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CARDIOVASCULAR RESPONSES IN THE SEA RAVEN, *HEMITRIPTERUS AMERICANUS*, ELICITED BY VASCULAR COMPRESSION

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SUMMARY

Increases in the water pressure (Pw) around the trunk of the sea raven (*Hemitripterus americanus* Gmelin) were used to evaluate the effects of vascular compression on cardiovascular variables. Cardiac output ($\dot{V}b$), heart rate (fH) and blood pressures in the ventral aorta, dorsal aorta and ductus Cuvier (Pva, Pda and Pdc, respectively) were measured. A 20 cmH_2O increase in Pw decreased vascular conductance by up to 25%. During vascular compression, a reflex bradycardia reduced $\dot{V}b$ and attenuated the accompanying rise in arterial blood pressure. Pretreatment of the fish with the sympathetic antagonist, propranolol, further attenuated the hypertension by accentuating the reflex bradycardia. Subsequent pretreatment with papaverine, a vascular smooth muscle poison, potentiated these effects and did not reveal any autoregulatory vasodilation in the periphery. Atropine pretreatment completely abolished the reflex bradycardia, indicating that the bradycardia resulted from increased vagal cholinergic tone. The fish also exhibited cardiovascular compensation during the 2 min vascular compression. An accommodation of the barostatic reflex (reduced vagal tone) and a sympathetic tachycardia raised $\dot{V}b$ and passively increased vascular conductance. The set point for the barostatic bradycardia was apparently temperature-sensitive.

INTRODUCTION

It is generally accepted that swimming in teleost fish is accompanied by increases in cardiac output ($\dot{V}b$) and systemic vascular conductance such that there are much smaller increases in arterial blood pressure relative to the change in $\dot{V}b$ (see review by Jones & Randall, 1978). The increase in systemic vascular conductance is attributed to several events including the opening of resistance vessels by loss of excitatory cholinergic and/or α-adrenergic tone, passive distension through the increase in blood pressure, changes in vascular compliance and possible autoregulation of blood flow. However, the vascular compression, which must accompany undulatory swimming, will compromise any increase in systemic vascular conductance. During rhythmic exercise in mammals, for example, blood flow falls sharply as muscles

Key words: cardiac output, teleost, barostatic reflex, vascular compression.

contract and rises when they relax (Lamb, 1978). Sustained contractions at levels greater than 15% of the maximal voluntary contraction can reduce muscle blood flow, while levels above 70% can completely occlude blood flow to active muscles (Lind & McNicol, 1967). In the present study, an increase in water pressure around the trunk of a resting, intact fish was used to examine how blood flow is affected by vascular compression. Various components of the cardiovascular response were differentiated by pretreating the fish with propranolol, a non-specific β-adrenergic antagonist, papaverine, a vascular smooth muscle poison which prevents autoregulation, and atropine, a cholinergic antagonist.

MATERIALS AND METHODS

Sea ravens, *Hemitripterus americanus*, were caught by otter trawl in Passamoquody Bay off St Andrews, New Brunswick, and were held at ambient temperature (7–12°C) prior to the experiments. The sea raven was selected because the major blood vessels can be cannulated with relative ease.

The fish was placed in an experimental chamber which permitted the water pressure (Pw) to be raised around the trunk. Each fish was subject to a series of trials in which Pw was rapidly elevated (3–4 $cmH_2O\,s^{-1}$), maintained at a stable level for 2 min and then returned to 0 cmH_2O (Pw0). All fish were exposed sequentially to a Pw of 10, 15 and 20 cmH_2O (i.e. Pw10, Pw15 and Pw20, respectively) to assess the effects of various levels of vascular compression. There was a 1-h recovery between trials, which was longer if the cardiovascular variables had not stabilized. If a fish struggled during a trial, the trial was terminated and repeated when the cardiovascular variables had stabilized. The components of the cardiovascular responses were assessed by repeating the Pw20 trial three more times following antagonist drug pretreatment ($N = 14$ fish). D,L-propranolol HCl, papaverine HCl and atropine sulphate (all Sigma Chemicals) were administered sequentially as a bolus into the ventral or dorsal aorta to achieve a final blood concentration of about 10 $\mu mol\,l^{-1}$ (blood volume was estimated as 3%, and up to a 1 ml saline carrier volume was used). Following each antagonist infusion, a 30-min waiting period preceded the Pw20 trial to ensure receptor blockade. The cardiovascular status of each fish was monitored continually during the 4–8 h experimental period.

Surgical protocols

All fish were anaesthetized (0·5 $g\,l^{-1}$, ethyl *m*-aminobenzoate; Sigma) prior to surgery and were maintained in an anaesthetized state on an operating sling by irrigating the gills with ice-chilled sea water containing the anaesthetic (0·15 $g\,l^{-1}$). An ice pack was placed on the trunk of the fish during the procedure.

Two fish preparations were used. For simultaneous measurement of blood flow and pressure in the ventral aorta ($\dot{V}b$ and Pva) the ventral aorta was exposed by a midline incision through the skin of the isthmus and blunt dissection between the muscle. Care was taken not to damage the main trunk of the hypobranchial artery,

but minor side branches were individually tied off as necessary. A small incision was made in the pericardium overlying the aorta and a cuff-type flow probe implanted around the vessel. The flow probe had a snug fit without excessive constriction. The blood pressure cannula, polyethylene tubing (PE 60) tipped with a 21 gauge Huber needle, was inserted into the aorta towards the heart. Both cannulae were secured to the adjacent muscle and the incision was closed with separate silk sutures in the muscle and skin.

In other fish, blood pressures were measured in the dorsal aorta (Pda) and ductus Cuvier (Pdc). A 2-cm incision was first made in the skin of a fourth gill arch to expose the efferent branchial artery. A polyethylene (PE 90) cannula was inserted occlusively into the artery and advanced to the level of the dorsal aorta. The wound was closed around the cannula with silk sutures. A polyethylene cannula (PE 190), with a tapered tip to promote better sealing of the puncture hole, was introduced into the ductus Cuvier using a Medicut intravenous cannula (Argyle, St Louis, MO). The cannula was sutured to the body wall.

A head-restraining device was fitted following the cannulations. Initially, the device consisted of an adjustable brass clamp which clamped onto a bony prominence in front of the eyes. Later, a small brass bolt was inserted through a bore hole made in the bone in front of the eyes. The fish was quickly revived by irrigating the gills with fresh sea water, removing the ice-pack and transferring the fish to the experimental box where it began normal gill ventilation. The surgical procedures usually took from 30 to 45 min.

The experimental box (Fig. 1) was divided into a posterior chamber, to contain the trunk of the fish beyond the pectoral fins, and an anterior chamber, to contain the head of the fish including the heart. Each chamber received its own inflow of sea water at ambient temperature. A 0·6-cm thick neoprene rubber seal around the fish, immediately posterior to the pectoral fins, prevented water flow between the two chambers. The head-restraining device was attached to a rigid frame outside of the box and prevented excessive lateral and forward movements of the fish. Tail movements were also limited by the tapered interior of the posterior chamber. This arrangement prevented excessive movement into and out of the posterior chamber without completely immobilizing the fish. Environmental disturbances were kept to a minimum and very few fish struggled excessively after the 16 h (minimum) recovery and acclimation to the darkened Plexiglas box.

Under control conditions the water levels in the anterior and posterior chambers were the same. Because the posterior chamber had a sealed cover, incremental increases of the outflow pressure head elevated Pw on the trunk without altering the water pressure on the heart or head of the fish. The water level in the anterior chamber was used as a pressure reference (i.e. Pw0).

Analysis

Cardiovascular variables were sampled for beat-to-beat analysis (a) during the 30 s prior to each trial, (b) throughout the 2-min period of elevated Pw, and (c) for 60 s during the recovery. Systolic and diastolic arterial blood pressures were obtained

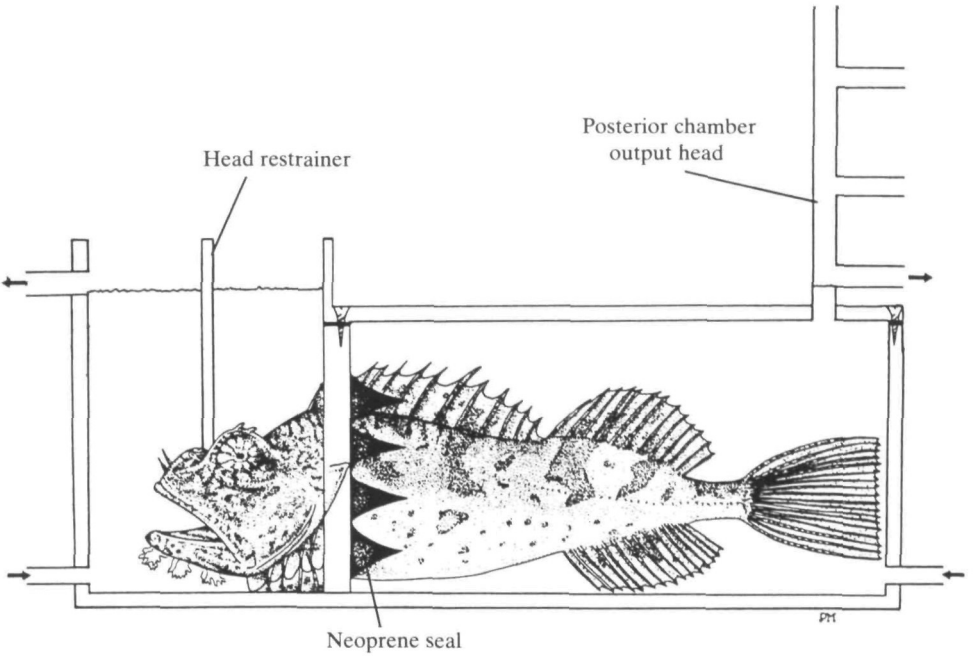

Fig. 1. A schematic diagram of the experimental chamber used to hold the sea raven and raise the water pressure around the trunk of the fish. The direction of water flow is indicated by the arrows.

from the chart recordings and mean pressure = [diastole + (pulse/3)], where pulse = (systole − diastole). Stroke volume (ml) and heart rate were determined from the area under the flow record and its periodicity, respectively. Cardiac output ($ml\,min^{-1}$) = (heart rate × stroke volume). Total vascular conductance ($ml\,min^{-1}\,cmH_2O^{-1}$) was calculated from ($\dot{V}b$/mean Pva). Stroke volume, vascular conductance and $\dot{V}b$ are expressed per kg body weight. The resting value is the average value for the 30-s pretrial beat-to-beat analysis.

Experiments were performed on 30 fish. One fish died overnight and data from six fish are not presented because of a loose-fitting flow probe and poor pressure recordings (two fish), excessive water leakage around the seal (one fish), excessive struggling (two fish) and a different experimental sequence to that described above (one fish). The cardiovascular data for 23 fish (body weight 1·9–3·9 kg; average 2·6 kg) is pooled into four groups. Groups 1 (average temperature, 7°C; $N = 6$) and 2 (average temperature, 8°C; $N = 3$) had ventral and dorsal aortic cannulae, respectively, and were tested with only the three increases in water pressure. Groups 3 (average temperature, 10·5°C; $N = 7$) and 4 (average temperature, 11°C; $N = 7$) had ventral and dorsal aortic cannulae, respectively, and were similarly tested, but with an additional Pw20 trial after each of the three drug pretreatments. Since each fish acted as its own control, some results are presented as the number of fish showing a particular response. Mean values and the S.E.M. are presented in the tables and $P < 0{\cdot}05$ was used as a level of significance using a Student t-test. The Wilcoxon

paired-sample test was used to determine statistically significant differences ($P < 0{\cdot}05$) between the points in Figs 3, 4, 6.

Instrumentation

Blood flow was measured with an electromagnetic flow meter and associated probes (Zepeda SWF4, Seattle, WA). The flow probe was calibrated after each experiment using a known saline flow through the excised ventral aorta. Pulsatile blood pressures (Pva alone, or Pda and Pdc simultaneously) and the water pressure in the posterior chamber were monitored with Micron pressure transducers (Narco Life Sciences, Houston, TX) *via* a heparinized saline or water-filled cannula. The transducers were calibrated against a static water column before and after each experimental series, and referenced to the water level in the anterior chamber before and after each trial. The signals from the flow meter and pressure transducers were suitably amplified and displayed on a chart recorder (Gould 2400, Cleveland, OH).

RESULTS

Pretrial cardiovascular status

The resting cardiovascular variables prior to each trial are presented for the four experimental groups in Table 1. The higher ambient water temperature for groups 3 and 4 is reflected in higher values for f_H, $\dot{V}b$, vascular conductance, mean Pva and mean Pda. The low S.E.M. for each of these resting variables indicates consistency within each experiment, even though the fish were subject to surgery and

Table 1. *The resting cardiovascular variables recorded prior to each Pw trial*

	$\dot{V}b$ (ml min^{-1} kg^{-1})	f_H (beats min^{-1})	Pva (cmH_2O)	Pda (cmH_2O)	Vascular conductance (ml min^{-1} kg^{-1} cmH_2O^{-1})
Group 1 (7°C)	10·8 ± 0·5 (5)	31·1 ± 0·7 (6)	37·2 ± 0·6 (5)	—	0·289 ± 0·024 (4)
Group 2 (8°C)	—	35·9 ± 0·6 (3)	—	32·3 ± 0·9 (3)	—
Group 3 (10·5°C)	14·6 ± 0·8 (6)	39·4 ± 1·4 (7)	41·2 ± 0·6 (7)	—	0·353 ± 0·021 (6)
Group 4 (11°C)	—	38·3 ± 1·2 (7)	—	35·8 ± 0·4 (7)	—
Propranolol pretreatment	12·2 ± 1·4 (6)	34·5 ± 1·5 (7)	40·9 ± 0·4 (7)	34·6 ± 0·5 (7)	0·301 ± 0·004 (6)
Papaverine pretreatment	10·5 ± 1·2 (6)	33·7 ± 1·5 (7)	40·2 ± 0·8 (7)	33·3 ± 0·6 (7)	0·265 ± 0·033 (6)
Atropine pretreatment	10·8 ± 1·1 (6)	37·3 ± 1·1 (7)	40·3 ± 1·0 (7)	32·7 ± 0·7 (7)	0·267 ± 0·030 (6)

The control values following propranolol, papaverine and atropine pretreatments were obtained from fish in groups 3 and 4.

Values are presented as means ± S.E.M. (N fish). The resting value is based on a 30-s average for three trials from each fish. The drug pretreatment control values are based on one trial per fish.

considerable restraint. Furthermore, the similarity between resting values for successive Pw trials (see subsequent Figures) indicates a satisfactory protocol to ensure cardiovascular stability between trials.

Propranolol, papaverine and atropine pretreatments are considered to be cumulative because preliminary experiments indicated that the effects of propranolol and papaverine persisted for more than 1 h. The pretrial data for 14 fish from groups 3 and 4 are summarized in Table 1. Propranolol pretreatment reduced f_H and a subsequent papaverine pretreatment had little additional effect. Atropine pretreatment abolished all beat-to-beat variation in f_H and partially restored f_H to the control value. These data indicate that the beat-to-beat variation in f_H was produced by inhibitory cholinergic control and that there was some excitatory sympathetic tone to the heart under resting conditions.

$\dot{V}b$ was significantly reduced following all pretreatments because f_H was reduced and stroke volume was either reduced or unchanged. Vascular conductance decreased proportionately with $\dot{V}b$ so that Pva was unchanged and Pda was reduced by only 1–3 cmH_2O.

Effect of raising the water pressure in the posterior chamber

Increasing the water pressure in the posterior chamber compressed the trunk vasculature and reduced vascular conductance. Higher water pressures elicited a more pronounced or a more prolonged vascular compression. At Pw20, vascular conductance was reduced by as much as 25 % (Fig. 2).

Vascular compression clearly produced bradycardia which was more pronounced and prolonged with greater degrees of vascular compression at higher water pressures (Fig. 2). Nonetheless, there were large beat-to-beat oscillations in f_H during vascular compression, tending to obscure the cardiovascular trends. Two types of analysis were therefore performed to highlight trends. Firstly, every trial was plotted with each variable averaged for 10 consecutive heart beats. These averages were then pooled to establish an overall cardiovascular response for each group (i.e. Figs 3, 4, 6). Cardiovascular variables for the 3–8 s while Pw was being changed were not included in this analysis and so the trends reflect cardiovascular responses while Pw was stable. These analyses revealed that the reduction in vascular conductance was clearly associated with a reflex bradycardia (61 out of 69 trials). The bradycardia did not consistently reduce $\dot{V}b$ because of a compensatory increase in stroke volume ($\dot{V}b$ was reduced in 21 out of 33 trials). Because the reduction in vascular conductance was proportionately greater than any reduction in Vb, mean arterial blood pressures increased significantly. Both arterial blood pressures increased by approximately one-third of the increase in the water pressure. Thus, for example, at Pw20 there was a 6–7 cmH_2O increase in both Pva and Pda.

Atropine pretreatment completely abolished the oscillations in f_H (Fig. 5), the reflex bradycardia and major oscillations in $\dot{V}b$ associated with vascular compression (Fig. 6). This clearly demonstrated that inhibitory cholinergic tone (a) normally regulated f_H on a beat-to-beat basis and (b) was central to both the reflex bradycardia and thus the regulation of $\dot{V}b$ during vascular compression.

The drug pretreatments also illustrated the fact that the reflex bradycardia could, by reducing $\dot{V}b$, attenuate the hypertension produced by vascular compression. After propranolol and papaverine pretreatments, an accentuated reflex bradycardia clearly resulted in significantly lower ventral aortic pressures (Fig. 6). In contrast, when the reflex bradycardia was abolished by atropine pretreatment, arterial pressures were significantly increased beyond those in the control Pw20 trials. The bradycardia probably reflects a barostatic reflex.

The efficacy of barostatic control can be evaluated by calculating 'normalized gain' (percentage change in heart rate per unit change in mean arterial pressure; Smith, Berger & Evans, 1981). Normalized gain values for the average of the first 10 heart beats during vascular compression were significantly greater than zero using either the ventral or dorsal aortic blood pressure (Table 2). Propranolol and papaverine pretreatments significantly enhanced the gain of the reflex, and atropine abolished the reflex entirely.

The cardiovascular changes were greatest at the outset of the Pw change (Figs 2, 3, 4) because f_H, $\dot{V}b$ and vascular conductance recovered to some degree while Pw was still elevated. Complete cardiovascular recovery during vascular compression occurred only for Pw10 and Pw15 trials and more commonly at the higher water temperature (Figs 3, 4). The recovery of f_H occurred through a reduction in vagal tone since it was present after propranolol and papaverine, but not atropine, pretreatments. This implies that the barostatic reflex adapted with time, which was substantiated by a decrease in the normalized gain by the final 10 heart beats of most trials (Table 2). Nonetheless, normalized gain after 2 min of vascular compression was still greater than 1·0% cmH_2O^{-1} in 10 out of 12 trials. Interestingly, the mean Pva and Pda oscillated around relatively fixed values throughout the 2-min period because $\dot{V}b$ and vascular conductance recovered proportionately (Figs 3, 4).

Other factors affected cardiac performance during vascular compression in addition to vagal tone. Excitatory sympathetic tone was present throughout the trial since bradycardia was accentuated following propranolol pretreatment. This is well illustrated in Fig. 5, where f_H during vascular compression was higher in the control trial than following propranolol and atropine pretreatments. Furthermore, the recovery of $\dot{V}b$ with time, in the absence of a change in f_H following atropine pretreatment, indicates that $\dot{V}b$ was not entirely regulated by f_H.

During vascular compression, the changes in vascular conductance with time were predominantly passive since proportionate changes in $\dot{V}b$ and vascular conductance were always present after pretreatment with a sympathetic antagonist and a vascular smooth muscle poison. Moreover, following atropine pretreatment, vascular compression was offset by the constant $\dot{V}b$ and increased arterial pressures (Fig. 6).

Post-trial recovery

Recovery of the cardiovascular variables when Pw was returned to zero was accomplished well within the 1 h between successive trials. f_H was restored within 20–30 heart beats (Figs 3, 4). $\dot{V}b$ and sometimes vascular conductance were still

Table 2. *Normalized gain values derived from the heart rate and arterial blood pressures for the initial and final periods of 10 heart beats during vascular compression*

	Gain with mean Pva (% cmH_2O^{-1})				Gain with mean Pda (% cmH_2O^{-1})			
	Group 1 ($N = 5$)		Group 3 ($N = 7$)		Group 2 ($N = 3$)		Group 4 ($N = 7$)	
Trial	Initial	Final	Initial	Final	Initial	Final	Initial	Final
Pw10	2·20 ± 1·22	4·21 ± 3·65	1·92 ± 0·71	0·87 ± 0·25	2·05 ± 1·15	2·54 ± 1·26	3·82 ± 1·09	24·54 ± 22·36
Pw15	6·68 ± 5·45	5·23 ± 5·00	2·88 ± 1·61	1·01 ± 0·42	1·35 ± 0·34	0·98 ± 0·17	3·32 ± 1·44	1·70 ± 0·55
Pw20	5·80 ± 2·77	3·61 ± 2·07	2·95 ± 1·22	1·37 ± 0·47	1·67 ± 0·72	3·90 ± 3·61	3·25 ± 0·56	2·86 ± 1·69
Pw20 after propranolol	—	—	34·89 ± 17·13	22·99 ± 19·97	—	—	5·70 ± 1·78	2·54 ± 0·69
Pw20 after papaverine	—	—	58·82 ± 32·92	10·57 ± 4·97	—	—	7·85 ± 2·44	3·43 ± 0·97
Pw20 after atropine	—	—	0	0	—	—	0	0

Normalized gain was calculated from the percentage change in heart rate per unit change in mean arterial pressure. Values are presented as means ± S.E.M. All gain values were significantly greater than 0 ($P < 0·05$) with the exception of those after atropine pretreatment.

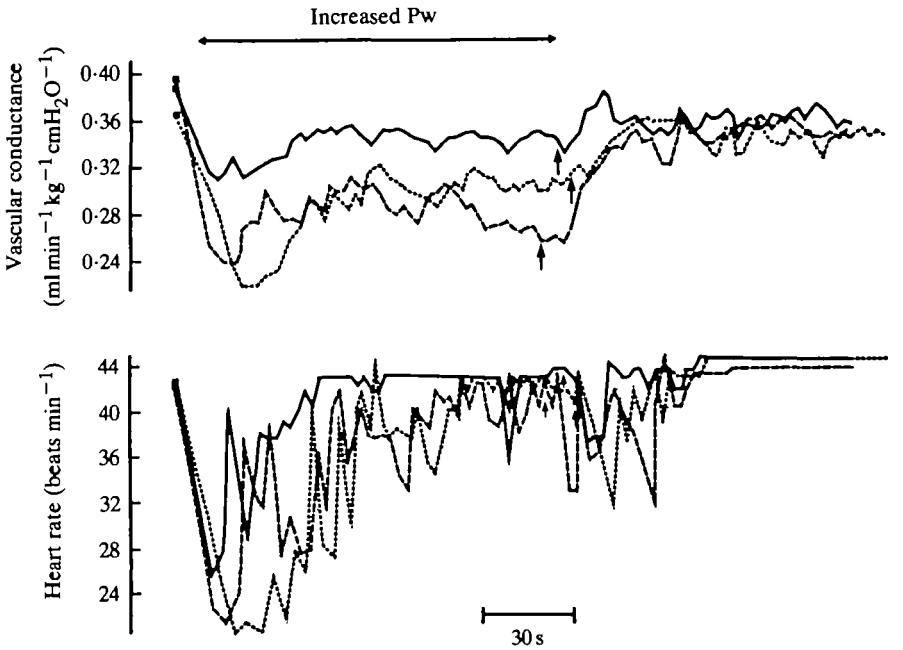

Fig. 2. A beat-to-beat analysis of heart rate and vascular conductance in a fish during and following vascular compression. The three increases in the water pressure [Pw10 (——), Pw15 (.....) and Pw20 (– – –)] are superimposed for comparison. The first point represents the resting value averaged from the 30 s preceding the trial. The arrows indicate the exact time when Pw was restored to the water level.

reduced after 30–60 s, and resulted in a transient undershoot of arterial blood pressures. This undershoot probably reflected a transient pooling of blood in the systemic vessels as normal vascular dimensions were restored at the control water pressure. Occasionally there was brief bradycardia (2–5 beats) as Pw was being lowered rapidly (Fig. 2).

Venous pressures

Reliable pressure measurements from the ductus Cuvier were obtained in only six of 10 fish. The resting venous pressure was usually between 0 and +1·5 cmH_2O and was about −0·5 cmH_2O in one fish. A detailed, quantitative analysis of the Pdc data was not made because pressure oscillations were superimposed on the blood pressure trace. These oscillations were often greater than any experimentally induced change in the mean Pdc and, since they were visually associated with ventilation, they were probably artifacts caused by the cannula movements as the operculum opened and closed. Nevertheless, a clear increase in Pdc (0·5–1 cmH_2O) was apparent in 12 out of the 18 trials when the water pressure was raised. The increase was always seen at Pw20 and was unaffected by any of the drug pretreatments (four fish). Pdc returned to control levels when the control water pressure was restored. The amplitude of ventilation also increased during the Pw trial (as indicated by the Pdc oscillations associated with ventilation) but ventilation frequency was unchanged.

DISCUSSION

The vascular compression was restricted to the trunk and predominantly affected systemic vascular conductance. However, changes in systemic vascular conductance are not discussed because Pda and $\dot{V}b$ (or dorsal aortic flow) were not measured simultaneously in an effort to minimize surgery. Instead, changes in total vascular conductance are discussed. This approach seems justified because (a) total vascular conductance is largely determined by the systemic circuit (Jones & Randall, 1978) and (b) the absolute changes in mean Pva and Pda were similar when Pw was elevated and so changes in total and systemic conductances paralleled each other. The calculation of total vascular conductance was, nevertheless, underestimated, since Pdc was not accounted for. Because Pdc was generally less than 1 cmH_2O, the error was small (<3 %) and only increased by 1–2 % during vascular compression.

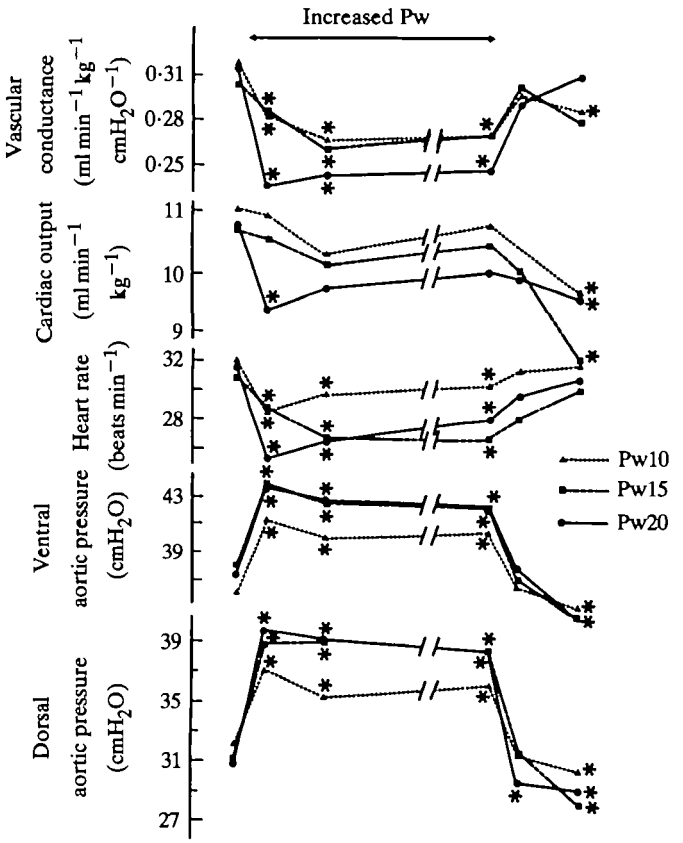

Fig. 3. An analysis of the cardiovascular trends during vascular compression for fish from groups 1 and 2. The first point is the resting value. The remaining points are mean values for periods of 10 consecutive heart beats (three to six fish). The second, third and fourth points represent the two initial and final periods during vascular compression. The first two periods of the recovery when Pw was restored to the control level are indicated by the last two points. The lines for different trials are superimposed for comparison. Cardiovascular changes while Pw was changing were excluded from this analysis. An asterisk indicates a statistically significant difference ($P < 0{\cdot}05$) from the resting value.

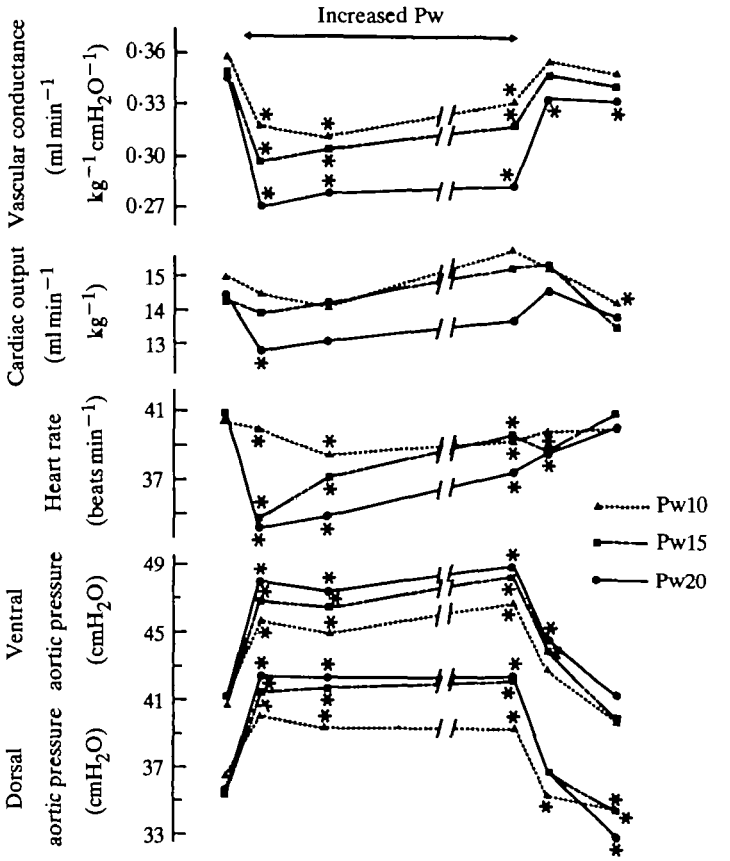

Fig. 4. An analysis of the cardiovascular trends during vascular compression for fish in groups 3 and 4. These fish were at a higher ambient temperature than the fish in groups 1 and 2. Data were derived in the same manner as described in Fig. 3 using six or seven fish for each point. An asterisk indicates a statistically significant difference ($P < 0{\cdot}05$) from the resting value.

During vascular compression of the trunk, a strategy of reducing $\dot{V}b$ and thereby limiting the increase in blood pressure was clearly adopted by the fish. (It is likely that there was a redistribution of blood flow, but to what degree is unknown.) The most important effector in this response was a reflex cholinergic bradycardia. The bradycardia accompanying vascular compression could be either a barostatic reflex or a general startle reflex, both of which are vagally mediated (Stevens, Bennion, Randall & Shelton, 1972; Priede, 1974; Jones & Randall, 1978; Farrell, 1982; Nilsson, 1984). The startle reflex is characterized by short periods of low heart rates and, at least in the lingcod, a brief ventilation apnoea (Farrell, 1982). A startle reflex, including the ventilation apnoea, was sometimes seen during the brief period when Pw was being changed and it was usually followed by a struggle. None of the trials analysed here incorporated struggles, and furthermore ventilation frequency was constant while Pw was constant. It is unlikely, therefore, that the bradycardia during the trial was a startle reflex. To further minimize any contribution of a startle reflex

to the bradycardia associated with a constant level of vascular compression, the analysis did not include cardiovascular changes when Pw was being changed.

Because the bradycardia helped attenuate the hypertension associated with vascular compression, it probably represents a barostatic reflex. Several observations support this conclusion. (a) Greater reductions in vascular conductance were associated with a more pronounced or prolonged bradycardia. (b) Propranolol and papaverine pretreatment accentuated the bradycardia and resulted in a lower arterial blood pressure during vascular compression, i.e. the sympathetic excitation of the heart antagonized the barostatic reflex. In contrast, atropine pretreatment abolished the bradycardia and enhanced the pressor effect of vascular compression. (c) The normalized gain values using either Pva or Pda compare favourably with other studies demonstrating baroreceptor function: about 1·3% cmH_2O^{-1} for the toad (Smith *et al.* 1981), 10% cmH_2O^{-1} for the lizard and 2·7–6·9% cmH_2O^{-1} for the dog, rabbit and man (Berger, Evans & Smith, 1980). Although normalized gain has been criticized because it only analyses one component of the response (Jones & Milsom, 1982), it is the best available method for comparing such data.

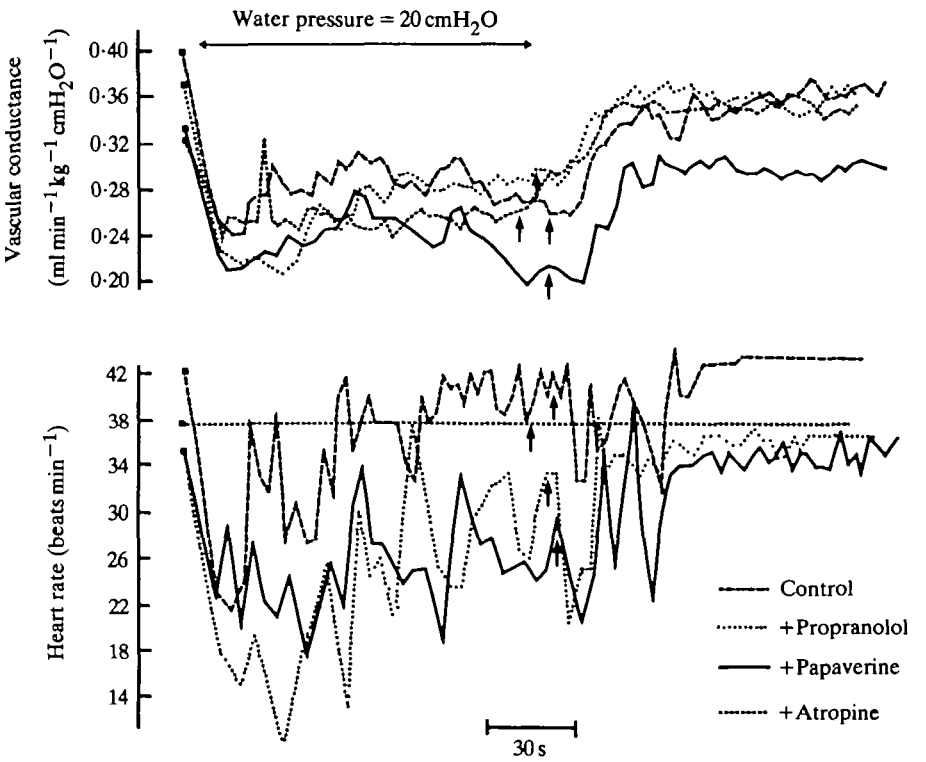

Fig. 5. A beat-to-beat analysis of heart rate and vascular conductance in a fish during and following vascular compression at Pw20 with and without drug pretreatment. The first point represents the control value averaged from the 30 s preceding the trial. The three trials following cumulative pretreatments of propranolol, papaverine and atropine are superimposed with the control trial for comparison.

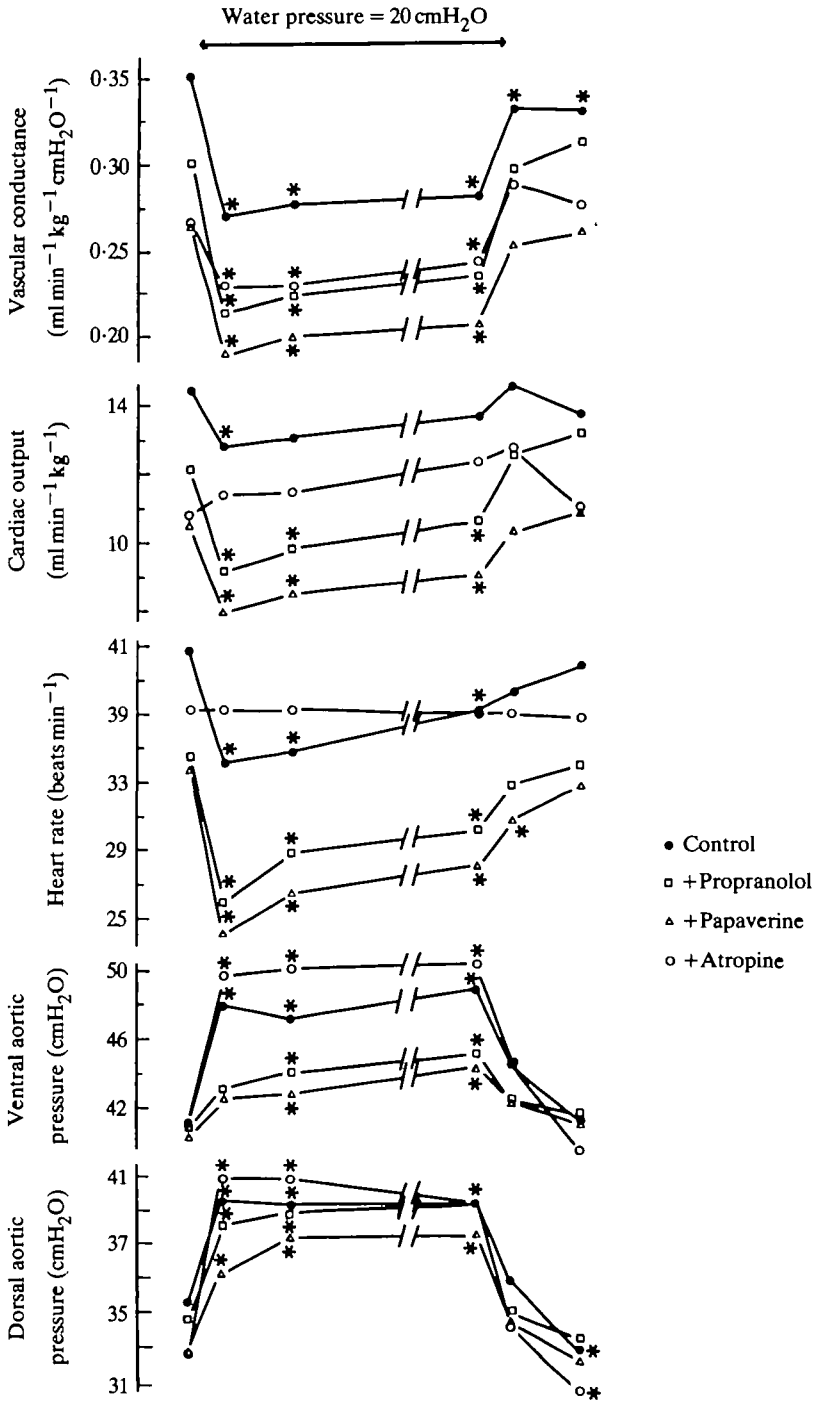

Fig. 6. An analysis of the cardiovascular trends during vascular compression at Pw20 following cumulative pretreatments with propranolol, papaverine and atropine. The control trial (from Fig. 4) is included for comparison. Derivation of data points is explained in the legend for Fig. 3. An asterisk indicates a statistically significant difference ($P < 0{\cdot}05$) from the corresponding resting value.

(d) Regulation of blood pressure was also evident during the pretrial period. For example, Pva was well regulated despite the decrease in $\dot{V}b$ following each of the drug pretreatments (see Table 1).

The present work clearly complements the definitive work with rainbow trout which established that a barostatic reflex can effect a vagal tachycardia during hypotension produced by either haemorrhage, α-adrenoceptor blockade or low doses of acetylcholine (Wood & Shelton, 1980*b*). Together these studies represent the most quantitative and comprehensive demonstrations of the barostatic reflex in teleost fish. Previous studies, though often quoted as indicative of barosensitivity, were not always definitive. In anaesthetized eels, pressures in afferent branchial arteries needed to be increased substantially (30–40 cmH_2O) to elicit bradycardia lasting apparently one beat (see fig. 7 in Mott, 1951), and an increase of 50 cmH_2O was needed to increase discharge in sensory fibres of the branchial nerves. Similar experiments were performed on spinalectomized carp (Ristori, 1970; Ristori & Dessaux, 1970), but quantification of the barosensitive reflex was simply a 15 % reduction in f_H. Evidence supporting a barostatic reflex bradycardia has also been based on pressor effects of injections of adrenaline *in vivo*. Careful scrutiny of such work provides clear examples of decreases in f_H in phase with the pressor responses (Helgason & Nilsson, 1973; Pettersson & Nilsson, 1980), but authors more frequently report that bradycardia was not in phase with the pressor response or was absent (Randall & Stevens, 1967; Helgason & Nilsson, 1973; Chan & Chow, 1976; Stevens *et al.* 1972; Wood & Shelton, 1980*a*; Farrell, 1981). Perhaps a more suitable conclusion is that adrenaline infusions are not an appropriate method to demonstrate the barosensitive reflex in fish.

Since a barostatic reflex is present in teleosts, it cannot represent a general circulatory compensatory mechanism which evolved solely in response to the effects of gravitational stresses on the circulatory system (see Jones & Milsom, 1982). Instead, in teleosts, it may represent a mechanism to ensure that the maximum mechanical power output of the heart is not exceeded. There are at least three lines of evidence which support this idea. First, comparable data from studies with perfused hearts (i.e. $\dot{V}b = 11\ ml\ min^{-1}\ kg^{-1}$ at 10°C) indicate that the maximum mean afterload ($\equiv$ mean Pva) at which $\dot{V}b$ can be maintained with a constant preload is 50–55 cmH_2O. Beyond this pressure the heart becomes pressure-sensitive and $\dot{V}b$ decreases (Farrell, MacLeod & Driedzic, 1982). In the present study, bradycardia prevented the maximum afterload from being exceeded, i.e. the mean Pva increased to about 43 cmH_2O at 6–9°C and about 48 cmH_2O at 10–12°C. Following atropine pretreatment, mean Pva approached the maximum afterload (i.e. slightly above 50 cmH_2O during vascular compression) although $\dot{V}b$ was maintained. Second, Wood & Shelton (1980*a*) noted that the trout heart was pressure-sensitive and the major portion of the bradycardia was associated with the very high blood pressure (mean Pva often exceeding 60 cmH_2O) after an adrenaline injection was passive and atropine-insensitive. The reflex bradycardia in the present study was completely eliminated by atropine pretreatment. Last, the set point for the barostatic reflex evidently increases with water temperature. This would be expected if power output

of the heart was limiting since its maximum power output increases with temperature in the sea raven heart (Graham & Farrell, 1985). Whether the bradycardia *per se* also provides a mechanical advantage to the heart, e.g. reduced dP/dt or improved oxygen extraction, is unclear. It is clear, however, that a barostatic reflex is important to fish and it can override chemoreceptive drives (Wood & Shelton, 1980*b*).

One consequence of vascular compression in swimming fish is now evident; a reflex bradycardia will decrease $\dot{V}b$ if the vascular compression is extensive enough to elevate arterial blood pressure. Furthermore, accommodation of the barostatic reflex with time ensures that blood flow to the tail is not reduced for extensive periods. The brief bradycardia that can precede sustained swimming in trout may reflect accommodation of a barostatic reflex. However, in fish which burst-swim and where muscular contraction is more extensive, the reflex bradycardia may last long enough (2 min or more) to reduce $\dot{V}b$ throughout most of the exercise period (e.g. lingcod, Stevens *et al.* 1972; Farrell, 1982). Thus, while baroreceptors in the sea raven adapt with time, they may not adapt as rapidly as previous studies with other species suggest (see Jones & Milsom, 1982).

The recovery of vascular conductance during vascular compression tended to restore blood flow to muscle, even though the level of vascular compression was constant. Cholinergic and adrenergic influences in this response can be ruled out because the recovery was not abolished by atropine and propranolol pretreatment. Autoregulation is also unlikely since papaverine did not abolish recovery. In a similar study with isolated, perfused tails from ocean pout (*Macrozoarces americanus*) no myogenic autoregulation was observed with vascular compression (Canty & Farrell, 1985). It is, therefore, more likely that vascular conductance increased passively as $\dot{V}b$ increased with time. Passive increases in vascular conductance occurred when $\dot{V}b$ changed in the lingcod (Farrell, 1982) and may also contribute to the fall in arterial blood pressure in the early period of sustained swimming when $\dot{V}b$ is elevated (Kiceniuk & Jones, 1977), as suggested by Wood (1974).

In summary, vascular compression, if profound enough, can have important cardiovascular consequences. It can elicit a barostatic reflex bradycardia which decreases $\dot{V}b$, reduces blood flow to the muscle, and thereby may prevent the heart from exceeding its intrinsic capacity for pressure work. The accommodation of the barostatic reflex with time, plus a modest, passive vasodilatation as $\dot{V}b$ is restored, ensures that the reduced blood flow is only temporary. The set point for the barostatic reflex appears to be temperature-dependent.

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