J. exp. Biol. (1977), 69, 247–260
With 4 figures
Printed in Great Britain

THE OXYGEN TRANSPORT SYSTEM IN TROUT (*SALMO GAIRDNERI*) DURING SUSTAINED EXERCISE

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(*Received* 16 *February* 1977)

SUMMARY

1. The capabilities of the oxygen transport system of rainbow trout in supplying the increased oxygen demands in exercise, in a water tunnel at 9–10·5 °C, have been investigated by increasing the velocity of water flow, with a 1 h period between increments, up to the maximum swimming speed (critical velocity, U_{crit}).

2. At U_{crit}, $\dot{M}_{O_2}$ was elevated above the resting level by 7·5 times. The logarithm of $\dot{M}_{O_2}$ was linearly related to the swimming speed expressed as a proportion of U_{crit}. $\dot{V}_g$ increased in almost direct proportion to the increase in $\dot{M}_{O_2}$.

3. Heart rate rose slightly at half U_{crit} and reached a maximum, 1·6 times the resting rate, as U_{crit} was approached. Ventral and dorsal aortic mean blood pressures rose by 60% and 20% respectively at U_{crit} while their pulse pressures doubled. Central venous pressure was virtually unchanged.

4. P_{a,O_2} fell slightly during exercise but C_{a,O_2} was unaffected. On the other hand $P_{\bar{v},O_2}$ halved and $C_{\bar{v},O_2}$ fell from 3·17 (S.E. = 0·3) to 0·6 (S.E. = 0·7) mmol/l. Cardiac output increased by about 3 times resting values.

5. The results are discussed and an attempt is made to estimate the maximum capabilities of the components of the oxygen transport system in sustained exercise.

INTRODUCTION

When in a steady state the oxygen uptake of an animal is the result of the interplay of a large number of components which determine oxygen transport from the respiratory medium to the sites of intracellular utilization. In salmonid fishes the oxygen transport system may, in response to demand, increase its delivery of oxygen by 5–15 times the 'basal' level (standard metabolic rate), depending on the size of the animals and/or temperature (Brett, 1964, 1965, 1972). Major adjustments must be made by the oxygen transport system to achieve these increases, but studies have not made clear the relative contributions of the various components of the system. For instance, utilization of oxygen from water flowing over the gills may reach 80% in resting fish while in active animals values of 10–30% are reported

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(Shelton, 1970). Therefore, to increase oxygen uptake 10 times in the face of fall in utilization demands perhaps a 20–30 times change in gill ventilation. However, the maximum increase in ventilation volume, provoked by exercise, hypoxia, or CO_2, would appear to be in the range of 10–15 times the resting level (Holeton & Randall, 1967; Randall & Jones, 1973). The cardiovascular adjustment may be achieved by increases in heart rate and stroke volume alone, for although heart rate has a maximum exercise capability of only 2 times the resting rate (Sutterlin, 1969; Priede, 1974) stroke volume can increase by at least 4–5 times in response to moderate exercise (Stevens & Randall, 1967*a*, *b*) or anaemia (Cameron & Davis, 1970). On the other hand, accumulation of lactic acid or CO_2 in the blood during exercise might be expected to affect both the loading and unloading of oxygen due to a combination of the Bohr and Root effects (Randall, 1970). Since venous oxygen contents in trout are low (Itazawa, 1970) and appear to remain constant during exercise (Stevens & Randall, 1967*b*), then any restriction in oxygen haemoglobin affinity due to the reduction in blood pH which accompanies severe exercise (Black, 1957, 1958; Black *et al.* 1959; Stevens & Black, 1966) would be expected to restrict the animal's capability for exercise. Basu (1959) has reported that moderate levels of CO_2 in the water reduce the exercise capability of trout, a result he attributed to the effect of CO_2 on oxygen-haemoglobin affinity.

The purpose of the present study was to dermine the capabilities of the oxygen transport system in trout (*Salmo gairdneri*) during steady-state exercise, at different swimming speeds, with a 1 h period between each increment in velocity, up to the maximum swimming speed (critical velocity, U_{crit}). Furthermore, since all cardiovascular and respiratory variables contributing to the oxygen transport system were monitored, it was hoped that these experiments might throw some light on the potential maximum oxygen consumption capability of these fishes.

MATERIALS AND METHODS

The trout used in this study were barren females, 40–53 cm long, weighing 0·9–1·5 kg, all from a common stock. Fish were continuously exercised for a minimum of 14 days by being forced to swim against a water current (average velocity 20 cm/s) produced by a pump in the holding tank. Fish were fed daily with trout pellets or crabmeat. Holding and experimental water temperatures were in the range of 9–10·5 °C.

Surgical procedures

A fish was anaesthetized in a bucket of MS222 solution (1/15000, w/w), weighed, measured, and placed on an operating table similar to that described by Smith & Bell (1964). Flow of water and anaesthetic (MS222, 1/20000, w/w) was maintained over the gills during all surgical procedures. Cannulae for blood sampling were placed in the dorsal aorta, ventral aorta and common cardinal vein. The dorsal aorta was cannulated as described by Smith & Bell (1964) using a 45 cm length of PE 60 tubing terminating in a 1 cm section of the end of a Huber pointed 21 G needle. Ventral aortic cannulation was accomplished using a cannula similar to the one for the dorsal aorta except that the needle was 2 cm long and bent at a 60° angle 6 m

[illegible]m the tip. This cannula was inserted into the ventral aorta through the tongue [illegible] the level of the third gill arch. The ventral aortic cannula was firmly sutured to the tongue and led straight out of the mouth.

The right common cardinal vein cannula consisted of a 3 cm section of Huber pointed 18 G needle bent at 90° 18 mm from the tip and attached to a 45 cm length of PE 160 tubing. This cannula was inserted perpendicularly to the right side of the fish at a point about 3 mm posterior to the cliethrum and 3 mm ventral to the lateral line. The cannula was then turned so that the open end in the vein was directed ventrally before being sutured in place. All blood vessel cannulae were filled with heparinized (10 i.u./ml) Courtland saline (Wolf, 1963) and plugged with pieces of wire of the appropriate diameter. Wires for e.c.g. recording were inserted and sutured in place; one ventrally, immediately posterior to the middle of the pectoral girdle, and one medio-dorsally, posterior to the operculum.

To measure ventilation volume a skirt was made of the wrist portion of a disposable surgical glove and placed over the head of the fish to lie in contact with the body between the mouth and opercular openings. After proper orientation, the anterior edge of the glove was sutured to the skin around the lower jaw and dorsally over the operculae, so that the anterior edge adhered tightly to the head of the fish whereas the posterior part was loose to allow free movement of the opercula. The excess membrane was then trimmed off, leaving a length of free membrane which extended about 2 cm posterior to the opercula. A water sampling cannula (PE 60, 45 cm long) was placed with its open end directed ventro-medially anterior to the pectoral girdle (under the skirt) and sutured in place, being led dorsally and posteriorly past the left pectoral fin and sutured to the skin dorsally. To measure breathing rate a buccal cannula (PE 60, 45 cm long) was inserted as described by Saunders (1961).

After completion of surgical procedures the fish was placed in a Brett (1964) type water tunnel and allowed 18–24 h to recover from the anaesthetic and surgery. During this time water velocity in the water tunnel was about 10 cm/s and oxygen tension and temperature were held constant by continual replacement of tunnel water with fresh dechlorinated water.

Experimental protocol

Sanborn 267B transducers were used for dorsal and ventral aortic blood pressure measurements and a Statham P23v transducer was used to measure common cardinal vein blood pressure. Buccal pressures were measured with a Sanborn 267B transducer. Water velocity in the water tunnel was recorded by monitoring the pressure change across one of the contraction cones in the tunnel using a Sanborn 268B differential pressure transducer and the output was monitored on a strip chart recorder along with the other variables.

The transducers and cannulae used for blood pressure recording were tested by the free vibration method described by McDonald (1974). By this method, the resonant frequency of a Sanborn 267B transducer and cannula was found to be 15 Hz with damping 35% of critical. The Statham transducer and cannula used for common cardinal vein pressure measurement had a natural frequency of 10 Hz and damping was 36% of critical. Pressure transducers used for dorsal and ventral [illegible]tic pressures were calibrated against a pressure head of 40 cm of saline (zero

being the water level in the tunnel). The Statham P23v transducer, used for comm[illegible] cardinal vein blood pressure, was calibrated against a head of 15 cm of saline, [illegible] was the transducer used for buccal pressure recording. All calibrations were checked periodically. An analogue ratemeter was used (triggered by the QRS wave of the e.c.g.) to obtain beat to beat heart rate. Recording of all electronic signals was done on either a Brush model 220 two channel pen recorder or a Techni-rite 8 channel recorder, model number TR-888; both recorders writing on rectilinear co-ordinates.

Oxygen tensions were measured with a Radiometer type E5046 electrode in a type D616 thermostatically controlled cell. The zero setting was established using a 0·01 M-Na_2BO_4 solution with 5 mg/20 ml Na_2SO_3 added twice daily. The span was set before each sample, or group of samples in the case of duplicates, using air equilibrated water at the ambient water temperature. Both calibrations were reproducible to 0·067 kPa over the period of measurements. Measurement of pH was done with a Radiometer type G297-G2 blood pH electrode calibrated using precision buffers (type S1500 and S1510). One or both calibrations were done before each blood sample depending on the stability of the electrode. Readout from the oxygen and pH electrodes was on a Radiometer Acid-Base Analyzer.

Oxygen content of arterial and venous blood was determined by the method of Tucker (1967). All oxygen content determinations were done at 32 °C as described by Tucker (1967) except that the blood volumes were larger (50 μl for venous and 25 μl for arterial blood) because the chamber was larger. The blood samples were pipetted into the chamber within 30 s of sampling. Arterial and venous samples were done serially 5–10 min apart.

The blood remaining in the microburette after the oxygen content determination was transferred into three 20 μl micropipettes which were sealed with seal-ease (Clay Adams, Parsippany, N.J., U.S.A.). The excess length of pipette was cut off, the samples were spun in a commercial micro-haematocrit centrifuge and haematocrit was recorded. After haematocrit determination the samples were labelled and frozen for blood iron determination. The micropipettes, containing 20 μl blood samples saved from haematocrit determinations, were rinsed on the outside and the pipettes broken up inside clean borosilicate glass scintillation vials. After an overnight drying period at 80 °C the vials and contents were placed in a muffle furnace and ashed at 680 °C for a minimum of 8 h or until only a white powder remained in the pipette sections in the vials. Upon cooling to room temperature the contents were dissolved in 0·2 N-HCl. Recovery of sample was checked by an identical treatment of standard solutions in the pipettes, and found to be 100%. The samples were analysed on a Tectron model AA120 atomic absorption spectrophotometer using a wavelength of 248·3 nm from a Varian FeCo hollow cathode lamp against standards ranging from 0 to 10 mg Fe/l. The standard was made by dissolving 0·1 g of Iron Powder (analytical grade) in a small quantity of Analar HCl and diluting with 0·2 N-HCl to the appropriate concentrations.

In an initial series of experiments on cardiovascular adjustments to swimming, attempts were made to monitor heart rate, ventilation rate and blood pressure in the dorsal aorta, ventral aorta, and common cardinal vein, along with the velocity of the water in the water tunnel. All variables were not necessarily measured on each fish, i.e. many heart rate experiments were done on fish carrying only e[illegible]

electrodes. After recording the different variables at rest the water velocity was increased abruptly by about one-sixth of the expected critical velocity and maintained at that velocity for 60 min. The variables were recorded after 0·5, 1, 3, 10, 15, 30, 45 and 60 min, at which time the velocity was increased again by about one-sixth of the expected critical velocity and the measurements repeated. In later experiments, investigating the blood gases as well as cardiovascular variables, measurements of heart rate, arterial and venous oxygen content, venous and arterial oxygen tension, venous and arterial haematocrit, blood pressure, arterial and venous pH, oxygen tension of inhaled water, and oxygen consumption, were attempted on each individual. The fish was checked to make certain that it was swimming constantly and not tangling on the cannulae and wires. After swimming for 50 min of a test increment, blood sampling commenced and all the variables were measured before the swimming speed was further increased after 60 min at each speed. Only when it was suspected that the fish was about to fatigue was the sampling procedure carried out before the 50 min time period. To determine the effect of instrumentation drag on the swimming speed some uninstrumented fish were swum in the same manner as instrumented fish. U_{crit} was estimated, in all cases, by the method of Brett (1964), on allowance being made for partial blocking when the fish cross-section was greater than 10% of the tube area (Webb, 1971*a*).

$\dot{N}_{O_2}$ was determined for virtually every fish used in this study. Before each oxygen consumption determination the water tunnel was checked for trapped air bubbles which were then removed. Oxygen consumption was determined by closing the system (shutting off the input of fresh water) and measuring the rate of change of the oxygen tension of the water circulating in the water tunnel. A 5 ml water sample was taken from the water tunnel at the same time that the inflowing water was shut off, and another sample at the end of a suitable time period. The length of time between the samples was determined by the size of the fish and intensity of swimming, but usually, at a given velocity, $\dot{N}_{O_2}$ was determined as close to the end of the one hour test period as was possible. For instance in experiments in which cardiovascular and blood gas values were also obtained, determination of $\dot{N}_{O_2}$ was not attempted until after these variables had been recorded at 50 min, so at most there was only a 10 min period between water samples. However, in these experiments the largest fish were used so accurate determinations of $\dot{N}_{O_2}$ was still possible. In no experiment was the oxygen tension in the water allowed to fall by more than 2 kPa when measuring $\dot{N}_{O_2}$. The oxygen tension of the water samples was measured in duplicate immediately after each was taken. Oxygen consumption was then calculated using the formula

$$\dot{N}_{O_2} = \frac{\Delta P_{O_2} . V . \alpha}{t . 0{\cdot}0224},$$

where $\dot{N}_{O_2}$ = oxygen consumption (μmol/min), ΔP_{O_2} = change in oxygen tension (kPa), V = volume (34·5 l), α = solubility coefficient of O_2 in H_2O at the experimental temperature (ml O_2 (STPD)/l H_2O.kPa), t = time (min) and 0·0224 converts ml O_2 (STPD) to μmol.

In some experiments oxygen tension of inhaled and exhaled water (samples taken from the cannula located under the opercular skirt) was measured throughout exercise period. In particular these variables were measured just before the water

tunnel was closed to record $\dot{N}_{O_2}$ towards the end of the hour spent swimming at particular velocity. These values were used to calculate % utilization of oxygen from water flowing over the gills (% $U = 100 \times [P_{I,O_2} - P_{E,O_2}/P_{I,O_2}]$) and ventilation volume using the Fick equation as follows

$$\dot{V}_g = \dot{N}_{O_2}.0{\cdot}0224/(P_{I,O_2} - P_{E,O_2}).\alpha,$$

where $\dot{N}_{O_2}$ = oxygen consumption (μmol/min), $\dot{V}_g$ = ventilation volume (ml H_2O/min), P_{I,O_2} = oxygen tension of inhaled water (kPa), P_{E,O_2} = oxygen tension of exhaled water (kPa), α = solubility coefficient of O_2 in water at the experimental temperature (ml O_2 (STPD)/l H_2O.kPa), and 0·0224 converts μmol to ml O_2 (STPD).

RESULTS

Determination of every variable that was required for a complete description of the oxygen transport system in exercise was not made on each fish. The experimental plan called for an attempt to be made to increase the number of variables recorded in successive experiments and to relate these to $\dot{N}_{O_2}$. Unfortunately, increasing complexity of the experiments increased the number of failed attempts to measure a particular variable. Consequently, in the results these somewhat fragmentary data have been pooled in sections on $\dot{N}_{O_2}$ (*a*) and cardiovascular adjustments to exercise (*c*). $\dot{N}_{O_2}$ was recorded for nearly every fish used, so pooled data contain measurements from partially up to fully instrumented fish. Another section (*b*) reports determinations of $\dot{N}_{O_2}$, ventilation rate and volume during exercise in five fish. These $\dot{N}_{O_2}$ measurements are also included in section (*a*). The final section (*d*) contains data from six animals in nine swimming trials which yielded the most complete sets of information on the function of the oxygen transport system during exercise and all data are exclusive to this section.

(*a*) *Oxygen uptake in exercise.* Determination of $\dot{N}_{O_2}$ was made on 25 animals. Resting oxygen uptake, obtained in the respirometer with water velocity at the lowest speed, was 26 μmol/kg.min (S.E. = 0·45, n = 31) and $\dot{N}_{O_2}$ increased as the swimming speed (U) increased and attained a level of 194 μmol/kg.min (S.E. = 3, n = 13) at velocities which caused fatigue in less than the 1 h test period. Although Brett (1964) has shown that $\dot{N}_{O_2}$ increases exponentially with U the present data yielded a poor relationship between $\dot{N}_{O_2}$ and speed. We felt that this was because many fish carried instrumentation which increased their drag and therefore reduced U and, in fact, normalization of swimming speed by dividing U at a particular $\dot{N}_{O_2}$ by the maximum U attained by that fish in the experiment (U_{crit}; Brett, 1964 method) yielded a good exponential relation between $\dot{N}_{O_2}$ and % U_{crit} with little scatter around the 'best straight line' (Fig. 1). Only 13 determinations of $\dot{N}_{O_2}$ at maximum swimming speed were obtained but these were remarkably constant and independent of the actual swimming speed. The relationship between U and length (L) in a series of experiments on uninstrumented fish was $U = 12{\cdot}4\ L^{0{\cdot}55}$ with the maximum speed being of the order of 2 L/s in relative terms or 80–90 cm/s in absolute terms, while the instrumented fish had U_{crits} between 0·5 L/s and 1·5 L/s depending on the amount of instrumentation carried and the size of the fish.

(*b*) *Ventilatory adjustments to exercise.* Ventilation rate was extremely variab

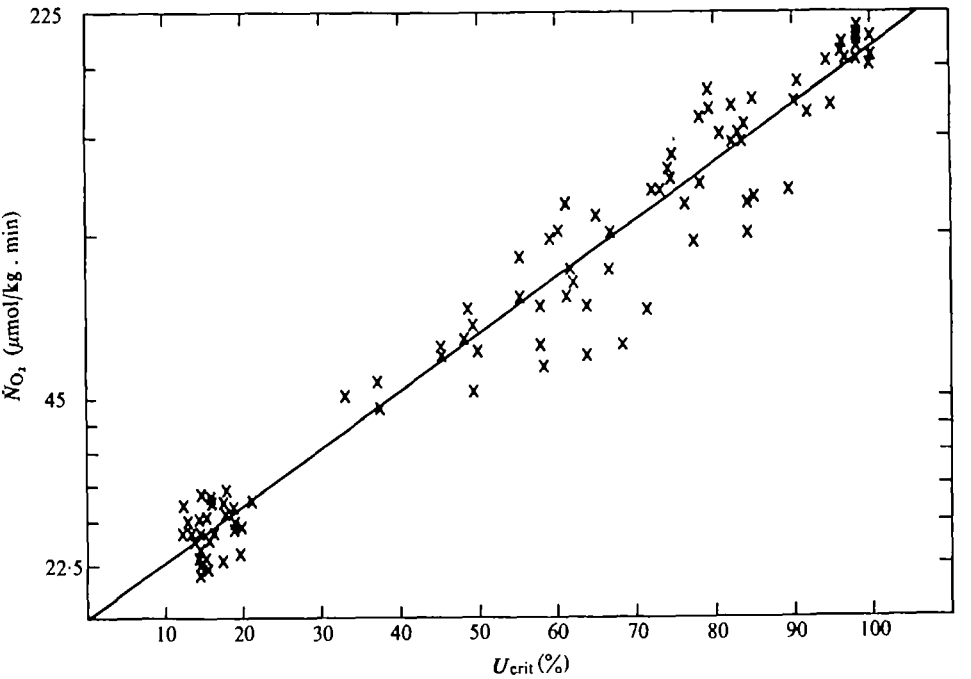

Fig. 1. The relationship between oxygen consumption and swimming speed expressed as per cent of each individual's critical velocity. Points at velocities of less than 25% U_{crit} are for animals at rest. One hundred and one determinations are presented from 25 individual trout and the regression equation is $\log y = 1{\cdot}27 + 0{\cdot}01\,x$.

even at rest, and this variability tended to increase with exercise. The main trend was for the rate to increase at the start of each increment in U and then to decline over the next 15–30 min. During swimming both ventilation volume ($\dot{V}_g$) and $\dot{N}_{O_2}$ increased. The mean resting $\dot{V}_g$ was 211·4 ml/kg.min (S.E. = 5·8, n = 5) which increased with $\dot{N}_{O_2}$ and, in two animals, reached 1700 ml/kg.min at maximum $\dot{N}_{O_2}$. In these experiments the increase in $\dot{N}_{O_2}$ was slightly less than the change in $\dot{V}_g$ so that a double log plot of $\dot{N}_{O_2}$ on $\dot{V}_g$ gave a line with a slope of 0·85, implying that over the range of $\dot{V}_g$ investigated there was a slight decrease in utilization of oxygen from the water flowing over the gills with exercise (Fig. 2). However, determinations of utilization alone from measurement of P_{I,O_2} and P_{E,O_2} over the whole range of $\dot{N}_{O_2}$ failed to confirm this and yielded a virtually constant value with the mean of all 74 determinations being 33·0% (S.E. = 0·5%).

(*c*) *Cardiovascular adjustments to exercise.* The mean heart rate in resting trout was 31·75 beats/min (S.E. = 1, n = 32) and rose, as swimming speed increased, to attain a maximum at 90% U_{crit} of 52 beats/min (S.E. = 1, n = 28). From 50 to 90% U_{crit} heart rate increased linearly with U_{crit}, rising less rapidly below 50%, and hardly at all above 90% (Fig. 3). The values given above are for the last 30 min of an hour exercise period when the animals were assumed to be in a steady state; somewhat different values were obtained in the early part of the exercise period. As the water velocity was increased, at the start of exercise or when imposing an increment in swimming velocity, the animals displayed bradycardia which was

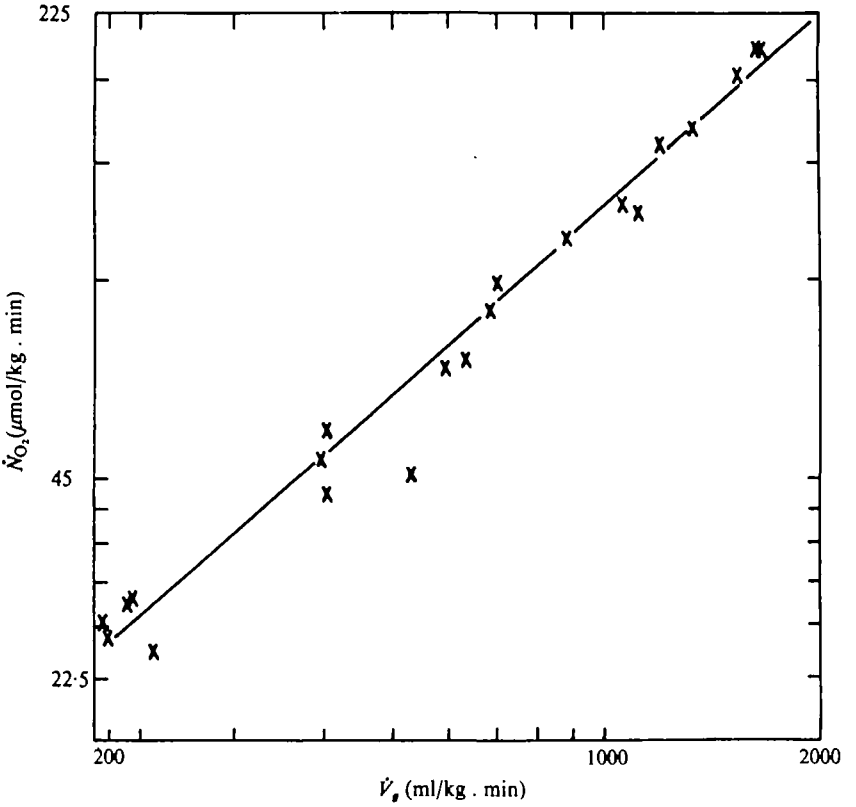

Fig. 2. The relationship between oxygen consumption ($\dot{N}_{O_2}$) and ventilation volume ($\dot{V}_g$) at rest and during exercise of increased intensity up to and including U_{crit}. Twenty-one determinations were done on five trout.

immediately followed by tachycardia. On some occasions several cycles of bradycardia and tachycardia were observed in the first few seconds after a velocity increment. This apparent hunting disappeared after about 30 s, whereas the heart rate continued to increase and reached a maximum value for that particular swimming speed after 3–15 min.

Ventral and dorsal aortic blood pressures also showed transient changes following an increase in swimming speed (Fig. 4). Systolic and diastolic pressures increased in both aortae about 5 s after the water velocity was increased and peaked between 5 and 10 min (Fig. 4). After this period the pressures fell and were reasonably stable over the last 30 min of the exercise period (Fig. 4). However, as can be seen from Fig. 4, the pressures were elevated from their initial values at the end of each exercise period so that, in the steady state, pressure increased as U_{crit} was approached (Fig. 3).

Mean pressure in the ventral aorta at rest was 5·17 kPa (S.E. = 0·48, n = 7) and rose to a mean of 8·22 kPa (S.E. = 0·93, n = 7) at 80–100% of critical velocity. The corresponding pulse pressures were 1·53 kPa and 3·47 kPa. Blood pressure in the dorsal aorta showed a smaller increase over the same speed range, from 4·13 kPa (S.E. = 0·5, n = 8) to 4·93 kPa (S.E. = 0·6, n = 6) while pulse pressure at rest was 0·77 kPa and rose to 1·33 kPa during exercise (Fig. 3). Blood pressure in th

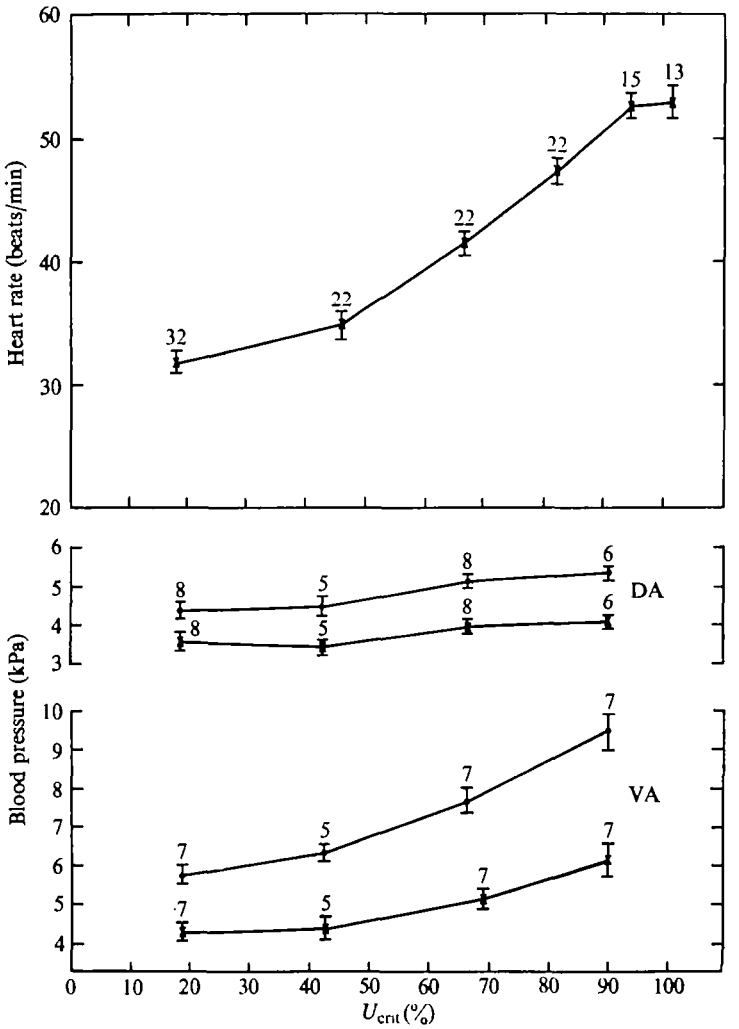

Fig. 3. Heart rate, diastolic (X) and systolic (●) blood pressures in both the dorsal aorta (DA) and ventral aorta (VA) of trout at rest and during swimming (expressed as a per cent of each individual's critical velocity). Values at less than 25 % U_{crit} are for animals which were resting. The points are means of individual determinations, the vertical bars denote one standard error, and the numbers above the points indicate the numbers of determinations on 29 animals.

right common cardinal vein at rest was 0·19 kPa (S.E. = 0·04, n = 4), and increased by an insignificant amount to 0·25 kPa (S.E. = 0·05, n = 4) at critical velocity. No increases in venous pressure were observed at intermediate speeds.

Following fatigue of an animal, blood pressure remained constant for about 2 min and then fell gradually, reaching a resting value after about 2 h. On the other hand, heart rate remained at the maximum rate for 10–30 min before starting to decline to reach the resting level in 12–18 h.

(*d*) *Oxygen transport in exercise.* The mean values of resting and maximum $\dot{M}_{O_2}$ for these six cannulated fish (Table 1) were almost identical to the mean values for [redacted]ner fish, given in section (*a*), although U_{crit} was considerably lower (0·5–1 L/s in

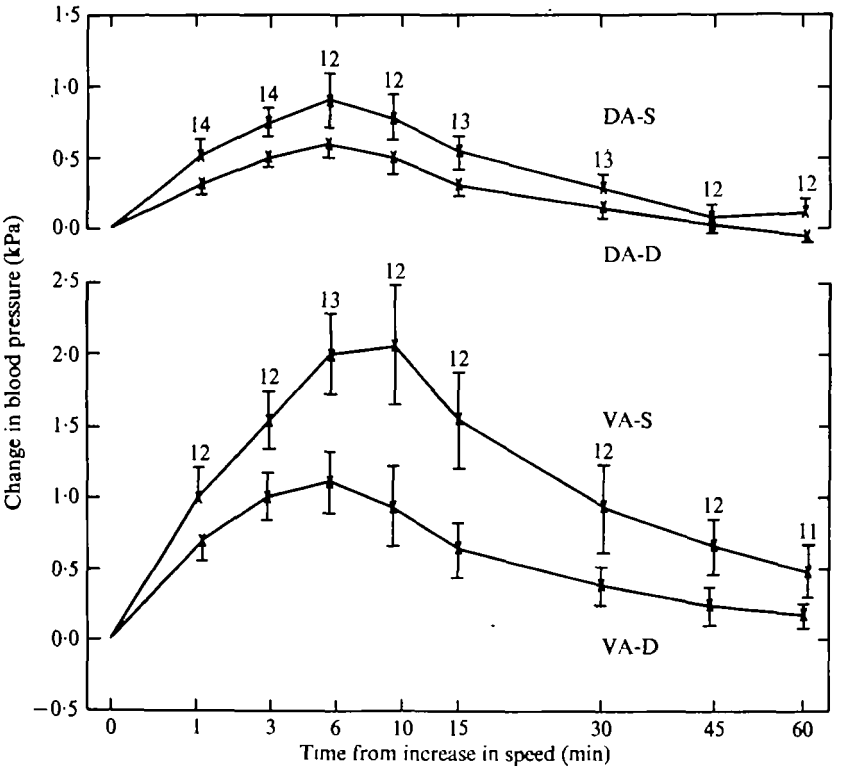

Fig. 4. Change in systolic (S) and diastolic (D) blood pressure in the dorsal aorta (DA) and ventral aorta (VA) following an increase in swimming speed. Xs are means of (pressure at time t_n) − (pressure at time t_0), pressure at t_0 being the pressure immediately before a speed increase was imposed. Vertical bars denote one standard error and numbers above the points represent the number of determinations on five individuals.

relative terms or 25–50 cm/s in absolute terms). Therefore, $\dot{M}_{O_2}$ increased by 7·75 times (Table 1).

P_{a,O_2} at rest in these animals was 18·27 kPa (S.E. = 0·560) (Table 1), which was almost identical to the mean value of P_{a,O_2} obtained from 21 other resting fish in this study (17·92 kPa, S.E. = 0·280). At this P_{a,O_2}, C_{a,O_2} was 4·64 mmol/l (S.E. = 0·2) which corresponded to 97 % (S.E. = 1·3 %) oxygen saturation (Table 1), as determined from blood iron concentrations. P_{a,O_2} fell with the onset of exercise and remained about 1·33 kPa below the resting level throughout, although mean arterial oxygen saturation was maintained at the resting level (Table 1). On the other hand, $P_{\bar{v},O_2}$ was halved at the highest sustained swimming speeds and this was associated with a fall in $C_{\bar{v},O_2}$ from 3·17 (S.E. = 0·3) to 0·6 (S.E. = 0·18) mmol/l (Table 1). As C_{a,O_2} remained between 4 and 4·7 mmol/l throughout exercise then the A–V_{O_2} difference increased from 1·47 (S.E. = 0·12) to 3·7 (S.E. = 0·22) mmol/l, a change of 2·5 times. An *in vivo* oxygen dissociation curve derived from plotting % oxygen saturation (obtained from oxygen content and iron concentrations) against the respective arterial and venous oxygen tensions yielded a P_{50} of 3·2 kPa on the sigmoidal curve. Arterial and venous pH appeared to be maintained up to speeds of 92 % U_{crit}, above which they both tended to decline (Table 1). Venous haematocrit was above arte-

Table 1. *An evaluation of the oxygen transport system in trout during exercise*

(Data obtained from six trout and n = the number of determinations. All values given as means ± S.E.M. except pH_a and $pH_{\bar{v}}$ when the values given are means + and − S.E.M.*)

Swimming speed U	Heart rate HR	Arterial O_2 content C_{a,O_2}	Venous O_2 content† $C_{\bar{v},O_2}$	Arterial O_2 tension P_{a,O_2}	Venous O_2 tension† $P_{\bar{v},O_2}$	Haematocrit		pH_a	$pH_{\bar{v}}$	Arterial − venous O_2 content $A-\dot{V}O_2$	Oxygen consumption $\dot{N}_{O_2}$	Cardiac output $\dot{Q}$	Stroke volume SV	Inspired water O_2 tension P_{I,O_2}	Arterial O_2 saturation S_{a,O_2}
						Hct_a	$Hct_{\bar{v}}$								
% U_{crit}	/min	mmol/l	mmol/l	kPa	kPa	%	%			mmol/l	μmol/kg.min	ml/kg.min	ml/kg	kPa	%
Rest	37·8 ±1·5 $n=9$	4·64 ±0·22 $n=9$	3·17 ±0·3 $n=9$	18·27 ±0·560 $n=8$	4·43 ±0·800 $n=8$	22·6 ±1·0 $n=9$	24·2 ±1·8 $n=8$	7·932 +7·991 −7·879 $n=5$	7·959 +8·025 −7·902 $n=4$	1·47 ±0·12 $n=9$	25 ±0·9 $n=9$	17·6 ±1·1 $n=9$	0·46 ±0·02 $n=9$	20·39 ±0·253 $n=8$	97·0 ±1·3 $n=9$
41–63	42·7 ±3·18 $n=3$	4·375 ±0·33 $n=3$	1·96 ±0·35 $n=3$	16·47 ±1·00 $n=2$	— —	22·7 ±1·4 $n=3$	24·45 ±1·05 $n=2$	— —	— —	2·4 ±0·04 $n=3$	67·8 ±10·7 $n=3$	28·4 ±5·0 $n=3$	0·62 ±0·8 $n=3$	20·27 ±0·308 $n=3$	96·0 ±5·00 $n=3$
70–78	49·0 ±1·00 $n=5$	4·03 ±0·22 $n=5$	1·5 ±0·18 $n=5$	16·40 ±0·560 $n=4$	3·13 ±0·280 $n=4$	20·34 ±1·4 $n=5$	21·85 ±2·4 $n=4$	7·924 +8·046 −7·829 $n=3$	7·988 +8·081 −7·911 $n=3$	2·5 ±0·26 $n=5$	84·8 ±12·00 $n=5$	34·8 ±4·8 $n=5$	0·7 ±0·09 $n=5$	20·77 ±0·127 $n=5$	98·75 ±1·00 $n=5$
81–91	51·3 ±4·6 $n=3$	4·55 ±0·58 $n=3$	1·29 ±0·66 $n=3$	17·07 ±0·667 $n=3$	3·91 ±0·827 $n=3$	22·5 ±1·35 $n=3$	25·8 ±0·9 $n=3$	7·859 +7·970 −7·770 $n=2$	7·883 +7·950 −7·825 $n=2$	3·26 ±0·22 $n=3$	139·3 ±17·00 $n=3$	42·9 ±5·4 $n=3$	0·86 ±0·16 $n=3$	19·56 ±0·089 $n=3$	99·7 ±0·67 $n=3$
Maximum 92–100	51·4 ±2·48 $n=4$	4·33 ±0·3 $n=4$	0·6 ±0·18 $n=4$	16·80 ±0·720 $n=4$	2·13 ±0·280 $n=4$	25·7 ±0·8 $n=4$	27·4 ±1·2 $n=4$	7·610 +7·620 −7·600 $n=2$	7·548 +7·630 −7·480 $n=2$	3·7 ±0·22 $n=4$	193·7 ±7·6 $n=4$	52·6 ±2·2 $n=4$	1·03 ±0·07 $n=4$	20·24 ±0·347 $n=4$	98·5 ±0·87 $n=4$

* Mean and standard errors were calculated on hydrogen ion concentration, since an increase of 1 pH unit represents a different number of H^+, than a decrease of 1 pH unit.

† Values obtained from the common cardinal vein and ventral aorta were similar.

haematocrit at rest and at all exercise levels; both tended to increase at the highest swimming speeds although the change from resting values was not significant.

Cardiac output, derived from $\dot{M}_{O_2}$ and the A–V_{O_2} difference, was 17·6 ml/kg.min (S.E. = 1·1) at rest and increased with exercise, reaching a peak value of 52·6 ml/kg.min (S.E. = 2·2) (Table 1). Resting heart rate in these cannulated trout was some 6 beats/min above the mean value reported in section (*c*) above (Fig. 3) but the maximum value obtained was almost identical to that given in section (*c*). The increase in heart rate with exercise was about 1·36 times. Since cardiac output increased some 2·2 times more than heart rate, this represented the degree of change in stroke volume with exercise. The changes in heart rate, stroke output and A–V_{O_2} difference represented an increase in the oxygen transport capabilities of the trout cardiovascular system of some 7·6 times.

DISCUSSION

During sustained exercise, at critical velocity, $\dot{M}_{O_2}$ was some 7·5 times higher than the resting level. This increase in $\dot{M}_{O_2}$ was achieved by means of the following adjustments in the oxygen transport system: (*a*), a linear increase in $\dot{V}_g$ with little change in utilization at the gills; (*b*), a three-fold increase in cardiac output, the main component of this increase being the change in stroke volume; (*c*), increased utilization at the tissues giving a 2·5 times expansion of the A–V_{O_2} difference. However, the maximum weight specific $\dot{M}_{O_2}$ observed in these experiments (194 μmol/kg.min) was considerably below that reported by Rao (1968) and Webb (1971*b*) for smaller trout at 15 °C. For active metabolism of salmonids the weight specific $\dot{M}_{O_2}$ is virtually independent of body weight (Brett, 1965) while Q_{10} over the range from 5° to 15 °C is between 1·8 and 2 (Brett, 1964; Rao, 1968; Brett & Glass, 1973). Hence, the present trout at 15 °C would have had a maximum $\dot{M}_{O_2}$ of about 275 μmol/kg.min, which is not much below values given by Rao (1968) and Webb (1971*b*), and it therefore seems safe to conclude that the animals were forced to attain a level of aerobic energy conversion close to their maximum. Maximum $\dot{M}_{O_2}$ was fairly similar between fish, and independent of U_{crit}, so it is not possible to support Webb's (1971*b*) claim that since work rate is proportional to U_{crit}, a fall in U_{crit} due to increased load will reduce power output of the fish and therefore $\dot{M}_{O_2}$.

An increase in temperature, up to 15 °C at least, promotes an increase in the absolute amount of oxygen transported. Leaving aside the physical effects of temperature change (i.e., on blood and water viscosity, oxygen solubility and diffusion rate) it is interesting to consider what biological compensations are left to the animal to increase oxygen transport. Although maximum heart rate is increased with temperature (Priede, 1974) so too is the basal rate and therefore the scope for change can be little more than two times, while it is unlikely that A–V_{O_2} difference could be expanded substantially over what was obtained in the present experiments. However, stroke volumes two to three times those obtained in the present fish have been reported for trout during hypoxia (Holeton & Randall, 1967) and anaemia (Cameron & Davis, 1970). This degree of change in stroke volume allied with a small increase in heart rate change would give a maximum potential $\dot{M}_{O_2}$ some 3·3 times greater than we recorded. In fact, Webb (1971*a*, *b*) reports two animals in his control grou

achieving active $\dot{N}_{O_2}$ levels of 500 μmol/kg.min, at 15 °C, which is 2·5 times the oxygen transport rate observed in our fish at 10 °C.

What proportion of the increase in $\dot{N}_{O_2}$ actually goes to the locomotory muscles is a matter for speculation. The metabolic cost of circulation, respiration, and osmoregulation must increase with activity (Rao, 1968; Farmer & Beamish, 1969; Jones, 1971*a*, *b*), thereby reducing the proportion of oxygen available for locomotion. In mammals about 40% of the increase in oxygen uptake by muscle during exercise is met by redistribution of blood flow away from other tissues but Stevens (1968) failed to demonstrate any significant changes in the blood content of most organs in fish following exercise, suggesting that shunting of blood away from certain tissues during exercise is insignificant. Consequently, the rise in dorsal aortic blood pressure combined with the fall in peripheral resistance of the systemic circuit from 0·234 kPa.min/ml at rest to 0·094 kPa.min/ml (or 2·5 times) at maximum exercise would be expected to increase flow to all organs. Since gill resistance changed little (rest = 0·059 kPa.min/ml; maximum exercise = 0·062 kPa.min/ml) ventral aortic blood pressure rose substantially more than dorsal aortic pressure. It would seem to be very inefficient to direct an increased blood flow to an organ or tissue not displaying an increase in metabolic demand yet it is difficult to see, for instance, why $\dot{N}_{O_2}$ of the gut should increase in step with metabolic demand of the locomotory muscles. Obviously, this is an area of exercise physiology in fishes which is worthy of further investigation.

Swimming at speeds less than 91% U_{crit} caused little change in pH_a or $pH_{\bar{v}}$ but above this speed pH_a and $pH_{\bar{v}}$ fell markedly. This suggests either respiratory or metabolic acidosis. Measurement of whole blood and plasma CO_2 content was attempted by the method of Cameron (1971) but in our hands this method did not work very well on blood sampled from fish. From those results which were obtained it does not seem likely that a net accumulation of CO_2 is the cause of the change in blood pH. At the fastest swimming speeds animals often swam erratically, symptomatic of white muscle activity, which would be expected to increase the levels of blood lactate and thereby lower blood pH. However, the effects of metabolic acidosis on the oxygen transport system were insignificant since arterial blood remained 98·5% saturated with oxygen at the maximum exercise level.

We are grateful to the Fisheries and Marine Service of Environment Canada for provision of a post-graduate fellowship (J.W.K.) and some operating support. Other funds in support of this project were awarded by the National Research Council of Canada. We also wish to express our thanks to Dr D. J. Randall for lending us the respirometer.

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