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CARDIOVASCULAR CHANGES IN THE LINGCOD (*OPHIODON ELONGATUS*) FOLLOWING ADRENERGIC AND CHOLINERGIC DRUG INFUSIONS

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SUMMARY

Adrenergic and cholinergic agonists were infused into the ventral aorta to evoke gill vasoactivity in the lingcod, *Ophiodon elongatus*. Arterial blood pressures were changed, and cardiac output and stroke volume were increased. As a consequence both the pressure and flow profiles across the gill were altered, and these changes should alter the pattern of lamellar perfusion. The changes in cardiac function were apparently reflexly mediated.

INTRODUCTION

The control of lamellar perfusion patterns in fish is not fully understood. Neural and humoral mediated vasoactivities are presumeably important in the control since many investigations have indicated that adrenergic and cholinergic vascular receptors are present in the gills (Wood, 1974, 1975, 1977; Smith, 1977; Payan & Girard, 1977; Dunel & Laurent, 1977). Nevertheless, unequivocal evidence of lamellar innervation or vasoactivity is lacking. Thus neural or humoral control of lamellar arterioles has still to be unequivocally demonstrated. In view of this, it is possible that changes in the pressure and flow profiles across the gill cause important, passive changes in lamellar perfusion.

The lamellar blood space and the number of lamellae perfused are pressure and flow dependent in *Ophiodon elongatus* (Farrell, Daxboeck & Randall, 1979; Farrell *et al.*, 1980) and in *Ictalurus punctatus* (Holbert, Boland & Olson, 1979). Also, Opdyke, Holcombe & Wilde (1979) concluded for *Squalus acanthias* that the reduced gill resistance associated with an increase in flow was due to passive changes in the branchial vasculature. Changes in lamellar perfusion may therefore be mediated in several ways. Arteriolar vasoactivity may regulate flow and pressure to the lamellae. Alternatively, the pressure and flow profiles across the gills may be modulated, which could be achieved by either vasoactivity in larger branchial and systemic vessels, or by alterations in cardiac output and rate. Furthermore, these mechanisms need not be independent since changes in vascular tone will almost certainly be accompanied by changes in cardiac activity, but they cannot be predicted.

The purpose of this study was to establish what cardiovascular changes are asso-

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ciated with vasoactivity in the lingcod, *O. elongatus*. Unlike in previous studies, cholinergic and adrenergic agonists were infused into the ventral aorta to evoke rapid branchial vasoactivity, but delay or avoid direct cardiac stimulation. The lingcod was selected as the experimental fish so that simultaneous and direct recordings of the pre- and post-branchial blood pressures and cardiac output could be made.

MATERIALS AND METHODS

Lingcod weighing 3·5–6·5 kg were obtained from local fishermen and held in large tanks supplied with running, aerated salt water at 9 to 11 °C. They were fed a maintenance diet of live rainbow trout, but were starved several days before experiments. Cortland saline (Wolf, 1963), modified for saltwater fish (an additional 4·35 $g.l^{-1}$ NaCl), was used for all drug dilutions and injections. The saline was heparinized (10 $i.u.ml^{-1}$) prior to injection.

Routine surgical protocol to allow in vivo measurements of cardiac output, pre- and post-branchial pressure, and ventilation

Lingcod were deeply anaesthetized (0·02%, w/v tricaine methane sulphonate (MS 222) in sea water), placed ventral side up in an operating sling and cooled with an ice pack on the body. The gills were continuously irrigated with cool (8–14 °C), recirculating sea water containing anaesthetic (0·005%, w/v, MS222). A 4–6 cm incision was made in the skin along the inside of the opercular cavity wall in line with, and slightly ventral to the ventral aorta (VA). The muscle mass, which is thinnest in this area, was carefully teased aside to expose the pericardium and the more ventral hypobranchial artery. The pericardium surrounding the VA was opened and a pneumatic cuff (Shoukas, 1977) was implanted around the anterior end of the VA before the point where it divides. A cuff-type electromagnetic flow probe (Biotronics) was selected for good fit (10–20% vessel constriction; B. W. Langille, personal communication) and was implanted around the VA near to the bulbus arteriosus. The pneumatic cuff and the flow probe were thus maximally separated on the vessel. Both cuffs were anchored to their adjacent muscle mass with silk thread sutures. The bevelled tip of a nonocclusive cannula (50 cm of polyethylene tubing, PE 60) was introduced upstream into the VA with the aid of a Medicut cannula (Aloe Medical, St Louis). The PE tubing was secured to the body of the flow probe and to the adjacent muscle mass. The wounds in the muscle mass and in the skin were closed separately with continuous silk thread sutures. The dorsal aorta (DA) was cannulated via the efferent branchial artery in one of the 4th gill arches (Jones *et al.* 1974). The cannula (50 cm of PE 100 tubing) was introduced downstream into the ligated efferent arch artery and advanced 2 to 4 cm towards the DA. The artery was secured around the cannula, the skin incision was closed, and the cannula was firmly anchored to the body wall. The effect of the DA cannulation on cardiac output was not assessed. If anything the cardiac output may have been reduced by up to one-eighth. (The venolymphatics were unaffected, so some 4th gill arch blood would still flow through these vessels). The buccal and opercular cavities were cannulated (PE 200) in some experiments using methods similar to those described by Holeton & Randall (1967*a*). The

mplete surgical procedure routinely lasted about 1 h and was accomplished with tle blood loss.

Recovery of the fish from anaesthetic began during the final stages of surgery when the icepack was removed and the gills were irrigated with fresh saltwater. The fish usually began strong opercular movements within 5 min and could then be placed in the experimental holding aquarium where gill irrigation was ensured by fitting a temporary gill irrigation tube. Within 5 to 10 min the fish were sufficiently revived to swim off the irrigation tube and sustain their own gill ventilation. The experimental aquarium was a darkened, covered plexiglass box that limited forward and lateral movements, but did not prevent them. The saltwater supply was flow-through, aerated and at 10–11 °C.

Cardiac output, blood pressures and ventilation pressures were monitored simultaneously on individual lingcod after a 12–24 h postoperative recovery and acclimation. Records were subsequently collected for up to 6 days and often continuously for 10–15 h in any one day. On one occasion monitoring was continued over night on an undisturbed fish. Noise was minimized and kept constant (white noise) during experimentation. Experiments were commenced only on resting fish. Results were not analysed if the fish struggled or was disturbed during the experiment.

Intravascular administration of adrenergic and cholinergic drugs

Adrenergic and cholinergic agonist drugs were infused intravascularly via the indwelling VA and DA cannulae (pre-gill infusions respectively). All drugs were slowly infused in concentrated form using a 0·1–0·25 ml carrier volume of heparinized saline. (Saline infusions evoked no measurable cardiovascular changes.) The drug concentrations given below are expressed as final blood concentrations using a blood volume estimate of 5%, and were selected after trials to determine the minimum dosage required to produce a measureable cardiovascular response. (Higher concentrations often promoted struggling in the fish.)

O-acetylcholine chloride (ACh; 0·1 $\mu g.ml^{-1}$) and carbamylcholine chloride (CARB; 1–10 $\mu g.ml^{-1}$) were used as cholinergic agonists. CARB was used specifically to evoke a bradycardia so that a zero ventral aortic flow could be obtained. L-arterenol bitartrate (NA; 0·1 $\mu g.ml^{-1}$) was used as a strong α- and weak β-adrenoceptor stimulant. DL-isoproterenol HCl (ISOP; 0·01–0·1 $\mu g.ml^{-1}$) was used as a β-adrenoceptor agonist.

ACh and NA are naturally occurring neuro-transmitters in other fish and both are rapidly metabolized. ISOP and CARB, however, are synthetic agonists which are slowly metabolized over a period of up to 1 h. Recovery periods between successive drug injections were 1 h following NA or ACh, and 2–3 h or overnight following ISOP or CARB administration. These recovery periods were prolonged if there was any doubt concerning the fish's recovery. The observed cardiovascular effects of NA and ACh never lasted more than 15 min and those of CARB and ISOP never lasted more than 25 min.

Recording systems

Blood and water pressures were detected using saline-filled pressure transducers (Statham P23Db, P23BB and P23V) connected to the saline-filled cannulae. The

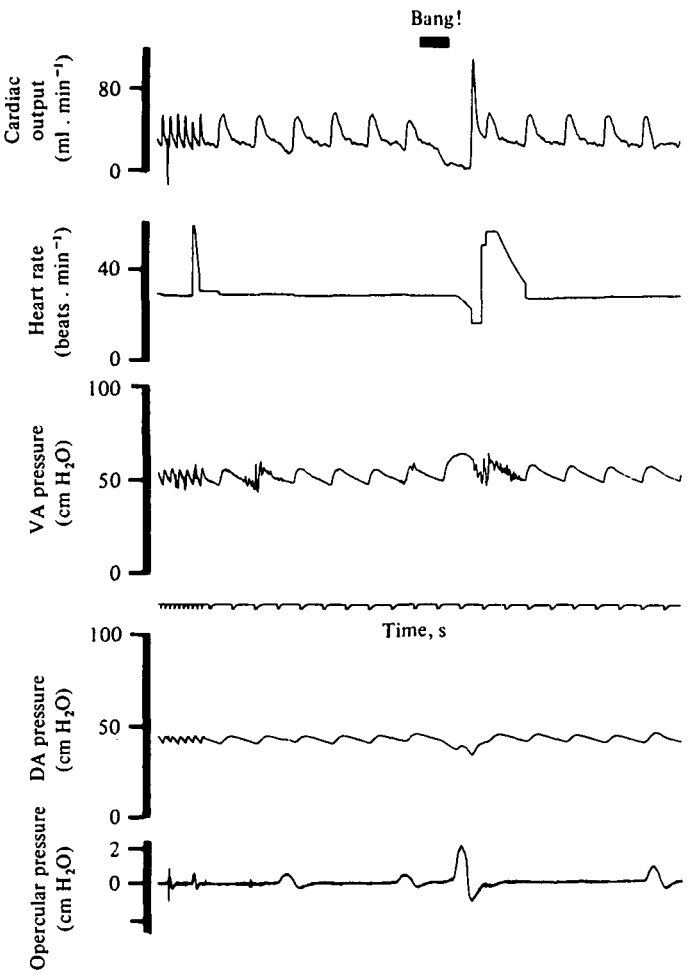

Fig. 1. Simultaneous records of cardiovascular and respiratory variables that were normally recorded during every experiment. The 4 kg lingcod was unanaesthetized and resting. 'Bang' represents a banging on the tank, used to establish a zero VA flow during diastole.

transducers were calibrated daily using pressures generated by a static water column. The transducers were always balanced to a zero signal at the appropriate water or saline level. This balance point was checked frequently throughout the course of an experiment. The transducers faithfully reproduced oscillating pressures in the range being monitored since the frequency response of the fluid-filled transducers and their associated catheters was in excess of 10 Hz, as determined by the Hanson 'pop' test (McDonald, 1960).

Pulsatile VA blood flow ($\dot{Q}$) was monitored from the signals of the implanted electromagnetic flow probe using a BL610 flow meter (Biotronix Ltd), set for a 12·5 Hz frequency response. Zero VA flow, for calibration of the flowmeter, was achieved either by briefly occluding the vessel with the pneumatic cuff, or by disturbing the fish, or by a CARB infusion. The latter two procedures evoked a bradycardia during which VA blood flow stopped for a brief period during diastole. An example of zero VA blood

flow during diastole following a disturbance (banging the aquarium) is illustrated in Fig. 1. Changes in signal level due to flow probe movement were either corrected for, or a new zero signal was established using one of the above techniques. Drift of the flow probe signal during lengthy recording periods was not extensive between zero calibrations and was ignored. The signals from individual flow probes were calibrated either *in situ* or *in vitro* with measured saline perfusions. Heart rate was monitored in some experiments using a rate meter triggered by the pulsatile blood flow signal.

All electrical signals were suitably amplified for continuous monitoring and recording on a Brush 260 six channel chart recorder. The blood pressures and blood flow signals were also stored on magnetic tape using a Tandberg series 115 tape recorder (equipped with a flutter compensation device).

The stored signals were used for subsequent analyses, when they were replayed through 5 Hz band pass filters onto the chart recorder. The filters and the flutter compensation device reduced the background electrical noise produced by the tape recorder, but did not alter the signals significantly.

Analysis

Ventral aortic (VA) and dorsal aortic (DA) pressures are described by their mean and pulse pressures, where pulse = systole − diastole and mean = diastole + $\frac{1}{3}$ pulse. All pressures are expressed in cm H_2O (1 cm H_2O = 0·098 kPa). Stroke volume (SV) was determined by planimetry of the pulsatile flow record. A high chart speed (5 $mm.s^{-1}$) was used to improve the accuracy of area measurements. Beat to beat heart rate (HR) was also determined from the flow record and was converted into beats. min^{-1}. Cardiac output ($\dot{Q}$) is the product of stroke volume and heart rate. SV and $\dot{Q}$ are expressed per kg fish weight (ml. kg^{-1} and ml. min^{-1}. kg^{-1} respectively). Gill resistance (R_g) is defined as $\triangle P_g/\dot{Q}$ and systemic resistance (R_s) as DA mean/$\dot{Q}$,with resistance units of cm H_2O. min. ml^{-1}. $\triangle P_g$ is the blood pressure drop across the gill (VA mean − DA mean). The R_g value assumes the major resistance is in the lamellar units where all $\dot{Q}$ passes (Farrell, 1980*a*, *b*), since some post-lamellar gill vessels do not receive all the $\dot{Q}$ on account of the post-lamellar origin of the venolymphatic system in the lingcod. The R_s value assumes that venous return to the heart is at ambient pressure (see Randall, 1968; Randall, 1970*a*).

The cardiovascular data were compiled, initially graphed and statistically analysed using a PDP 11 computer. A 95% confidence limit was used in tests for significant differences from the resting values using a single tailed Student's *t* test.

RESULTS

Blood flow and respiration in unanaesthetized, resting lingcod

Fish were observed in the sea at depths of up to 30 m. They rested on the sea floor propped on their large pectoral fins. Each ventilation was perceptible only by slight opercular movements that were separated by several seconds. In the holding aquaria, fish adopted a similar resting behaviour. In the experimental aquarium, buccal cavity movements during ventilation in resting fish were hardly perceptible. The mouth remained slightly open, but was sealed by the buccal flap during buccal compression.

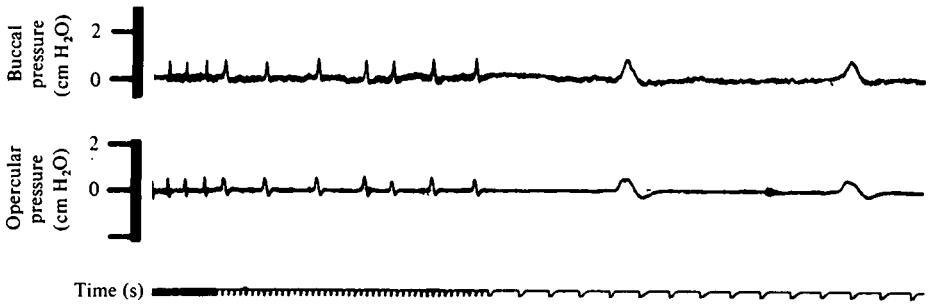

Fig. 2. Typical simultaneous records of the buccal and opercular water pressures associated with gill ventilation in resting, unaesthetized lingcod. The respiratory pause is indicated by the period of ambient pressure (0) in both the opercular and buccal cavities.

The period of reduced pressure in the opercular cavity was visually correlated with opercular abduction. The hydrostatic pressure gradient across the gills was small (Fig. 2). The maximum differential pressure between the opercular and buccal cavities was 0·4 cm H_2O, but was at ambient pressure during the respiratory pause prior to inhalation (Fig. 2). The ventilation rates ranged from 6 to 16 breaths.min^{-1} with a mean resting value of $11{\cdot}9 \pm 0{\cdot}4$ (n = 60 for 8 fish).

Heart rate often fluctuated from beat to beat ($\bar{x} = 29{\cdot}8 \pm 0{\cdot}4$, Table 1), but $\dot{Q}$ was unchanged. The mean resting heart rate for each fish ranged from 22 to 35 beats.min^{-1}, which was a greater variation than the beat to beat fluctuations. VA blood flow was continuous throughout the cardiac cycle at resting heart rates (Fig. 1).

The effects of adrenergic and cholinergic drug infusions

To assess the time delay between a pre-gill drug infusion and direct cardiac stimulation by the infused drug, pre-gill CARB infusions were made (n = 5; 5 fish). CARB is a synthetic cholinergic agonist that will stimulate cardiac cholinoceptors and thereby reduce heart rate. 90 to 120 s after a CARB infusion the heart rate was only one or two beats slower. A minimum heart rate was subsequently attained 3 min after the infusion. Based on these findings it seems unlikely that *peak* cardiovascular changes occurring in less than 100 s following a pre-gill drug infusion (t < 100 s) will be a result of direct agonist effects on the heart. The peak responses to ACh and NA infusions are described below, and frequently occurred when t < 60 s and always before t = 100 s.

The peak cardiovascular changes following a pre-gill ACh infusion are summarized in Table 1. There was always (n = 11; 8 fish) an increase in Rg. (Rg was also increased after CARB infusions.) Since $\dot{Q}$ was also elevated after an ACh infusion, the increased Rg must reflect cholinergic vasoactivity.

Generally a marked pressor response followed the ACh infusion (Fig. 3B), but in two experiments similar ACh dosages evoked only a weak pressor response (Fig. 3A). The elevated VA pressure was primarily a result of the branchial vasoconstriction. The increased $\dot{Q}$ also contributed to raise arterial pressures, especially the DA pressure since Rs was unchanged.

In nine experiments $\dot{Q}$ was increased by 15 to 50%. In two experiments $\dot{Q}$ was reduced by 10%. Increased $\dot{Q}$ was largely brought about by changes in stroke volume

Table 1. *Cardiovascular variables in the unanaesthetized lingcod at 10 °C. Resting values and the results for pre-gill infusions of acetylcholine (ACh), noradrenaline (NA), and isoproterenol (ISOP) are presented*

		ACh		NA		ISOP	
	Resting (n = 103)	Rest (n = 30)	Peak (n = 11)	Rest (n = 22)	Peak (n = 12)	Rest (n = 13)	t < 100s (n = 12)
Cardiac Output $ml.min^{-1}.kg^{-1}$	10·9 ± 0·2	11·5 ± 0·1	12·7 ± 0·6*	10·7 ± 0·4	15·2 ± 1·1*	12·0 ± 0·4	12·9 ± 0·5
Stroke volume $ml.kg^{-1}$	0·37 ± 0·01	0·40 ± 0·02	0·44 ± 0·02*	0·36 ± 0·01	0·48 ± 0·04*	0·40 ± 0·02	0·42 ± 0·02
Heart rate (beats.min^{-1})	29·8 ± 0·4	29·1 ± 0·7	29·1 ± 1·2	30·6 ± 1·0	32·9 ± 2·2	30·4 ± 0·8	31·5 ± 1·1
Gill resistance (cm H_2O.min.ml^{-1})	0·253 ± 0·009	0·244 ± 0·015	0·374 ± 0·052*	0·255 ± 0·090	0·192 ± 0·020*	0·212 ± 0·018	0·168 ± 0·019*
Systemic resistance (cm H_2O.min.ml^{-1})	0·788 ± 0·002	0·732 ± 0·060	0·717 ± 0·084	0·841 ± 0·087	0·915 ± 0·131	0·643 ± 0·016	0·563 ± 0·038*
Ventral aortic pressure (cm H_2O)							
Mean	52·6 ± 0·4	52·7 ± 0·6	63·7 ± 2·0*	52·6 ± 0·8	74·7 ± 2·6*	49·3 ± 0·6	46·8 ± 1·2*
Pulse	12·4 ± 0·2	12·4 ± 0·4	22·4 ± 2·3*	12·2 ± 0·5	24·3 ± 1·8*	13·0 ± 0·5	14·6 ± 1·0
Dorsal aortic pressure (cm H_2O)							
Mean	39·6 ± 0·3	39·7 ± 0·6	42·3 ± 1·4*	39·4 ± 0·8	60·9 ± 2·0*	37·7 ± 0·8	34·7 ± 0·9*
Pulse	6·0 ± 0·2	6·0 ± 0·3	7·9 ± 0·6	6·1 ± 0·4	12·5 ± 1·2*	6·4 ± 0·4	6·2 ± 0·6

All values are $\bar{x} \pm$ S.E. for n observations. Resting values were taken from eight fish. Eleven ACh experiments were performed on eight fish. Twelve NA experiments were performed on eight fish. Six ISOP experiments were performed on five fish.

* Denotes a statistically significant difference between the peak value (or t < 100 s) and the resting value recorded immediately before the drug infusion (Rest).

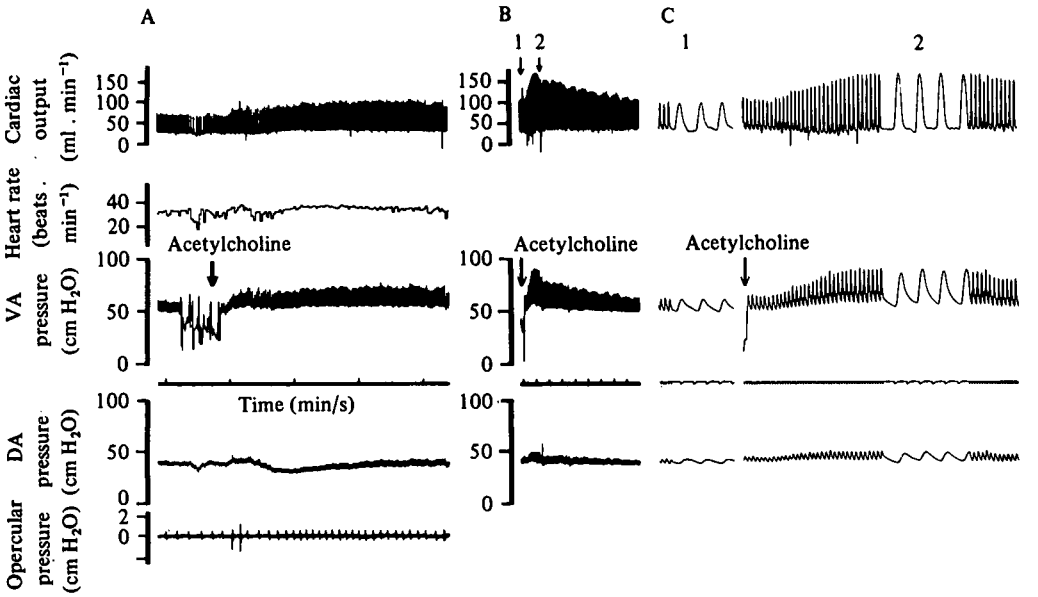

Fig. 3. Cardiovascular changes to pre-gill ACh infusions (0·1 μg.ml blood^{-1}) in a resting 4·5 kg lingcod. (A) illustrates a weak response to the infusion. (B) illustrates the more typical effects of ACh. This record was produced from stored information on magnetic tape, which accounts for the absence of beat to beat heart rate and ventilation pressure traces. (C) presents a more detailed view of (B) using an expanded time scale and compares resting (1) with peak changes (2). It is clear from this record that the changes in VA blood pressure and $\dot{Q}$ closely parallel each other. There is no change in heart rate. Furthermore changes in $\dot{Q}$ are noticeable eight heart beats after the ACh infusion was complete.

(Table 1; Fig. 3 B, part 2). The change in stroke volume markedly altered the VA flow velocities and pulse pressure during the peak response (compare parts 1 and 2 of Fig 3 B). Notably the VA flow velocity during the diastolic period remained relatively stable, in contrast to its gradual fall in the resting fish (see Fig. 1 also). Overall the heart rate was unchanged during the peak ACh response. Furthermore there was no subsequent (t > 100 s) bradycardia as was the case with CARB infusions. This result indicates that the ACh concentration reaching the heart may have been reduced by metabolism (unlike CARB) and was too low to stimulate the cardiac receptors.

The above results indicate that a pre-gill ACh infusion evoked gill vasoactivity and that there were considerable modifications of the pressure and flow profiles across the gill.

Peak cardiovascular changes following a pre-gill NA infusion are summarized in Table 1. There was always (n = 12; 8 fish) a reduction in Rg of between 17 and 49%. At the same time $\dot{Q}$ was increased by 9 to 56%. Thus the peak R_g value reflects combined active and passive changes in the gill vasculature. Inhibitory β-adrenoceptors are likely to have produced the active vasodilation, since the presence of β-adrenoceptors in the lingcod branchial vasculature has been established (A. P. Farrell & D. J. Smith, in preparation). This interpretation is also consistent with the reduced Rg following ISOP infusion (see below).

Even though R_g fell, both arterial pressures were elevated markedly (Fig. 4;

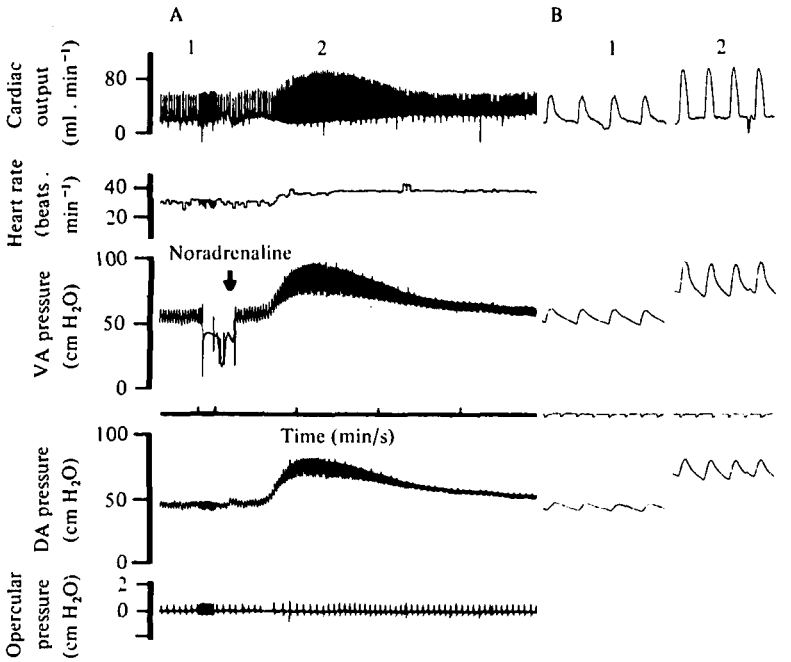

Fig. 4. (A) A typical response to a pre-gill NA infusion 0·1 μg.ml blood^{-1}) in a resting 4 kg lingcod. Note that although heart rate is increased, it remains elevated for several minutes after the peak response. This change was not typical of all NA infusions. A respiratory pause is also associated with the rise in $\dot{Q}$ and arterial blood pressures. The same phenomenon was seen in some other fish after a pre-gill NA infusion and cannot be explained. (B) illustrates the marked changes in the flow and pressure profiles between rest (1) and the peak (2) response to NA.

Table 1). In 8 of the 12 experiments R_s was increased up to 30% during the peak response. R_s was unchanged in the remaining 4 experiments. Even so the mean change in R_s (Table 1) was not significant when comparing absolute R_s values. The lack of significance is largely due to the very variable basal R_s values for different fish, as is demonstrated by using a paired analysis test. With this test the mean R_s increased by $0{\cdot}122 \pm 0{\cdot}035$ during the peak response, and this increase is significant at a 95% confidence level. The pressor response to a NA infusion was therefore due to increases in R_s and in $\dot{Q}$.

$\dot{Q}$ was increased largely through changes in stroke volume (Table 1). Heart rate did, however, change in some individual fish, but not with any consistent pattern. Fig. 4 illustrates an experiment where heart rate was increased. Here the heart rate remained elevated for several minutes after the peak blood pressure and flow. Thus the rise, but not the fall, in heart rate was associated with the pressure and flow changes, and even though heart rate was still elevated, $\dot{Q}$ had returned to a resting value. It seems therefore that the change in stroke volume is more closely associated with the pressure and flow changes accompanying vasoactivity. Increased stroke volume also had marked effects on the VA flow velocities and arterial pulse pressures (Fig. 4B, parts 1 and 2). Notably the VA flow velocity during diastole remained relatively stable, which was not unlike that seen during the peak response to ACh (Fig. 3).

In order to assess the importance of the α- and β-adrenergic components of the NA

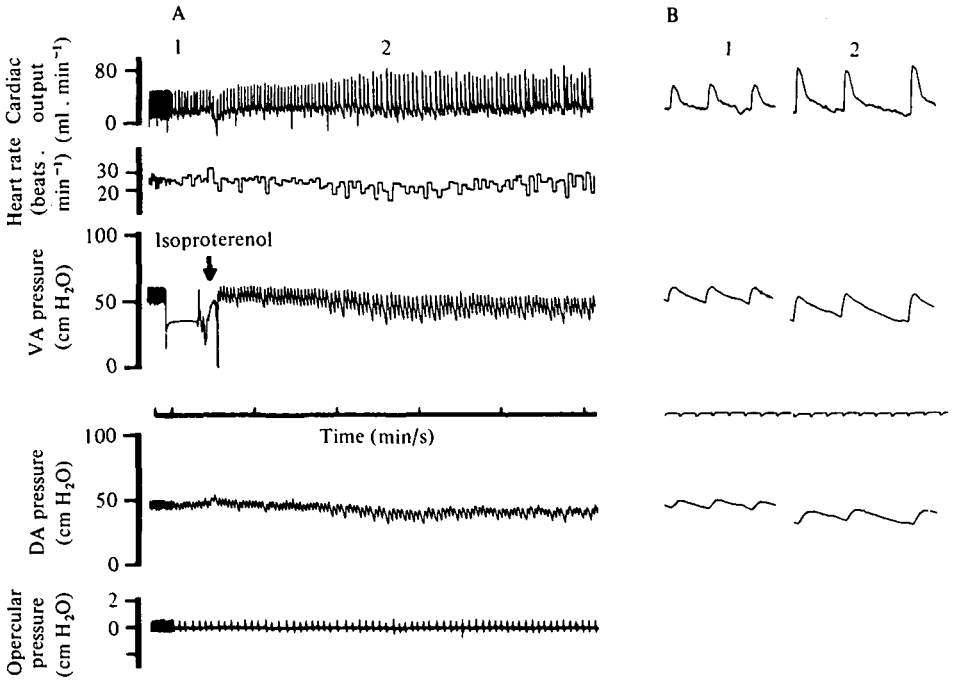

Fig. 5. (A) the effect of a pre-gill ISOP infusion (0·01 μg.ml blood^{-1}) in a resting 4 kg lingcod. (B) illustrates the differences between resting (part 1) and 100 s after the infusion (part 2) using an expanded time scale.

response, pre-gill infusions of the β-agonist ISOP were made (n = 6; 5 fish). A peak response was not obvious (Fig. 5) so group mean values for t < 100 s after the infusion are presented (Table 1). Pre-gill ISOP infusion reduced R_g and R_s significantly. In three experiments there were small increases in $\dot{Q}$, but the mean $\dot{Q}$ value (Table 1) was not significantly different from the pre-infusion value. The changes in vascular resistance were thus more a consequence of inhibitory β-adrenoceptors than passive changes due to flow. The reduction in vascular resistance reduced arterial blood pressures significantly. These results clearly contrast with those for a NA infusion and therefore emphasize the importance of the systemic α-vasoconstriction (or even a lack of vasodilation) in evoking a pressor response and an accompanying increase in $\dot{Q}$.

DISCUSSION

A detailed examination of cardiovascular variables pertaining to gill blood flow in *O. elongatus* has been presented. To make simultaneous and direct measurements, the fish underwent considerable surgery. Nevertheless, all indications are that the fish were not stressed during the experimental protocol. As in their natural environment, resting fish in the experimental aquarium had low ventilation rates, and the buccal and opercular pressures associated with gill ventilation (Fig. 2) were small. During the study increased ventilation rates and pressures (and reduced $\dot{Q}$ or high heart rates) compared to resting values were observed in obviously stressed fish.

Blood flow has been previously measured in *O. elongatus* using smaller specimens (1–4 kg) and a higher water temperature (13 °C) then the present study (Stevens *et al.* 1972). Compared to the present study, $\dot{Q}$ was lower (5·9 ml.min^{-1}.kg^{-1} compared to 10·8), and heart and ventilation rates were higher (47 beats.min^{-1} compared to 29·8, and 26·8 breaths.min^{-1} compared to 11·9).* Despite the above differences between the two studies on *O. elongatus* the ratios of systolic to diastolic blood flow a similarity (34:66 compared to 40:60 in the present study). In fish more active than in *O. elongatus*, $\dot{Q}$ is higher. For instance in trout, $\dot{Q}$ is about 20 ml.min.kg^{-1} (Randall, Holeton & Stevens, 1967; Kiceniuk & Jones, 1977) and in *Gadus morhua*, $\dot{Q}$ is 52 ml.min^{-1} (n = 5 fish, wt = 2–3 kg; Jones *et al.* 1974).

It is likely that R_g is increased in teleosts by a neural mechanism rather than a humoral one since ACh is quickly metabolized in the blood. Indeed, branchial parasympathetic vasoconstriction has been demonstrated in *Gadus morhua* (Pettersson & Nilsson, 1979). Experimental data from several teleost species (Dunel & Laurent, 1977; Smith, 1977), including the lingcod (A. P. Farrell & D. J. Smith, in preparation), indicate that a cholinergic vasoconstriction site is probably located at the base of the efferent filament arteries, where nerve terminals have recently been observed (P. Laurent, unpublished). Based on this information a pre-gill ACh infusion probably mimics branchial parasympathetic activity and produces an efferent vasoconstriction. If so, then the elevated VA pressure following a pre-gill ACh infusion also reflects an increase in the lamellar input pressure and in the lamellar transmural pressure. Consequently, cholinergic branchial vasoactivity is accompanied by passive changes in lamellar perfusion because of the elevated blood pressures (and cardiac output).

In trout, β-adrenoceptors dominate over α-adrenoceptors in the branchial circulation (Wood, 1974), while the opposite is true for the systemic circulation (Wood & Shelton, 1975). The present study indicates that branchial β-adrenoceptors dominate over any branchial α-adrenoceptors in the lingcod. There was a decrease in R_g after NA and ISOP infusions. With respect to the systemic vasculature in the lingcod, the adrenergically mediated vasoactivity evoked by NA was variable, as was the basal Rs value for different fish. Nevertheless, the small but significant reduction in R_s is consistent with systemic inhibitory β-adrenoceptors. However, systemic excitatory α-adrenoceptors dominate over these β-adrenoceptors since R_s was never reduced following a NA infusion. The reason why Rs was not increased in 4 of the NA experiments is probably because several accompanying events tended to oppose any increase in R_s. These could be (*a*) the metabolism or adsorption of the catecholamine as the blood passes through the gills, (*b*) a counteracting passive vasodilation as the flow increases, and (*c*) the weak β-inhibition. In summary, the adrenoceptors in the lingcod appear similar to those in the trout. A pre-gill Na infusion reduces R_g and raises R_s by changing the vascular tone. Thus the accompanying pressor response is a result of a systemic vasoconstriction and an increase in $\dot{Q}$. Without the systemic vasoconstriction there is a fall in blood pressure and little or no change in $\dot{Q}$.

Low levels of catecholamines are normally found in the blood of teleosts. During stress or exercise these levels rise to concentrations which can alter vascular tone

* The maximum ventilation rate attained during hypoxic exposure of lingcod at 10 °C was 33 breaths.min^{-1} (Farrell, 1979).

(Nakono & Tomlinson, 1967; Nilsson, Abrahamson & Grove, 1976). Thus a pre-gill NA infusion probably mimics catecholamine release in its effects on the branchial and systemic vasculature, but not on the heart. (Catecholamines released into the blood from the chromaffin tissue would pass through the heart before reaching the branchial circulation.) Circulating catecholamines are likely to decrease R_g and raise R_s. Pre- and post-branchial blood pressures will be elevated, as must the lamellar input pressure and transmural pressure. Consequently, in addition to changing the branchial and systemic vascular tone, catecholamine release will produce passive changes in lamellar perfusion because of the pressure and flow changes.

The changes in $\dot{Q}$ accompanying pre-gill NA and ACh drug infusions are unlikely to be a direct result of the agonist drug infusions. The increased $\dot{Q}$ following NA and ACh infusions could begin as soon as eight heart beats after the infusion. Furthermore, the increase tended to parallel that in blood pressure (Figs. 3, 4). Why changes in $\dot{Q}$ are so closely associated with increased vascular tone and what mechanisms are responsible are not clear. A reflex mechanism seems the most likely explanation, especially since cardiac reflexes can accompany vasoactivity in elasmobranchs (Lutz & Wyman, 1932; Satchell, 1962) and a reflex branchio-cardiac coupling has been argued for (Satchell, Hanson & Johansen, 1970). The receptors involved with such a reflex in the lingcod might be baroreceptors. ACh and NA infusions produced branchial and systemic vasoconstrictions, respectively. The vasoconstriction resulted in elevated blood pressures, which in turn could stimulate baroreceptors. Why does $\dot{Q}$ increase since this should only further raise the blood pressure? The answer may lie in the changes in branchial perfusion that are brought about when vascular tone and blood pressure are increased as a result of catecholamine release or branchial parasympathetic activity. These changes will no doubt distend the compliant blood vessels and recruit more lamellae, and thereby reduce vascular resistance and blood pressure if there are no other changes. An increase in volumetric flow through the gills would therefore achieve two things. Firstly, blood pressure could remain elevated and thereby sustain any changes in lamellar flow. Secondly, it would help stabilize the blood transit time through the gills. Future experiments demonstrating baroreceptors in teleosts and measuring the blood transit time in the gill would help to subtantiate this hypothesis.

In conclusion, this study has reported the peak cardiovascular events that accompany adrenergic and cholinergic alterations in vascular tone. It demonstrates that important changes in blood pressure and flow accompany branchial and systemic vasoactivity. It appears that alterations in vascular tone, pressure and flow profiles, and cardiac output all contribute in a complex fashion to change branchial perfusion. Lastly, the mechanism behind the possibly reflex cardiac changes accompanying vasoactivity clearly needs further investigation.

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