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BLOOD PRESSURE CONTROL DURING EXERCISE IN THE ATLANTIC COD, *GADUS MORHUA*

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SUMMARY

Atlantic cod were subjected to 12–15 min swimming exercise at 2/3 body lengths s^{-1} in a Blazka-type swim tunnel. Pre- and postbranchial blood pressures, cardiac output (ventral aortic blood flow) and heart rate were continuously recorded, and blood samples for measurement of arterial and mixed venous oxygen tension were taken before and at the end of the exercise period. In a second group of fish, subjected to similar exercise regimes, blood samples were taken for analysis of the plasma concentrations of catecholamines.

Pre- and postbranchial blood pressures and cardiac output increase during exercise, while the mixed venous oxygen tension decreases. The effect on cardiac output is due to an increase of both heart rate and stroke volume. There are no significant changes in either systemic or branchial vascular resistances, or in the plasma concentrations of catecholamines.

Injection of the adrenergic neurone-blocking drug bretylium produces a decrease in postbranchial blood pressure in resting cod, due to a decrease in the systemic vascular resistance. Exercising cod treated with bretylium have a significantly lower pre- and postbranchial blood pressure than exercising control cod. This is due mainly to a dramatic reduction in the systemic vascular resistance. The α-adrenoceptor antagonist phentolamine does not further affect the blood pressure in cod treated with bretylium.

It is concluded that the exercise hypertension observed in cod depends on the effect of adrenergic vasomotor fibres maintaining the systemic vascular resistance, and also on the increase in cardiac output. An adrenergic innervation of the heart may play some role in the control of cardiac performance both at rest and during exercise, but the main cardioregulatory mechanism is likely to be non-adrenergic, most probably including cardiac control *via* variation of the cholinergic vagal cardioinhibitory tonus.

INTRODUCTION

The effects of exercise (swimming) on cardiovascular and respiratory parameters in teleost and elasmobranch fish have been described by numerous authors (for references see, e.g., Jones & Randall, 1978; Randall & Daxboeck, 1982; Nilsson &

Key words: *Gadus morhua*, teleost fish, adrenergic control, circulatory system, blood pressure, exercise.

Axelsson, 1986; Butler, 1986). The comprehensive study by Kiceniuk & Jones (1977) of the rainbow trout (*Salmo gairdneri*) can be used to illustrate typical cardiovascular changes associated with exercise in teleosts. During swimming near the critical swimming speed, U_{crit} (for definition of U_{crit}, see, e.g., Beamish, 1978), in a Brett-type water tunnel (Brett, 1964), there is a three-fold increase in cardiac output due to an increase in heart rate (1·36 times) and stroke volume (2·24 times). The branchial vascular resistance changes little, while the systemic vascular resistance drops substantially during exercise. Both ventral and dorsal aortic blood pressures increase, the former more than the latter as a consequence of the relative branchial/systemic vascular resistance changes. With increasing swimming velocity, there is an increase in the difference between arterial and venous oxygen content, and at swimming speeds near U_{crit} the oxygen transport capacity of the rainbow trout circulatory system increases to 7·6 times that at rest (Kiceniuk & Jones, 1977).

The control systems underlying the cardiovascular effects of exercise have been relatively little studied, and the role of nervous and humoral mechanisms in cardiovascular control is not clear. At rest, the systemic blood pressure in the cod (*Gadus morhua*) is influenced by an adrenergic tonus which appears to be due solely to adrenergic nerves innervating the vasculature (Smith, Nilsson, Wahlqvist & Eriksson, 1985).

In the study of Smith *et al.* (1985), the adrenergic neurone-blocking agent bretylium was used, and the ability of this drug to abolish selectively the influence of adrenergic nerves was established. The effect of bretylium was related to that of the α-adrenoceptor antagonist phentolamine, which was shown to impair the α-adrenoceptor effects of both adrenergic nerves and humoral catecholamines. In the present work a similar experimental protocol, using bretylium and phentolamine, was adopted to elucidate the role of adrenergic mechanisms in the control of the cardiovascular system of the cod during exercise.

MATERIALS AND METHODS

Fifteen Atlantic cod, *Gadus morhua*, of either sex and with a body weight of 400–800 g, and body length of 37–41 cm were used in this study. The fish were kept in well-aerated, recirculating sea water at 10°C, and either used within 2 weeks of capture, or fed until 1 week before surgery. Animals with gill parasites (*Laciocarpa branchialis*) were not used. The study was performed from November to May.

Surgical procedure

Fish in one group ($N = 8$) were anaesthetized in MS 222 (tricaine methanesulphonate; 100 mg l^{-1}) until breathing movements ceased. The animal was then transferred to an operating table, and aerated sea water containing the anaesthetic (50 mg $^{-1}$) was passed over the gills throughout the operation. A catheter (PE 50) was inserted into the afferent branchial artery of the second gill arch for measurement of ventral aortic blood pressure (PVA) and sampling of blood (for determination of mixed venous oxygen tension, Pv_{O_2}). The catheter was passed through the upper

part of the operculum and secured with a skin suture. A second catheter (PE 50) was implanted into the efferent branchial artery of the same gill arch and secured in a similar way. This catheter was used for measurement of dorsal aortic blood pressure (P_{DA}) and blood sampling (efferent arterial oxygen tension Pa_{O_2}). Both catheters were filled with heparinized (50 i.u. ml^{-1}) 0·9 % NaCl. During the experiments the catheters were attached to Statham P23 pressure transducers connected to a Grass polygraph recorder system model 7D. Calibration of pressure was made against a static water column.

The animal was then placed on its back and the ventral aorta exposed through an incision just anterior to the base of the pectoral fins. The ventral aorta was freed for a length of 4–7 mm without rupturing the pericardium, and a non-cannulating electronic flow probe (Biotronex BLI) was placed around it for recording of cardiac output (= ventral aortic blood flow) on the same Grass polygraph system *via* the Biotronex BLI unit. To obtain zero blood flow for calibration of the system, the fish was injected with acetylcholine (100 nmol kg^{-1} fish) (Jones, Langille, Randall & Shelton, 1974). The flow probe was calibrated after every third experiment by perfusing isolated segments of cod ventral aortas with 0·9 % NaCl solution at known flow rates.

Fish in a second group ($N = 7$) were equipped with a catheter in an afferent branchial artery only, to allow sampling of blood for analyses of plasma catecholamine concentrations.

Water channel

After surgery the fish were transferred to a water channel, modified after Blazka, Volf & Cepela (1960) (see Fig. 1), where they recovered rapidly from the anaesthesia. Water velocity in the water channel was monitored throughout the experiment using an impellor (Braystoke BFM.002, Valeport Development Ltd, UK, sensitivity 0·04 cm s^{-1}) situated in the centre of the water flow. Velocity profiles at different levels within the water channel were checked using a pressure transducer and injections of dye and, as far as could be judged, a microturbulent water flow with an approximately even pressure distribution across the tube was present at the water velocities employed in the present study. The pressure drop in the centre of the tube during the highest swimming speed used in this study did not exceed 0·15 kPa, and no correction for this difference was made. From the measurements made it was concluded that the water channel employed in the study could be used to provide readily reproducible swimming exercise.

The water of the channel was steadily replaced (2 l min^{-1}) from the departmental seawater system. The water temperature was 10–11 °C in all experiments.

Experimental protocol

Fish were allowed to recover in the water channel for 24 h before any experiment was started. During this time the effects of the anaesthesia and handling wore off (see Smith *et al.* 1985) and the cardiovascular parameters reached steady levels. Throughout the experiment care was taken to avoid any stressful stimuli which could

disturb the fish, thus ensuring, as far as possible, that the cardiovascular effects recorded were indeed due to the exercise and not due to unspecified 'stress'.

Each experimental run was started by recording the resting values of P_{VA}, P_{DA}, heart rate (HR) and cardiac output ($\dot{Q}$) and sampling of blood (0·2 ml from each catheter) for determination of Pa_{O_2} and Pv_{O_2}. The water flow through the swim tunnel was then started and adjusted to $2/3\,L\,s^{-1}$ (L = body length). In a preliminary study, this swimming velocity was shown to be within the range of sustained swimming speed in the cod, i.e. the maximal swimming speed the fish will sustain for more than 200 min (see Beamish, 1978). Almost every cod would swim at this speed without further provocation; fish that would not swim were discarded from the study. Estimations of U_{crit} (uncorrected for the presence of a fish in the channel; see discussion by Jones, Kiceniuk & Bamford, 1974) for cod using the same water channel give values of approximately $1{\cdot}2\,L\,s^{-1}$ (M. Axelsson, P. J. Butler, J. D. Metcalfe & S. Nilsson, in preparation). After 12–15 min exercise, new values of the

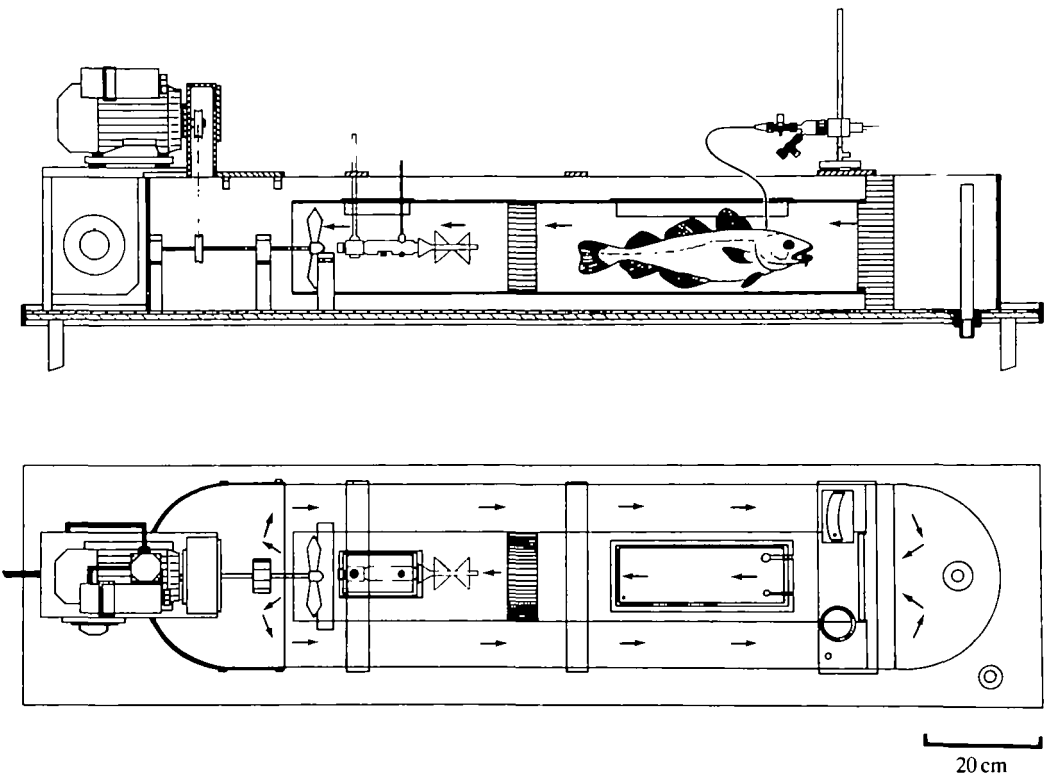

Fig. 1. Digram of the water channel used in the experiments. Upper diagram: side view; lower diagram: top view. The fish swims in a Perspex tube (i.d. 150 mm) in a current of water pulled through the tube and returning on the outside as shown by the arrows in the lower diagram. The water current is generated by a plastic propeller driven by a 0·37 kW speed-controlled motor, and the flow is continuously monitored by an impellor (Braystoke BFM.002) situated in the centre of the swim tube. Total capacity of the system is 130 l, and sea water at 10°C is continuously replenished ($2\,l\,min^{-1}$) from the departmental aquarium.

cardiovascular parameters were obtained and new blood samples were taken. It is likely that the U_{crit} after bretylium and phentolamine injections is lower than in the control situation, and that the swimming velocity employed is therefore closer to U_{crit} after injection of the drugs. Under the conditions of the exercise experiments, all fish could maintain a swimming velocity of $2/3\ L\ s^{-1}$ for an exercise period of 12–15 min, an exercise which is constant in absolute terms rather than in terms of U_{crit}.

After about 1 h recovery, bretylium tosylate ($10\ mg\ kg^{-1}$) was injected *via* the dorsal aortic catheter. The fish was left to recover for another 24 h, during which time the side effects of bretylium wore off, leaving a blockade of the adrenergic nerves only (see Smith *et al.* 1985). At this point another exercise run was performed, including recording of cardiovascular parameters and taking of blood samples before and after exercise as described above. Finally, phentolamine ($2\ mg\ kg^{-1}$) was injected and after 5 h recovery a final exercise run similar to the previous one was performed. In the study of Smith *et al.* (1985), it was demonstrated that bretylium, administered as described, produces blockade of adrenergic nerve functions without affecting the vascular responses to circulating catecholamines, and that phentolamine can be used to block both nervous and humoral catecholamine effects mediated by α-adrenoceptors.

The heart rate (HR) was derived from the pulsatile PVA signal *via* a Grass 7P44 tachograph unit. Mean blood pressure was determined as $(P_{systol}+2P_{diastol})/3$, and is expressed in kilopascals (kPa). Heart rate is expressed as beats min^{-1}, and cardiac output ($\dot{Q}$) and cardiac stroke volume (SV) as $ml\ min^{-1}\ kg^{-1}$. Relative vascular resistance of the branchial (VRG) and systemic (VRS) vascular beds was estimated from the blood pressure drop across the vascular bed divided by the blood flow through the same vascular bed (Pettersson & Nilsson, 1980). No compensation for the venous drainage of the gills, which produces a systemic blood flow that is lower than the ventral aortic flow, was made in the calculations. Furthermore, it is assumed that the central venous pressure (PCV) is close to zero and not dramatically affected by the exercise. This assumption is supported by the study of Kiceniuk & Jones (1977), who demonstrated that the PCV of the rainbow trout increased by only an insignificant amount during exercise at U_{crit}, and observed no change in the PCV at the intermediate water velocities. If there is a significant increase in PCV in the cod during exercise, then the exercise values given for VRS are overestimated.

Blood samples for catecholamine analyses were taken from fish subjected to exactly the same experimental protocol as described above. Blood samples (0·5 ml) from the ventral aortic catheter were taken before and after 15 min exercise under control conditions, 24 h after bretylium injection and, finally, 5 h after injection of phentolamine as described.

Analyses of blood samples

Oxygen tension in the blood samples was determined using a Radiometer system (PHM 73).

Plasma catecholamine levels were estimated by the radio-enzymatic method of Peuler & Johnson (1977) as described previously (Smith *et al.* 1985).

Drugs used

The following drugs were used in the study: bretylium tosylate (a generous gift from the Wellcome Foundation Ltd) and phentolamine methanesulphonate (Sigma). The drugs were dissolved in 0·9 % NaCl.

Statistical evaluation

Statistical evaluation of the results was made using the Friedman two-way analysis of variance, and the Walsh test (Siegel, 1956); both were used as one-tailed tests. Differences where $P \leq 0{\cdot}05$ were considered significant.

RESULTS

Resting values of the cardiovascular parameters from the eight cod studied (Table 1) compare well with those obtained in previous studies of the cod (e.g. Pettersson & Nilsson, 1980; Smith *et al.* 1985). Although the resting PVA and PDA values recorded were very stable within one animal, there were great differences between individuals (range in resting animals: PVA, 3·0–9·0 kPa; PDA, 2·2–5·0 kPa).

During exercise, there was a statistically significant ($P < 0{\cdot}05$) increase in PVA, PDA, HR and $\dot{Q}$ and a decrease in Pv_{O_2} (Table 1; Fig. 2). There was very little change in the vascular resistance of either the branchial or the systemic vascular bed, but there was an increase in cardiac output (1·46 times) which was caused by increases in both heart rate (1·19 times) and stroke volume (1·26 times).

Injection of bretylium caused a significant ($P < 0{\cdot}05$) decrease in PDA, VRS and HR, and an increase in stroke volume in resting cod 24 h after the injection (Table 1). There were no further changes following phentolamine injection (cf. Smith *et al.* 1985).

PVA, PDA , VRS, Pv_{O_2} ($P < 0{\cdot}05$) and HR ($P = 0{\cdot}05$) were all significantly lower in swimming bretylium-treated cod than in swimming control cod, and Pv_{O_2} was further reduced after phentolamine treatment.

Under control conditions, the exercise-induced changes in the cardiovascular parameters were rapidly restored to normal after cessation of the exercise (see Fig. 2). In the bretylium-treated fish and, especially, in the bretylium- and phentolamine-treated fish, however, there was a profound and long-lasting post-exercise hypertension. In addition the cardiac output, which increased slowly during exercise, remained at an elevated level for a similar time after cessation of the exercise (Fig. 2).

Analysis of plasma catecholamine levels showed a significant ($P < 0{\cdot}05$) decrease in the resting adrenaline concentration after bretylium treatment, and, in addition, there was a marked increase in the plasma level of adrenaline during swimming in cod treated with both bretylium and phentolamine (Fig. 3).

In the bretylium-treated cod, there was no increase in PVA during swimming compared to the resting value, and the change in PDA was even reversed to a decrease during swimming (Fig. 2; Table 1). This decrease in PDA is associated with a marked decrease in VRS ($P < 0{\cdot}05$) in swimming, bretylium-treated cod (Table 1).

Table 1. *A summary of simultaneously recorded and calculated cardiovascular parameters from Atlantic cod (*Gadus morhua*) during rest (upper part of Table) and swimming exercise (lower part of Table)*

	N	Control	Bretylium	Phentolamine
Resting values ± S.E.M.				
P_{VA} (kPa)	8	4·9 ± 0·6	4·7 ± 0·6	3·7 ± 0·4
P_{DA} (kPa)	8	3·2 ± 0·4	2·6 ± 0·2*	2·4 ± 0·2
HR (beats min^{-1})	8	43·2 ± 1·8	40·2 ± 1·5*	43·6 ± 2·9
Pv_{O_2} (mmHg)	7	41·3 ± 8·0	39·0 ± 7·3	41·8 ± 7·1
Pa_{O_2} (mmHg)	7	77·3 ± 6·7	78·0 ± 7·6	78·9 ± 7·8
$\dot{Q}$ (ml min^{-1} kg^{-1})	8	17·3 ± 1·0	18·8 ± 1·4	18·8 ± 1·4
V_{RG} (Pa min^{-1} kg^{-1} ml^{-1})	8	99 ± 25	110 ± 29	97 ± 34
V_{RS} (Pa min^{-1} kg^{-1} ml^{-1})	8	188 ± 27	129 ± 11*	137 ± 21
SV (ml min^{-1} kg^{-1})	8	0·39 ± 0·03	0·46 ± 0·03*	0·44 ± 0·04
Swimming values ± S.E.M.				
P_{VA} (kPa)	8	6·2 ± 0·7	4·5 ± 0·8*	3·8 ± 0·8
P_{DA} (kPa)	8	4·0 ± 0·4	2·0 ± 0·3*	1·6 ± 0·2
HR (beats min^{-1})	8	51·2 ± 1·7	48·5 ± 2·3*	49·1 ± 2·3
Pv_{O_2} (mmHg)	7	27·5 ± 2·3	22·3 ± 1·2*	20·2 ± 0·8*
Pa_{O_2} (mmHg)	7	82·2 ± 7·1	79·8 ± 9·4	67·9 ± 7·6
$\dot{Q}$ (ml min^{-1} kg^{-1})	8	25·4 ± 2·4	26·1 ± 3·4	28·9 ± 3·9
V_{RG} (Pa min^{-1} kg^{-1} ml^{-1})	8	92 ± 27	97 ± 25	100 ± 42
V_{RS} (Pa min^{-1} kg^{-1} ml^{-1})	8	174 ± 30	78 ± 17*	60 ± 13
SV (ml min^{-1} kg^{-1})	8	0·49 ± 0·05	0·55 ± 0·08	0·60 ± 0·09

The figures show ventral aortic blood pressure (P_{VA}), dorsal aortic blood pressure (P_{DA}), heart rate (HR), mixed venous (ventral aortic) oxygen tension (Pv_{O_2}), efferent arterial (dorsal aortic) oxygen tension (Pa_{O_2}), cardiac output (ventral aortic blood flow) ($\dot{Q}$), branchial vascular resistance (V_{RG}), systemic vascular resistance (V_{RS}) and cardiac stroke volume (SV).

Control refers to untreated animals, Bretylium to the same animals 24 h after injection of bretylium (10 mg kg^{-1}) and Phentolamine to the same animals 5 h after a further injection of phentolamine (2 mg kg^{-1}).

Asterisks indicate significant differences compared to control values ($P \leq 0{\cdot}05$). *N* indicates number of animals.

DISCUSSION

Values for the different cardiovascular parameters studied are comparable to those previously recorded in resting cod (Pettersson & Nilsson, 1980; Smith *et al.* 1985), and where slight differences occur (cf. Pettersson & Nilsson, 1980) they may be due to differences in the experimental equipment and procedure, for example the comparatively long post-surgical recovery period (>24 h) allowed in the present experiments.

As in the study by Smith *et al.* (1985), injection of bretylium produced a significant decrease in the P_{DA} of resting cod. The effect is not enhanced by the α-adrenoceptor antagonist phentolamine, and has been attributed to a blockade by bretylium of the adrenergic vasomotor innervation of the systemic vascular beds of the cod (Smith *et al.* 1985). In the study by Smith *et al.* (1985), no measurement of cardiac output was made, and it could only be assumed that the hypertensive

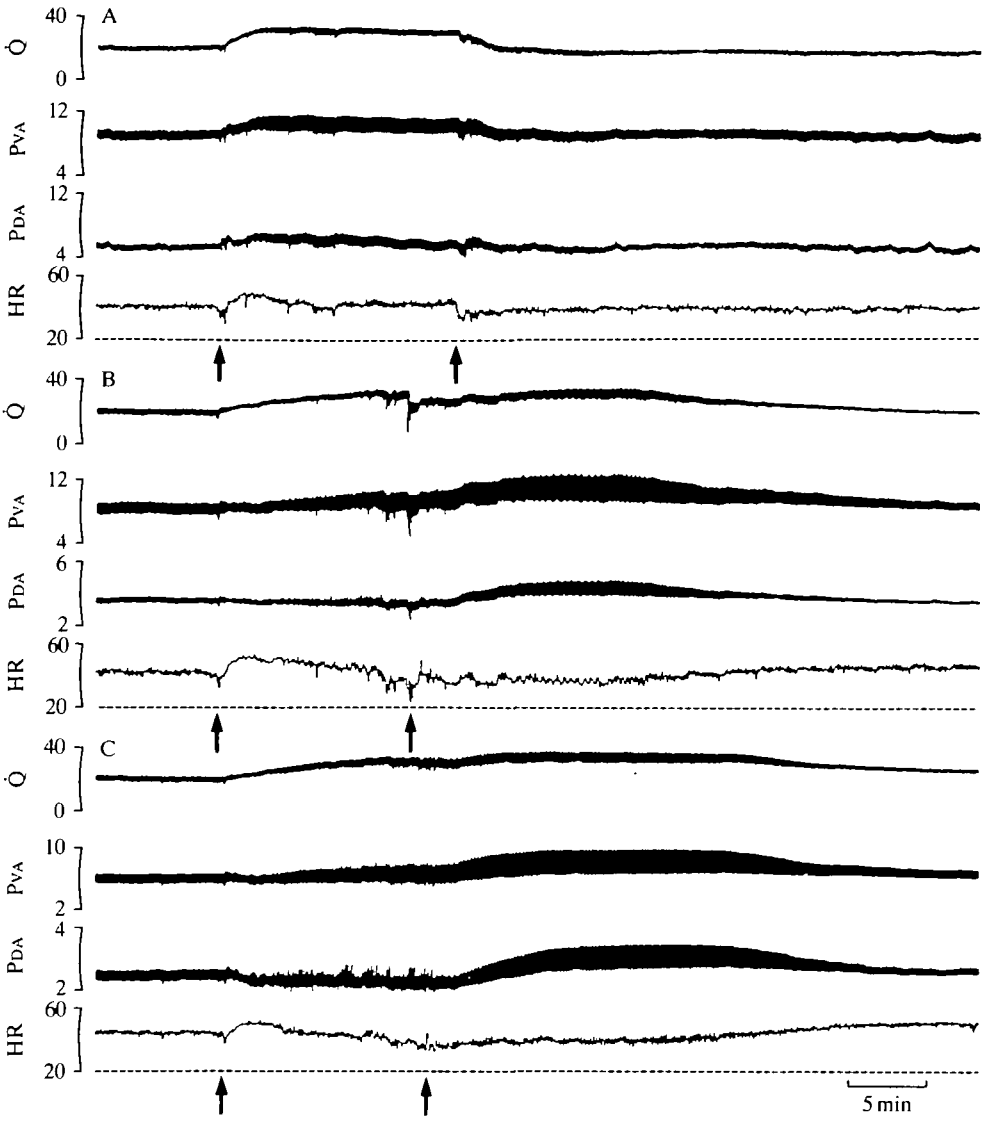

Fig. 2. Simultaneous recording of cardiac output (= mean ventral aortic blood flow) ($\dot{Q}$; ml min^{-1}), postbranchial (dorsal aortic) blood pressure (P_{DA}; kPa), prebranchial (ventral aortic) blood pressure (P_{VA}; kPa) and heart rate (HR; beats min^{-1}) in an exercising Atlantic cod (*Gadus morhua*). Between arrows, the water flow through the swim tunnel was started and adjusted to 2/3 body length s^{-1}. (A) Control. Note the increase in all four parameters in response to the swimming exercise, and the rapid return to pre-exercise levels at the end of the exercise period. (B) Bretylium. Injection with bretylium (10 mg kg^{-1} body weight) 24 h previously. Note slight reversal (compared to control conditions) of the P_{DA} response, the lack of increase in P_{VA} (apart from transient changes at the onset of swimming) and the increase in cardiac output and heart rate. (C) Bretylium + phentolamine. Additional injection of phentolamine (2 mg kg^{-1} body weight) 5 h previously. Note similarity with the bretylium recording (B), and the high post-exercise blood pressure and cardiac output.

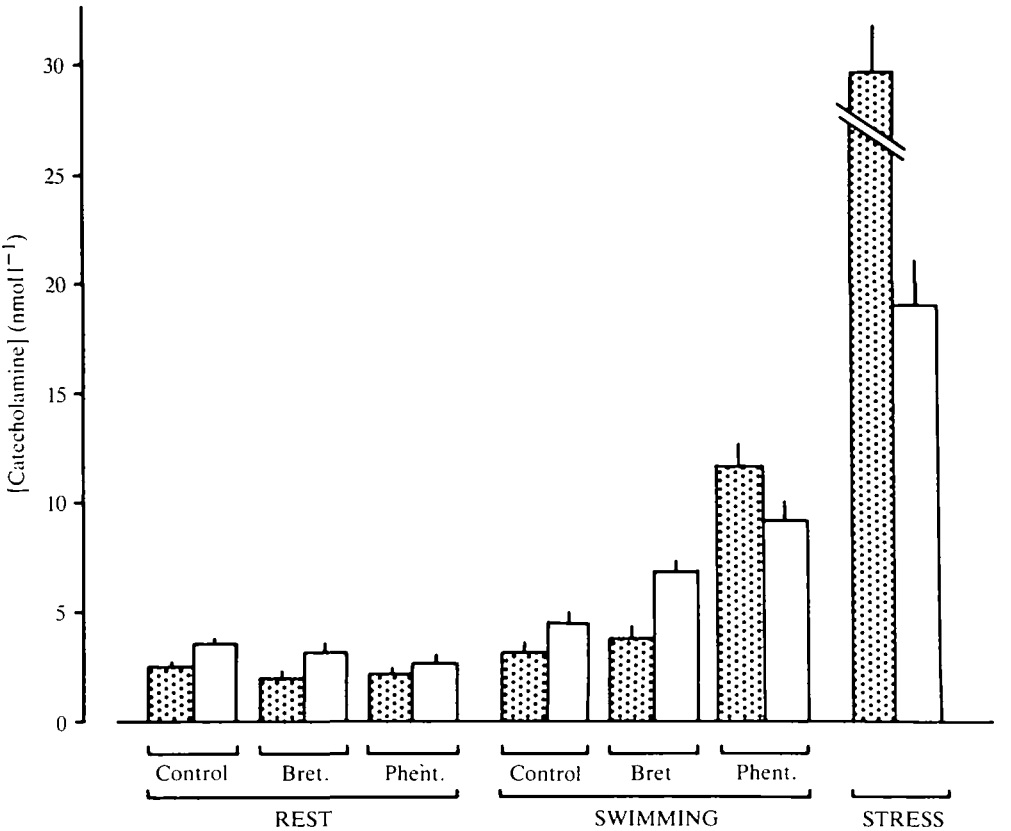

Fig. 3. Diagram showing plasma concentrations of adrenaline (stippled bars) and noradrenaline (plain bars) in resting, swimming and 'stressed' Atlantic cod (*Gadus morhua*) under control conditions, and after injections of bretylium (Bret.) and phentolamine (Phent.) as described in the text. There is a statistically significant decrease in the plasma concentration of adrenaline after bretylium treatment, and an increase in the adrenaline level in swimming cod after injection of bretylium + phentolamine. Values from a group of cod exposed to 'stress', induced by handling the fish within the swimming chamber, are included for comparison (from M. Axelsson, P. J. Butler, J. D. Metcalfe & S. Nilsson, unpublished data). Concentrations are expressed in nmol l^{-1}. Vertical bars show S.E.M.; $N = 7$ in all groups.

adrenergic tonus in resting cod was due to an effect on systemic vascular resistance. In the present study, an estimation of both the branchial and the systemic vascular resistance could be made, and it was shown that the resting P_{DA} is indeed influenced by an adrenergic nervous tonus affecting the systemic vascular resistance.

Under control conditions, there is a marked increase in both pre- and post-branchial blood pressure during exercise, but, contrary to the situation in the rainbow trout (Kiceniuk & Jones, 1977; Randall & Daxboeck, 1982), there is no decrease in the systemic vascular resistance in the cod. The increase in blood pressure can therefore, tentatively, be explained by the moderate increase in cardiac output, which, in turn, may be affected by an increase in the venous return of blood during exercise, and/or cardiac control by nerves and humoral factors.

The increase in pre- and postbranchial blood pressure associated with exercise is abolished by bretylium injection; the dorsal aortic blood pressure in fact decreases during exercise in bretylium-treated cod. The heart rate in both resting and exercising bretylium-treated cod is significantly lower than in exercising control fish, which may be interpreted in favour of an adrenergic chronotropic influence on the heart during both rest and exercise. The small reduction in heart rate is, however, compensated for by an increase in stroke volume in the bretylium-treated resting and exercising cod, leaving the cardiac output at the same level as in control fish. The effect on cardiac stroke volume may in part be due to a direct effect of bretylium on the myocardium, particularly the atrium, as shown in mammals (Markis & Koch-Weser, 1971).

The blockade (PVA) or even reversal (PDA) of the blood pressure increase during exercise caused by bretylium must be explained in terms of the loss of an adrenergic vasoconstrictor influence on the systemic vascular beds. The idea is supported by the very manifest reduction of systemic vascular resistance (VRS) observed in exercising bretylium-treated cod. It is possible that a compensatory vasoconstrictor influence (which is responsible for maintaining the systemic vascular resistance in control fish during exercise) is abolished by the bretylium treatment, unmasking the full effect of local vasodilatory mechanisms, for example the release of vasoactive metabolites ('active hyperaemia'; see Randall & Daxboeck, 1982). Since the effect of bretylium on blood pressure is not enhanced by the α-adrenoceptor antagonist phentolamine, it is concluded that the vasomotor tonus affecting VRS during exercise is due solely to adrenergic neurones and that circulating catecholamines play little or no role (cf. Smith *et al.* 1985).

The mechanism behind the dramatic post-exercise hypertension and elevated cardiac output seen in the fish treated with bretylium and with bretylium + phentolamine is not clear. Similar observations have been made in exercising toads (*Bufo marinus*), even after total autonomic blockade (bretylium + α- and β-adrenoceptor blockade + atropine), and it seems reasonable to postulate that the triggering of a non-adrenergic, non-cholinergic control system is responsible for the post-exercise cardiovascular effects (I. Wahlqvist & G. Campbell, personal communication). Further research into nervous and endocrine control systems, as well as possible local cardiac and vasomotor events, which are activated in the absence of the adrenergic and cholinergic control systems, is clearly called for.

As in the rainbow trout (Kiceniuk & Jones, 1977), there is a marked reduction in the mixed venous oxygen tension in the cod during exercise. The postbranchial oxygen tension, Pa_{O_2}, remains unchanged during swimming, and the cardiac output increases, which, quite reasonably, points to an increased oxygen consumption of the body muscle during exercise.

The plasma concentrations of catecholamines remain unchanged in control fish during the moderate exercise of the present study. This is remarkable in view of the general assumption that circulating catecholamines play an important role in cardiovascular control during exercise (e.g. Randall, 1982). However, similar experiments with rainbow trout have demonstrated that, in this species too, there is little or no

change in the plasma concentrations of catecholamines during exercise. Only during repeated burst swimming or 'stress' induced by handling of the fish is there any substantial increase in the levels of circulating catecholamines (Butler, Metcalfe & Ginley, 1986; Primmett, Randall, Mazeaud & Boutilier, 1986). The results thus emphasize the importance of differentiating between extended 'exercise' and various types of 'stress'.

Bretylium injection produces a small decrease in the resting plasma concentrations of catecholamines, and this effect may, to some extent, reflect a decrease in the overflow of catecholamines from adrenergic nerves which occurs during nervous activity. An increase in the plasma concentration of adrenaline is evident in exercising fish which have been injected with both bretylium and phentolamine. In these fish there exists no functional adrenergic innervation, nor can there be an adrenergic vasoconstrictor control *via* circulating catecholamines. The observed increase in the adrenaline level can be interpreted as a compensatory release of catecholamines from chromaffin cells, triggered by the marked drug-induced hypotension (see also discussion by Smith, 1978) or as a 'stress'-induced release due to the lowered blood oxygen tensions compared with control animals.

In conclusion, the present results suggest that the increase in both pre- and post-branchial blood pressure observed in the cod during exercise is due mainly to the effect of adrenergic vasomotor fibres maintaining the systemic vascular resistance, in combination with an increase in cardiac output. An adrenergic innervation of the heart may play some role in the control of the resting heart rate as well as in the tachycardia seen during exercise, but the main cardioregulatory mechanism is likely to be non-adrenergic, most probably regulating cardiac performance by controlling the cholinergic vagal cardio-inhibitory tonus. In addition, an increased venous return during exercise may affect the cardiac output. Catecholamine concentrations in blood plasma are not affected by the swimming exercise of the present experiments.

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