fig. 3*b*).

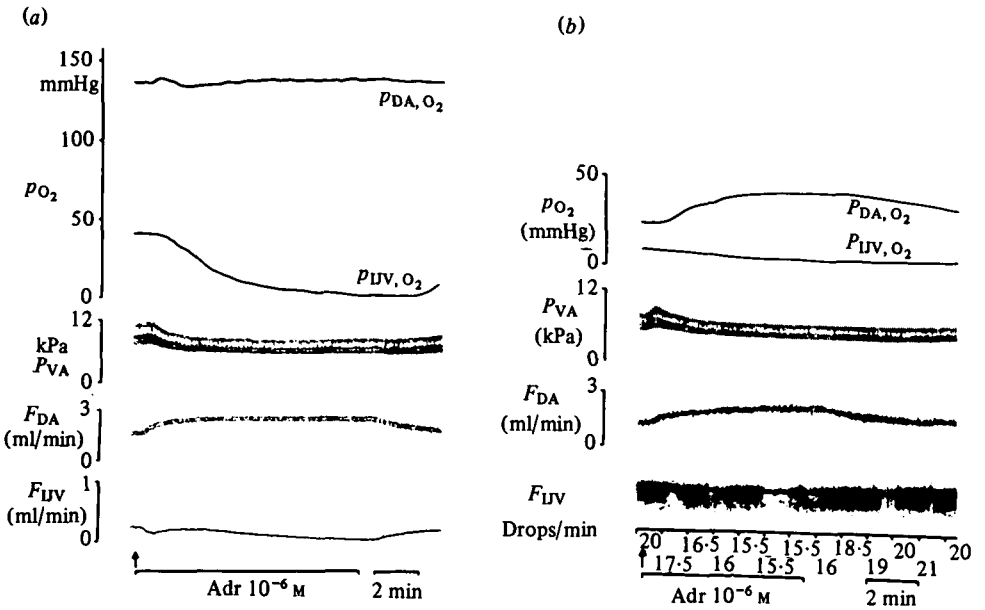

Fig. 3(*a*). Response to adrenaline (10^{-6} M) when $P_{i,O_2} = P_{VA,O_2}$ (series A). Traces, from top to bottom: P_{DA,O_2}; P_{IJV,O_2}; P_{VA}; F_{DA}; and F_{IJV}. F_{DA} increased in all preparations, F_{IJV} decreased in 6 of 7 preparations. (*b*) Response to adrenaline (10^{-6} M) when $P_{i,O_2} \gg P_{VA,O_2}$ (series B). Tracings as in (*a*). Note the marked increase in P_{DA,O_2}. F_{DA} increased in all preparations, F_{IJV} decreased in three out of seven experiments (as shown here), and increased or remained constant in the others. F_{DA}, dorsal aorta flow; F_{IJV}, inferior jugular vein flow; P_{DA,O_2}, dorsal aorta effluent p_{O_2}; P_{IJV,O_2}, inferior jugular vein effluent p_{O_2}; P_{VA}, ventral aorta pressure.

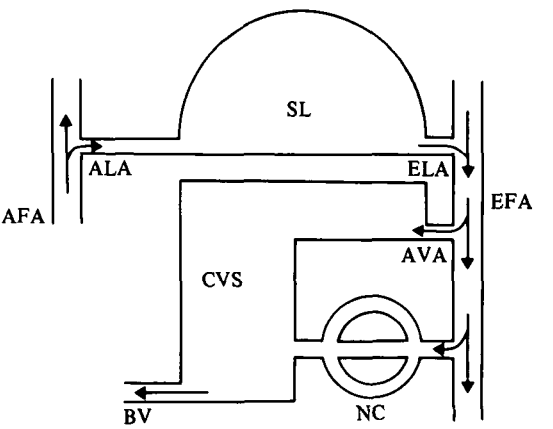

Fig. 4. A simplified model for the branchial circulation in the cod. AFA, Afferent filamental artery; ALA, afferent lamellar artery; AVA, arterio-venous anastomoses; BV, branchial vein; CVS, branchial venous compartment; EFA, efferent filamental artery; ELA, efferent lamellar artery; NC, gill nutritive circulation; SL, secondary lamellae.

DISCUSSION

Variable results have been reported on the effect of hypoxia on gill vascular resistance. In intact fish it seems that overall branchial vascular resistance increases during acute exposure to hypoxic water as shown for trout, *Salmo gairdneri* (Holeton & Randall, 1967). Farrell, Daxboeck & Randall (1979) recorded no vasoconstriction in perfused gills from the lingcod, *Ophiodon elongatus*, when the perfusion fluid was deoxygenated (the degree of hypoxia is not reported). Ristori & Laurent (1977), however, reported that gills from the rainbow trout showed an increased vascular resistance in response to a change in perfusion fluid p_{O_2} from aerated levels to about 20 mmHg in a perfused head preparation. They concluded that the major site of hypoxic vasoconstriction was located distal to the arterio-venous anastomoses (see Fig. 4), since the venous outflow increased while the arterial decreased.

They ascribe the decrease in dorsal aortic outflow to constriction of sphincters at the base of the efferent filamental arteries (EFA, Fig. 4) (Laurent & Dunel, 1976). We believe this explanation must be dismissed, since it would require that the arterial cephalic and/or systemic arterial 'leaks' would have to constrict in response to hypoxia as well, since in their constant pressure experiments both dorsal aortic and cephalic/branchial venous flow decreased. In our experimental series on the Atlantic cod no changes in the relative outflow levels occurred; a result suggesting that the vasoconstrictors occurs proximal to the arterio-venous anastomoses (Fig. 4). The slight decrease in dorsal aortic outflow recorded here could be explained by a hypoxic vasodilation of extrabranchial systemic arteries (including the 'leaks'), which is the typical response to hypoxia of systemic arterioles in other vertebrates. The inferior jugular vein flow is not affected since it consists of almost pure branchial venous outflow.

The perfusion experiments involving a hypoxia stimulus from the external, ambient water side gave, in our experiments, a qualitatively similar response as 'internal' hypoxia. This result has two important consequences. Firstly, the vasoconstriction must in part occur distal to the afferent filamental and lamellar arteries, since these sites cannot be reached by an 'external' hypoxic stimulus. Secondly, since the normal gill in an intact fish receives deoxygenated blood (often at O_2 tensions below those employed in others and the present perfusion experiments), a natural hypoxic stimulus should therefore influence vascular channels that are not 'normally' hypoxic. These must then be located distally within the secondary lamellae or, more probably, in the efferent lamellar arterioles. This site is normally perfused by oxygenated blood when the gills are functional in gas exchange. 'External hypoxia', which is the physiologically significant hypoxia for fish, can be rapidly screened by O_2-sensitive smooth muscle at this site. Vasoconstriction at such sites will, if it affects enough secondary lamellae, cause an increase in afferent lamellar pressure (Fig. 2). This response may in turn cause recruitment of unperfused lamellae (Booth, 1979), thereby increasing the available surface area for gas eschange, and perhaps alter the lamellar size and geometry for more effective diffusion exchange. Only in this way will the hypoxic constriction impart a compensatory advantage to a situation of external O_2 shortage.

The chief response to adrenaline is a decreased resistance to perfusion and has

been noted by many others (Krawkow, 1913; Östlund & Fänge, 1962; Reite, 1969; Belaud, Peyraud-Waitzenegger & Peyraud, 1971; Rankin & Maetz, 1971; Wood, 1975; Payan & Girard, 1977; Smith, 1977; D'Amico Martel & Cech, 1978; Pettersson & Nilsson, 1979*a*, *b*, 1980; Claiborne & Evans, 1980; Nilsson & Pettersson, 1981). In our experiments dorsal aorta flow increased in all cases, while flow in the inferior jugular vein exhibited great variations. The leak problem associated with head perfusions (discussed above) probably influenced these results. In this way, α-adrenergic constriction of systemic 'leakage' vessels increases the outflow rate on the venous side. Meanwhile, α-adrenergic stimulation of the arterio-venous anastomoses (Fig. 4) reduces venous outflow (Payan & Girard, 1977; Nilsson & Pettersson, 1981). The overall combined effect of these counteracting responses is expressed by the large variability in the outflow from the inferior jugular vein catheter, where no significant effects were found (Table 2).

Adrenaline injections *in vivo* increases dorsal aorta p_{O_2} (Steen & Kruysse, 1964; Peyraud-Waitzenegger, 1979). This result has now been confirmed in a perfused preparation, and demonstrated to depend on direct effect of adrenaline on the gills (Table 2, Fig. 3*b*). Adrenaline did not affect dorsal aorta p_{O_2} in series A, where no O_2 tension gradient existed between the irrigating water and the perfusion fluid (Table 2, Fig. 3*a*), showing that adrenaline does not affect the O_2 requirement of the branchial tissue. The increase in dorsal aorta p_{O_2} by adrenaline in series B is, therefore, due to an increased gas exchange efficiency. It is the relatively low flow rate through the venous system that causes the high utilization of O_2 apparent in the venous perfusion fluid (Table 2).

Booth (1979) reported an increase in the number of perfused secondary lamellae following adrenaline injections in the rainbow trout, a finding supported by data from Holbert, Boland & Olson (1979). Our results, which demonstrated an increased O_2 transfer across the gills, support this possibility. Another explanation for the increased efficiency in O_2 transfer from the irrigating water to the perfusion fluid could be increased permeability of the secondary lamellar membrane to lipophilic substances such as oxygen (Haywood, Isaia & Maetz, 1977; Isaia, Maetz & Haywood, 1978).

In conclusion, this paper describes a myogenic vasoconstriction of the branchial vascular bed in response to hypoxia, probably leading to lamellar recruitment and increased functional surface area in the gills. Furthermore, adrenaline enhances oxygen transfer and causes an increased systemic blood flow. The combination of hypoxic vasoconstriction and adrenaline release, such as is likely to occur during reduced ambient O_2 availability, will thus optimize O_2 transfer as well as improve the arterial O_2 transport.

REFERENCES

BELAUD, A., PEYRAUD-WAITZENEGGER, M. & PEYRAUD, C. (1971). Etude comparée des réactions vasomotrices des branchies perfusées de deux téleostéens: la carpe et la congre. *C. r. Séanc. Soc. Biol.* **165**, 1114–1118.

BOLAND, E. J. & OLSON, K. R. (1979). Vascular organisation of the catfish gill filament. *Cell Tissue Res.* **198**, 487–500.

BOOTH, J. H. (1979). The effect of oxygen supply, epinephrine and acetylcholine on the distribution of blood flow in trout gills. *J. exp. Biol.* **83**, 31–39.

CLAIBORNE, J. H. & EVANS, D. H. (1980). The isolated perfused head of the marine teleost fish, *Myoxocephalus octodecimspinosus*: Hemodynamic effects of epinephrine. *J. comp. Physiol.* **138** B, 79–86.

COOKE, I. R. C. & CAMPBELL, G. (1980). The vascular anatomy of the gills of the smooth toadfish, *Torquiginer glaber* (Teleostei: Tetraodontidae). *Zoomorphologie, Berlin* **94**, 151–166.

D'AMICO MARTEL, A. L. & CECH, J. J. (1978). Peripheral vascular resistance in the gills of the winter flounder, *Pseudopleuronectes americanus*. *Comp. Biochem. Physiol.* **59** A, 419–423.

DUNEL, S. & LAURENT, P. (1977). La vascularisation branchiale chez l'anguille: action de l'acétylcholine et de l'adrénaline sur la répartition d'une resine polymérisable dans les différents compartiments vasculaires. *C. r. hebd. Séanc. Acad. Sci., Paris* **284**, 2011–2014.

FARRELL, A. P., DAXBOECK, C. & RANDALL, D. J. (1979). The effect of input pressure and flow on the pattern and resistance to flow in the isolated perfused gill of a teleost fish. *J. comp. Physiol.* **133** B, 233–240.

GANNON, B. J., CAMPBELL, G. & RANDALL, D. J. (1973). Scanning electron microscopy of vascular casts for the study of vessel connections in a complex vascular bed – the trout gill. *31st Am. Proc. Electron, Microsc. Soc. Am.* **31**, 442–443.

HAYWOOD, G. P., ISAIA, J. & MAETZ, J. (1977). Epinephrine effects on branchial water and urea flux in rainbow trout. *Am. J. Physiol.* **232**, R110–R115.

HELGASON, S. ST. & NILSSON, S. (1973). Drug effects on pre- and post-branchial blood pressure and heart rate in a free swimming marine teleost, *Gadus morhua*. *Acta physiol. scand.* **88**, 533–540.

HOLBERT, P. W., BOLAND, E. J. & OLSON, K. R. (1979). The effect of epinephrine and acetylcholine on the distribution of red cells within the gills of the channel catfish, *Ictalurus punctatus*. *J. exp. Biol* **79**, 135–146.

HOLETON, G. & RANDALL, D. J. (1967). Changes in blood pressure in the rainbow trout during hypoxia. *J. exp. Biol.* **46**, 297–305.

HOUSE, C. R. & GREEN, K. (1965). Ion and water transport in the isolated intestine of the marine teleost, *Cottus scorpius*. *J. exp. Biol.* **42**, 177–189.

ISAIA, J. & MAETZ, J. & HAYWOOD, G. P. (1978). Effects of epinephrine on branchial non-electrolyte permeability in rainbow trout. *J. exp. Biol.* **74**, 227–237.

JOHANSEN, K. (1962). Cardiac output and pulsatile flow in the teleost, *Gadus morhua*. *Comp. Biochem. Physiol.* **7**, 169–174.

JOHANSEN, K. & PETTERSSON, K. (1981). Gill O_2 consumption in a teleost fish, *Gadus morhua*. *Resp. Physiol.* **44**, 277–284.

JONES, D. R., LANGILLE, B. L. RANDALL, D. J. & SHELTON, G. (1974). Blood flow in the dorsal and ventral aortas of the cod, *Gadus morhua*. *Am. J. Physiol.* **226**, 90–95.

KRAKOW, N. P. (1913). Über die Wirkung von Giften auf die Gefässe Isolierter Fischkiemen. *Pflugers Arch. ges. Physiol.* **151**, 583–603.

LAURENT, P. & DUNEL, S. (1976). Functional organisation of the teleost gill. I. Blood pathways. *Acta zool., Stockh.* **57**, 189–209.

NILSSON, S. & PETTERSSON, K. (1981). Sympathetic nervous control of blood flow in the gills of the Atlantic cod, *Gadus morhua*. *J. comp. Physiol.* (In the Press.)

ÖSTLUND, E. & FÄNGE, R. (1962). Vasodilation by adrenaline and noradrenaline, and the effects of some other substances on perfused fish gills. *Comp. Biochem. Physiol.* **5**, 307–309.

PAYAN, P. & GIRARD, J. P. (1977). Adrenergic receptors regulating patterns of blood flow through the gills of trout. *Am. J. Physiol.* **232**, H18–H23.

PETTERSON, K. & NILSSON, S. (1979*a*). Nervous control of the branchial vascular resistance of the Atlantic cod, *Gadus morhua*. *J. comp. Physiol.* **129** B, 179–183.

PETTERSSON, K. & NILSSON, S. (1979*b*). Control of the branchial blood flow in the Atlantic cod, *Gadus morhua*. In *Animals and Environmental Fitness*, pp. 39–40. Pergamon Press.

PETTERSSON, K. & NILSSON, S. (1980). Drug-induced changes in cardio-vascular parameters in the Atlantic cod, *Gadus morhua*. *J. comp. Physiol.* **137** B, 131–138.

PEYRAUD-WAITZENEGGER, M. (1979). Simultaneous modification of ventilation and arterial p_{O_2} by catecholamines in the eel, *Anguilla anguilla* L.: participation of α and β Effects. *J. comp. Physiol.* **129** B, 343–354.

RANKIN, J. C. & MAETZ, J. (1971). A perfused teleostean gill preparation: vascular actions of neurohypophysial hormones and catecholamines. *J. Endocrin.* **51**, 621–635.

REITE, O. B. (1969). The evolution of vascular smooth muscle responses to histamine and 5-hydroxytryptamine. I. Occurrence of stimulatory actions in fish. *Acta physiol. scand.* **75**, 221–239.

RISTORI, M. T. & LAURENT, P. (1977). Action de l'hypoxie sur le système vasculaire branchial de la tête perfusée de truite. *C. r. Séanc. Soc. Biol.* **171**, 809–813.

ROWING, C. G. M., TAYLOR, S. F. & RANKIN, J. C. (1979). Branchial circulation in the eel, *Anguilla anguilla*: anatomy, physiology and pharmacology. In *Abstracts from the 2nd Symposium on Fish Physiology*, Göteborg, June 1979.

SMITH, D. G. (1977). Sites of cholinergic vasoconstriction in trout gills. *Am. J. Physiol.* **223**, R222–R229.

STEEN, J. B. & KRUYSSE, A. (1964). The respiratory function of the teleostean gills. *Comp. Biochem. Physiol.* **12**, 127–142.

VOGEL, W., VOGEL, V. & KREMERS, H. (1973). New aspects of the intrafilamental vascular system in gills of a euryhaline teleost, *Tilapia mossambica*. *Z. Zellforsch. mikrosk. Anat.* **144**, 573–583.

VOGEL, W., VOGEL, V. & SCHLOTE, W. (1974). Ultrastructural study of arterio-venous anastomoses in gill filaments of *Tilapia mossambica*. *Cell Tiss. Res.* **155**, 491–512.

VOGEL, W., VOGEL, V. & PFAUTSCH, M. (1976). Arterio-venous anastomoses in rainbow trout gill filaments. *Cell Tiss. Res.* **167**, 373–385.

WAHLQVIST, I. (1980). Effects of catecholamines on isolated systemic and branchial vascular beds of the cod, *Gadus morhua*. *J. comp. Physiol.* **137** B, 139–144.

WAHLQVIST, I. & NILSSON, S. (1977). The role of sympathetic fibers and circulating catecholamines in controlling the blood pressure in the cod, *Gadus morhua*. *Comp. Biochem. Physiol.* **57** C, 65–67.

WAHLQVIST, I. & NILSSON, S. (1980). Adrenergic control of the cardiovascular system of the Atlantic cod, *Gadus morhua*, during 'stress'. *J. comp. Physiol.* **137** B, 145–150.

WOOD, C. M. (1975). A pharmacological analysis of the adrenergic and cholinergic mechanisms regulating branchial vascular resistance in the rainbow trout (*Salmo gairdneri*). *Can. J Zool.* **53**, 1569–1577.