The influence of temperature and anaemia on the adrenergic and cholinergic mechanisms controlling heart rate in the rainbow trout

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The capacity for cardioacceleration by adrenergic and cholinergic mechanisms was studied in resting rainbow trout, *Salmo gairdneri*, at 5, 12, and 20°C. The trout were fitted with chronic dorsal aortic catheters for heart rate and blood pressure measurements. At all three temperatures, muscarinic cholinergic blockade with atropine caused substantial tachycardia, thereby indicating the presence of cholinergic vagal tone in resting animals. The relative effect of atropine was significantly greater at 5°C (+53% of resting heart rate) than at 12 and 20°C (≈ +30%). Maximal adrenergic stimulation (via adrenaline) after atropine caused a small further cardioacceleration at all temperatures. The adrenaline effect increased significantly from +8% (of resting heart rate) at 5°C to +15% at 20°C. The findings provide qualified support for the hypothesis that a reduction in vagal cholinergic activity is relatively more important in tachycardia at low temperature, and that adrenergic stimulation is relatively more important at high temperature, although the cardioacceleratory capacity of the cholinergic mechanism remains dominant throughout. The increase in heart rate accompanying experimental anaemia (haematocrit ≤ 6%) at all three temperatures was almost entirely due to a removal of vagal cholinergic tone; the contribution of adrenergic mechanisms, if any, was small.

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Nous avons étudié le capacité de cardioaccélération par des mécanismes adrénergique et cholinergique chez des truites arc-en-ciel, *Salmo gairdneri*, au repos, à 5, 12 et 20°C. La pression sanguine et le rythme cardiaque ont été enregistrés grâce à des cathéters chroniques installés dans l'aorte dorsale. A toutes les températures, un blocage muscarinique cholinergique à l'atropine cause une tachycardie importante, ce qui révèle la présence d'un tonus vagal cholinergique chez les animaux au repos. L'effet de l'atropine est significativement plus important à 5°C (+53% du rythme cardiaque au repos) qu'à 12 ou à 20°C (≈ +30%). Une stimulation adrénergique maximale (par l'adrénaline) après l'administration d'atropine cause une nouvelle cardioaccélération de moindre importance, à toutes les températures. L'effet de l'adrénaline augmente significativement de +8% (du rythme cardiaque au repos) à 5°C à +15% à 20°C. Les résultats appuient l'hypothèse selon laquelle la réduction de l'activité vagale cholinergique est relativement plus importante à basse température, lors d'une tachychardie et la stimulation adrénergique est relativement plus importante à haute température, même si la capacité de cardioaccélération du mécanisme cholinergique demeure toujours le phénomène dominant. L'augmentation du rythme cardiaque durant une anémie expérimentale (hématocrite ≤ 6%) aux trois températures est presque entièrement due à la disparition du tonus cholinergique vagal; le contribution des mecanismes adrénergiques est négligeable, voire inexistante.

[Traduit par le journal]

Introduction

The regulation of heart rate in teleost fish has recently been reviewed in detail by Jones and Randall (1979). After assessing a wealth of contradictory information, these authors concluded that "the control of the fish heart during exercise is still not understood although it appears most likely that the tachycardia is a product of a vagal sympathetic contribution allied with a withdrawal of parasympathetic activity and a direct effect of circulating catecholamines." Much of the disagreement among various studies may reflect differences in experimental conditions. Temperature is one experimental variable which has not been systematically studied in relation to the mechanisms of heart rate control in teleost fish. However, there is circumstantial evidence that temperature may affect the relative importance of cholinergic mechanisms (i.e. vagal parasympathetic activity) and adrenergic mechanisms (i.e. vagal and spinal nerve sympathetic activity plus circulating catecholamines) in causing tachycardia.

For example, in the rainbow trout (*Salmo gairdneri*) at 5°C, Priede (1974) reported that bilat-

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eral vagotomy increased resting heart rate almost to the levels recorded during exercise in intact animals. Exercise heart rates were therefore unaffected by this procedure. However at 15°C, bilateral vagotomy did not alter resting heart rate but significantly reduced the exercise tachycardia. Similarly, cholinergic vagotomy by pharmacological means (atropine) did not affect either resting or exercise heart rate in trout at 10–15°C (Stevens and Randall 1967; Wood 1974). At a slightly lower temperature range (9.5–11.0°C), adrenergic sympathectomy by pharmacological means (bretylium) did not affect either resting or exercise heart rates (Smith 1978). In the carp (*Cyprinus carpio*), intravenous adrenaline caused bradycardia at low temperatures (1–8°C) but tachycardia at higher temperatures (9–20°C; Laffont and Labat 1966).

One interpretation that can be placed on these reports is that a reduction in cholinergic activity (i.e. withdrawal of vagal parasympathetic tone) is relatively more important in causing tachycardia at low temperature and that adrenergic stimulation (i.e. increased sympathetic activity and (or) plasma catecholamines) is relatively more important at higher temperatures. The present study was designed to test this hypothesis in the rainbow trout by examining the maximum increases in resting heart rate which could be elicited via cholinergic withdrawal (by atropine) or adrenergic stimulation (by adrenaline) at low (5°C), intermediate (12°C), and high (20°C) temperatures. During the course of the investigation, it became apparent that the chronotropic response could be influenced by the haematocrit of the animal. Therefore the influence of experimental anaemia on the cholinergic and adrenergic responses was also examined at the three temperatures.

Materials and Methods

Experiments were performed on 80 rainbow trout (*Salmo gairdneri*; 94–550 g) which had been acclimated for at least 2 weeks to one of the three experimental temperatures in flowing, dechlorinated fresh water. The actual experimental temperatures ($\bar{x} \pm 1$ SE (N)) were 5.25 ± 0.17 (29), 11.68 ± 0.21 (30), and 20.36 ± 0.07 (21)°C, or nominally 5, 12, and 20°C. Chronic dorsal aortic and subintestinal vein cannulae were installed while the fish were anaesthetized (1 : 15 000 tricaine methanesulfonate (MS-222), Sigma) on an operating table at the acclimation temperature. The dorsal aortic (DA) catheter was implanted as described by Smith and Bell (1964) and Holeton and Randall (1967) and consisted of 40 cm of Clay-Adams PE 60 (outside diameter 1.21 mm) polyethylene tubing tipped with a No. 21 Huber point needle. For subintestinal (SI) catheterization, the vein was exposed by dissection in the ventral pelvic midline, cannulated anteriorly with 40 cm of PE 50 (o.d. 0.97 mm) which had been stretched to PE 10 (o.d. 0.61 mm) diameter at its tip, and tied off posteriorly in a procedure similar to that of Randall and Stevens (1967). The wound was closed with silk suture. Both catheters were filled with heparinized (52 USP units/mL) Cortland saline (Wolf 1963). The trout were then placed in darkened Plexiglas or wooden boxes provided with a continuous flow ($\approx$ 200 mL/100 g min^{-1}) of aerated, dechlorinated water at the experimental temperature, and allowed to recover for at least 24 h.

Haematocrit was checked at the start of each experiment (except some low temperature trials, see Results) by centrifuging 50-μL blood samples in sodium heparinized capillaries at 5000 *g* for 5 min. Trout with haematocrits $\leq 6\%$ were considered anaemic. This level was chosen on the basis of previous studies on both trout and flounder (Cameron and Davis 1970; Wood *et al.* 1979; C. M. Wood, D. G. McDonald, and B. R. McMahon, unpublished results) showing that major changes in blood gas tensions and cardiovascular regulation only occur below this haematocrit. This anaemia occurred either naturally, or as a result of blood loss during or following surgery, or, in the majority of animals, as a result of sequential experimental bleeding during the recovery period. No differences could be detected amongst the results from animals rendered anaemic by the different causes. In the sequential bleeding procedure, approximately 1 mL/100 g of blood was removed via the DA catheter, centrifuged to separate out the erythrocytes, and then the homologous plasma was reinfused together with sufficient saline to replace the cells. The procedure was repeated until the desired haematocrit was reached. At least 6 h was allowed to elapse between the last bleeding and the start of an experiment.

Dorsal aortic blood pressure was continuously recorded throughout each experiment by a Hewlett-Packard 267 BC pressure transducer, amplified by a Sanborn 150-3000 carrier preamplifier, and displayed on a Sanborn 150 recorder writing on rectilinear coordinates. Heart rate was either counted visually from the pressure trace or automatically by a Biotach 4710 ratemeter (Transmed Scientific) fired from the rising phase of the blood pressure signal. Drugs were injected through either the SI or DA catheters (see Results) in a volume of 0.05 mL/100 g of Cortland saline and washed in with a further 0.10 mL/100 g of saline. The drugs and doses used were atropine sulphate (Sigma; 100 nmol/100 g), acetylcholine chloride (Sigma; 10 nmol/100 g), and l-adrenaline bitartrate (Sigma; 10 nmol/100 g). The doses were selected on the basis of previous studies (Wood 1974; C. M. Wood and G. Shelton, unpublished results) and preliminary experiments which indicated the following: acetylcholine at 10 nmol/100 g causes a profound bradycardia in the absence of muscarinic blockade, atropine at 100 nmol/100 g effects a complete muscarinic cholinergic blockade with negligible cardiodepressant action, and adrenaline at 10 nmol/100 g produces maximal adrenergic stimulation of the heart.

Resting values were recorded approximately 1 h after connecting the animal to the recording system; animals exhibiting overt signs of disturbance were not used. Atropine was then injected, followed at approximately 30-min intervals by acetylcholine and finally adrenaline. The acetycholine injection was used as a check on the efficacy of the muscarinic blockade caused by atropine. In rare instances when acetylcholine-induced bradycardia persisted, atropinization was repeated at the same dose level until the bradycardia disappeared. After atropine, heart rate and blood pressure reached stable values within 2–3 min, and consistent measurements could be taken any time during the succeeding 30 min. In practice, the values were sampled approximately 3–6 min after administration of the drug. Adrenaline, on the other hand, caused rapid (< 1 min) increases in both blood pressure and heart rate which slowly decayed (5–20 min). The values recorded represent the maxima for each parameter. Mean DA blood pressures were calculated as (1 systolic + 2 diastolic)/3 (Burton 1972). Statistical compari-

sons within groups were made by the paired Student's two-tailed t-test ($p \leq 0.05$), using each fish as its own control. The unpaired Student's two-tailed t-test ($p < 0.05$) was employed for comparisons between groups.

Results

The Influence of Methodology

The original plan for the study was to use the SI catheter for all injections, thereby allowing drugs to pass directly through the venous system to the heart without first being dissipated in the systemic capillary beds. However, in initial experiments at 12°C, we noted that the fish fitted with both SI and DA catheters tended to have higher resting heart rates than those bearing only DA cannulae. Closer analysis revealed that these higher resting rates occurred only in SI + DA fish with abnormally low haematocrits (≤ 6%), that the same phenomenon occurred in the occasional DA only fish with low haematocrit, and that the incidence of severe anaemia was much greater in the former than in the latter. Apparently the surgery involved in SI catheterization greatly increased the probability of blood loss.

In view of this problem, we systematically tested whether the experimental results differed between SI + DA fish (SI injection) of normal haematocrit and DA only fish (DA injection) of normal haematocrit at 12°C. Table 1 clearly shows that the results for the entire experimental protocol were identical in the two groups. Thus injection of drugs into the dorsal aorta produces exactly the same effects as injection into the subintestinal vein, despite the fact that the agents pass through the systemic circulation before reaching the heart. Consequently DA only fish were used in all experiments at the other two temperatures, and the results for both normal haematocrit groups at 12°C were combined in subsequent analyses (Fig. 1, Tables 2, 3). These findings also prompted a systematic study of the influence of severe anaemia on heart rate control at all three temperatures (Fig. 2, Table 2). Again, in all cases DA only fish were employed.

The Influence of Temperature

Because of a procedural error, haematocrits were only recorded in normal fish at 12 and 20°C. The normal haematocrit data reported in Table 2 for 5°C were actually taken from the anaemic group before the sequential bleeding procedure, and therefore should be representative of normal values at this temperature. Table 2 shows that normal haematocrits did not differ significantly at the three temperatures.

Resting heart rate in normal fish rose with a $Q_{10} = 2.6$ from 5 to 12°C and with a $Q_{10} = 1.5$ from 12 to 20°C (Fig. 1A). Atropine caused a significant increase in heart rate at all three temperatures. On an absolute basis, the mean increases were identical at 5 and 12°C and about twice as large at 20°C (Table 2). Between 5 and 12°C the Q_{10} for the atropinized heart rate was reduced to 2.0, but was nearly the same as the resting heart rate Q_{10} between 12 and 20°C (1.6 compared with 1.5). After atropine, the heart rate remained remarkably stable; the bradycardia associated with "startle" reflexes (e.g. as caused by tapping on the fish box) was abolished although the pressor response to such disturbance persisted. Adrenaline injected after atropine caused a further significant increase in heart rate at all three temperatures, but the elevation was very small at 5°C (Fig. 1A, Table 2). The adrenaline effect increased progressively with temperature on an absolute basis, but remained less than half the atropine-mediated increase at all three temperatures. There was no further significant change in Q_{10} (5–12°C = 2.1; 12–20°C = 1.6) from that determined for the atropinized heart rate.

Mean resting DA blood pressure in normal fish averaged about 31 cmH_2O (1 cmH_2O = 98.07 Pa) and was unaffected by temperature (Fig. 1B). The mean pressure did not change after atropine at any temperature (Fig. 1B), although there was a tendency for the pulse pressure to fall by up to 50%. Adrenaline caused a dramatic pressor effect at all

TABLE 1. A comparison of the responses in heart rate and dorsal aortic blood pressure to atropine and adrenaline between fish of normal haematocrit fitted with subintestinal and dorsal aortic catheters (subintestinal injection) and fish of normal haematocrit fitted with dorsal aortic catheters only (dorsal aortic injection) at 12°C. Means ±1 SE

	Subintestinal + dorsal aortic (N=10)	Dorsal aortic only (N=11)
Haematocrit, volume %	22.3±3.1	25.5±4.1
Weight, g	254.4±8.6	239.5±6.7
Resting heart rate, no./min	55.4±3.9	53.2±3.6
Atropine heart rate, no./min	68.5±3.1	67.4±1.8
Adrenaline heart rate, after atropine, no./min	74.4±4.2	73.7±1.8
Resting blood pressure, cmH_2O	31.9±2.0	33.4±1.8
Atropine blood pressure, cmH_2O	30.0±2.2	33.9±1.9
Adrenaline blood pressure after atropine, cmH_2O	80.7±2.2	86.7±3.3

NOTE: There are no significant differences between the two groups.

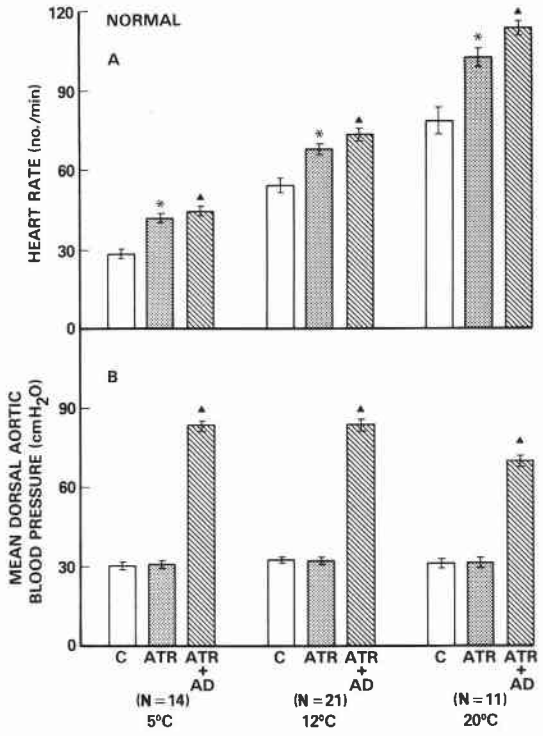

FIG. 1. The influence of atropine (100 nmol/100 g) and adrenaline (10 nmol/100 g) after atropine on (A) heart rate and (B) mean dorsal aortic blood pressure in rainbow trout of normal haematocrit at 5, 12, and 20°C. (means ± 1 SE). C, control resting value; ATR, atropine; AD, adrenaline; *, significantly different from control value; ▲, significantly different from both control value and atropine value.

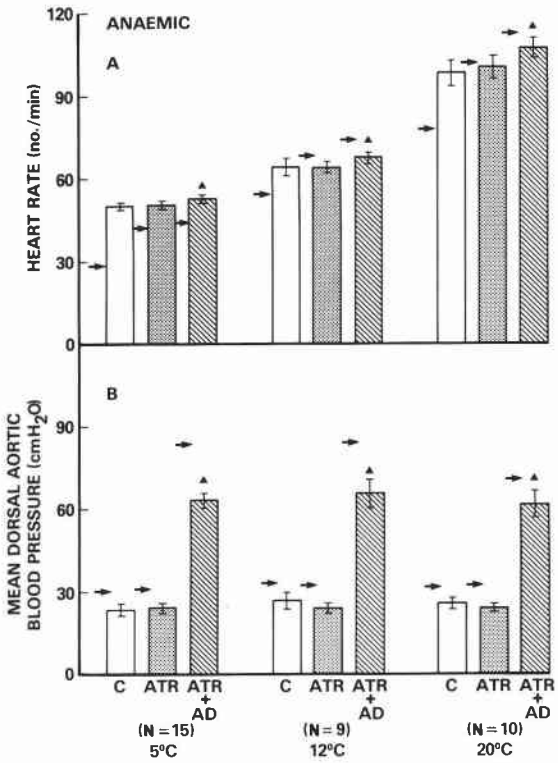

FIG. 2. The influence of atropine (100 nmol/100 g) and adrenaline (10 nmol/100 g) after atropine on (A) heart rate and (B) mean dorsal aortic blood pressure in anaemic rainbow trout (haematocrit ≤ 6%) at 5, 12, and 20°C (means ± 1 SE). *C*, control resting value; ATR, atropine; AD, adrenaline. There were no significant differences between control and atropine values. ▲, significantly different from both control value and atropine value. Arrows indicate the mean value for each parameter in the normal fish of Fig. 1.

three temperatures (Fig. 1B). The peak pressures were comparable at 5 and 12°C (≈ 84 cmH_2O) but significantly lower at 20°C (≈ 70 cmH_2O).

The Influence of Anaemia

The haematocrits of the anaemic trout did not differ significantly at the three temperatures (Table 2). Resting heart rates were consistently elevated above those in the normal groups at all temperatures (Fig. 2A). This difference was significant at 5 and 20°C, and just failed to obtain significance (p = 0.05–0.10) at 12°C. Indeed, the resting heart rates of anaemic fish were similar to the atropinized heart rates of the normal fish (Fig. 2A). Furthermore, atropine had no effect on heart rate at any temperature during anaemia (Fig. 2A, Table 2). However, adrenaline still significantly increased heart rate at all three temperatures above the atropinized levels (Fig. 2A). On an absolute basis, the adrenaline effect was identical to that in normal fish at 5°C, and only slightly (nonsignificantly) smaller at 12 and 20°C (Table 2). Therefore the effects of anaemia and atropine on heart rate are similar, and the elevated resting rate during anaemia appears largely attributable to a withdrawal of vagal cholinergic tone with negligible adrenergic involvement.

During anaemia, resting DA blood pressures were again unaffected by temperature (Fig. 2B). However, the mean pressures were consistently about 20% below the levels in normal fish (Fig. 2B), while the pulsatility of the trace was often much greater. Atropine again had no effect on mean pressure (Fig. 2B) but decreased pulsatility by up to 50%. As in the normal fish, adrenaline produced a dramatic rise in pressure at all temperatures, but the peak pressor effects were reduced by about the same relative amounts (≈ 20%) as the resting levels (Fig. 2B).

Discussion

When expressed on an absolute basis (Table 2), the present results indicate that the cardioacceleratory effects of both atropine and adrenaline increase with temperature. However, if tachycar-

TABLE 2. The influence of atropine and adrenaline on heart rate in normal and anaemic rainbow trout at 5, 12, and 20°C. Effects are expressed as absolute increases in heart rate. Means ±1 SE (*N*)

	5°C	12°C	20°C
Normal			
Haematocrit, volume %	30.4±4.2† (10)	23.9±2.4 (21) p_1 = n.s.	23.0±2.7 (11) p_1 = n.s. p_2 = n.s.
Atropine, Δ heart rate, no./min	13.8±1.9 (14)	13.6±1.8 (21) p_1 = n.s.	23.9±2.6 (11) $p_1 < 0.01$ $p_2 < 0.01$
Adrenaline, after atropine, Δ heart rate, no./min	2.2±0.6 (14)	6.1±1.1 (21) $p_1 < 0.01$	10.8±2.7 (11) $p_1 < 0.01$ p_2 = n.s.
Anaemic			
Haematocrit, volume %	3.4±1.4* (14)	3.1±0.7* (9)	4.2±1.8* (10)
Atropine Δ heart rate, no./min	0.4±0.5* (14)	−0.2±1.1* (9)	1.7±3.0* (10)
Adrenaline, after atropine, Δ heart rate, no./min	2.4±0.3 (14)	3.6±1.7 (9)	7.5±3.6 (10)

NOTE: p_1, significance relative to value at 5°C; p_2, significance relative to value at 12°C; *, significantly different ($p < 0.05$) from corresponding mean for normal fish; †, value taken from anaemic group before bleeding, see text for details; n.s., not significant.

TABLE 3. The influence of atropine and adrenaline on heart rate in rainbow trout at 5, 12, and 20°C. Effects are expressed as percentage increases of resting heart rate. Means ±1 SE (*N*)

	5°C	12°C	20°C
Atropine	53.0±9.8 (14)	28.9±5.0 (21) $p_1 < 0.05$	32.0±3.9 (11) $p_1 < 0.05$ p_2 = n.s.
Adrenaline, after atropine	8.1±2.4 (14)	12.2±2.7 (21) p_1 = n.s.	15.0±1.8 (11) $p_1 < 0.05$ p_2 = n.s.

NOTE: p_1, significance relative to increase at 5°C; p_2, significance relative to increase at 12°C; n.s., not significant.

dia is viewed as a mechanism for increasing cardiac output, it is the relative or percentage increase in resting heart rate rather than the absolute increase which is most important. Expressed on this basis, the data clearly show that the atropine effect is greatest at 5°C (≈ +53%) and declines significantly to approximately equal values (≈ +30%) at 12 and 20°C (Table 3). The adrenaline effect, on the other hand, progressively increases with temperature (Table 3). Note however, that even at the highest temperature, the influence of adrenaline after atropine remains less than half the influence of atropine alone. Thus the results indicate that the relative capacity for increase of heart rate by withdrawal of vagal cholinergic tone (mimicked by atropine) is greatest at low temperatures, and that the relative capacity for increase of heart rate by maximal adrenergic stimulation is greatest at high temperatures. In this context, the data provide qualified support for the original hypothesis (see Introduction) that a reduction in vagal cholinergic activity is relatively more important in tachycardia

at low temperatures, and that adrenergic stimulation becomes more important at higher temperatures.

However, considerable care must be exercised in interpreting the present results. The data only indicate the maximum capability for increase of heart rate by different mechanisms, not the extent to which the animal endogenously uses these mechanisms during stress or exercise. Furthermore, the adrenaline results show the maximum extent to which heart rate can be increased over and above levels achieved by vagal cholinergic withdrawal (i.e. atropinization). It would be interesting to know the extent of endogenous adrenergic stimulation at rest, and the effects of exogenous adrenergic stimulation in the absence of changes in endogenous cholinergic tone. Unfortunately, the measurement of such effects by an *in vivo* pharmacological experiment on an intact animal appears most difficult. In the absence of atropine, exogenous adrenaline causes a pronounced reflex bradycardia (barostatic reflex) due to the accompanying blood pressure rise (Randall and Stevens 1967; Stevens *et al.* 1972; Helgason and Nilsson 1973; Wood 1974). The use of β-adrenoreceptor blocking agents (e.g. propranolol) to block endogenous adrenergic input (cf. Wahlqvist and Nilsson 1977) is of little value since effective blocking doses have nonspecific effects on the heart (Bennion 1968; Wood 1974).

Our findings of significant vagal tone in resting trout at higher temperatures (12 and 20°C; Tables 2, 3) conflicts with previous studies which reported it absent (Randall and Smith 1967; Stevens and Randall 1967; Priede 1974; Wood 1974). This difference may reflect an absence of truly resting conditions in these other investigations. We have noted that if trout are stressed by handling, there occurs an initial bradycardia followed by a prolonged tachycardia associated with a reduction of vagal tone.

In an entirely different sense, the present results for atropine (Fig. 1A, Tables 2, 3) are also opposite to those of Taylor *et al.* (1977) on the dogfish *Scyliorhinus canicula*. In this elasmobranch, the extent of the atropine effect (i.e. vagal cholinergic tone) became progressively greater on both absolute and relative bases as the temperature increased, the latter in direct contrast with the situation in the trout (Tables 2, 3). However, despite these differences in resting vagal cholinergic tone, the influence of temperature on the bradycardia response to hypoxia appears similar in the trout and the dogfish. In both species, the hypoxic bradycardia is clearly a vagal cholinergic phenomenon (Randall and Smith 1967; Taylor *et al.* 1977). In the dogfish, bradycardia during severe hypoxia is negligible at 7°C and becomes progressively greater at 12 and 17°C, so that at extremely low oxygen tension ($P_{IO2} \approx 30$ torr; 1 torr = 133.322 Pa), heart rates are identical at the three temperatures (Butler and Taylor 1975; Taylor *et al.* 1977). Figure 3 presents previously unpublished results from Wood (1968) on heart rate changes during progressive hypoxia at 5 and 15°C in *Salmo gairdneri*. The trends are the same as in the dogfish: a profound bradycardia at 15°C, negligible bradycardia at 5°C, and almost identical heart rates under severe hypoxia (P_{IO2} = 40 torr) at the two temperatures. (Smith and Jones (1978) have recently presented similar, though not quite identical, findings on trout at 7° and 16°C.) This superficial similarity between the trout and the dogfish must belie very different mechanisms. In the resting trout at low temperature, vagal cholinergic tone is probably close to maximum so that hypoxia has little additional effect. However, in the dogfish at low temperature, resting tone is negligible yet no tone is invoked during severe hypoxia. At higher temperatures in the trout, resting tone is reduced and therefore more tone can be recruited during hypoxia. In contrast resting tone is increased in the dogfish at high temperatures yet even more is implemented during hypoxia. The reason for these differences is unclear, but may reflect the effective absence of sympathetic input to heart rate control in elasmobranchs (Short *et al.* 1977).

In contrast to the findings of Davis (1968) on the sockeye salmon (*Oncorhynchus nerka*), resting DA blood pressure was unaffected by temperature

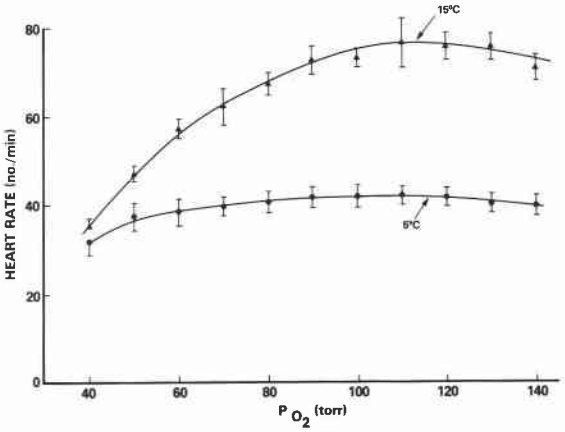

FIG. 3. The influence of progressive environmental hypoxia on heart rate in rainbow trout at 5°C and 15°C (means ± 1 SE). N = 14–22 at 15°C; N = 10 at 5°C. Data from Wood (1968); methods were similar to those of the present study.

(Figs. 1B, 2B). The absence of an atropine effect on mean pressure (Figs. 1B, 2B) indicates a lack of significant muscarinic cholinergic tone in the systematic circulation. This agrees with our previous finding that only nicotinic cholinergic receptors occur in the systemic vascular bed of the trout (Wood 1977). The marked pressor response to adrenaline (Figs. 1B, 2B) has been analyzed in detail by Wood and Shelton (unpublished data). The most important factor in its genesis is an increase in systemic vascular resistance mediated by α-adrenergic receptors, but a stimulation of cardiac output and a decrease in branchial vascular resistance, both mediated by β-adrenergic receptors, also make significant contributions. In view of this complexity, it is impossible to explain the depressant effect of high temperature (Fig. 1B) without further work. The lower blood pressures in anaemic fish (Fig. 2B) can be attributed to both a reduced blood viscosity and a systemic vasodilation (Cameron and Davis 1970; Wood *et al.* 1979).

Anaemia caused tachycardia at all three temperatures in the present study (Fig. 2A, Table 2), thereby confirming and extending the findings of Cameron and Davis (1970) at 12°C. Holeton (1971, 1977) has reported tachycardia at 5°C during hypoxaemia induced by carbon monoxide. The current results clearly demonstrate that the major mechanism by which this cardioacceleration is accomplished at all three temperatures is a complete removal of vagal cholinergic tone; the contribution of adrenergic mechanisms, if any, appears very small (Fig. 2A, Table 2). Holeton (1977) has argued that this increase in heart rate must be mediated through an internal hypoxaemia receptor different from the external hypoxia receptors on the first branchial arches (Daxboeck and Holeton 1978; Smith and Jones 1978) because stimulation of the latter promotes bradycardia. It is interesting that both mechanisms utilize the same efferent pathway, the parasympathetic vagus, to induce these opposite effects.

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