WATER FLOW AND GAS EXCHANGE AT THE GILLS OF RAINBOW TROUT, *SALMO GAIRDNERI*

By JOHN C. DAVIS AND JAMES N. CAMERON

Department of Zoology, University of British Columbia, Vancouver 8, B.C., Canada

(Received 10 August 1970)

**INTRODUCTION**

The Fick principle has often been used to estimate the volume flow of water per unit time over the gills of fishes. Application of the Fick principle requires accurate measurement of mean oxygen tensions in water entering and leaving the gills and of total oxygen uptake. Cannulae of various types have frequently been used to sample water entering and leaving the gills (van Dam, 1938; Holeton & Randall, 1967a, b; Randall, Holeton & Stevens, 1967; Saunders, 1961, 1962; Stevens & Randall, 1967a, b). Recent studies have indicated that these cannulation techniques sometimes provide poor estimates of mean expired oxygen tensions (Davis & Watters, 1970; Garey, 1967). Procedures of this type therefore could lead to inaccurate estimates of volume flow of water over the gills. Hence it was necessary to devise another method of measuring ventilation volume in this study.

Variations in ventilation volume influence gas exchange across the gills. Hughes (1966) and Saunders (1961) discussed the effects of high gill water flow, pointing out that anatomical deadspace increases under these conditions since portions of the ventilatory flow are shunted past the tips of adjacent gill filaments. It was a further objective of this study to see how the velocity of water flow over the gills influenced a fish's ability to exchange gases with the water.

**METHODS**

*Explanation of symbols used*

$V_Q$, volume of water passing over the gills/unit time (ventilation volume)

$VR$, ventilation rate in mouth or opercular closures/minute

$V_{sv}$, volume of water pumped over the gills/breathing cycle (ventilatory stroke volume)

$\% U$, percentage of oxygen utilized from inspired water

$V_{Op}$, oxygen uptake/unit time

$P$, partial pressure of gas in mmHg

$\alpha$, solubility coefficient for gas in water or blood

$Q$, cardiac output/unit time

s.d., standard deviation

s.e., standard error of mean

$Hct$, packed cell volume of blood in vol. % (haematocrit)
Experiments were carried out during 1969 at the Vancouver Public Aquarium, Vancouver, B.C. Stocks of rainbow trout, *Salmo gairdneri*, were purchased from the Sun Valley Trout Farm, Coquitlam, B.C., and were held in 250 gallon tanks prior to use. The fish were fed with a mixed diet of Clark's trout pellets and chopped horse heart twice weekly.

Test fish were anaesthetized in a bucket containing 1:10000 MS 222 solution and were transferred to an operating table similar to that described by Smith & Bell (1964). This device was equipped with a pump and nozzles to perfuse the gills with anaesthetic solutions during operations.

A dorsal aortic cannula was implanted in the fish as described by Smith & Bell (1964) and a 40 cm length of number 0 silk was tied through the anterior portion of
Gill water flow in trout

the upper jaw as a restraint upon the fish. Next, a 12 cm circle which included the thumb was cut from a number 8\textfrac{1}{2} latex surgeon's glove. The tip of the thumb was cut to fit the fish's head so that it covered the dorsal aortic cannula at its point of emergence but left the eyes uncovered. The membrane was then stitched around the margin of the fish's mouth with number 000 silk sutures placed 2–3 mm apart. When properly cut and positioned the membrane exerted little tension on the head or jaws and had sufficient slack to permit easy movement of the branchial apparatus. On two of the subjects the membrane was too tight and the fish were unable to open and close their mouths properly. Results from these two fish were discarded.

Fish with attached oral membranes were placed in a Lucite box divided into two chambers by a partition with a hole through it. The membrane was attached to the edge of the hole by a Lucite retaining ring and brass wingnuts. Thus the membrane served as a barrier between the two chambers and separated inspired and expired water. A rectangular Lucite box, open at both ends, supported the fish while its head was anchored to the retaining ring by the string through the upper jaw. Movement was further restricted by a Lucite bar across the retaining ring so that the fish could not swim forward and tear loose from the membrane. When in place the trout could move its head freely about 2 cm in any one direction but could not exert a direct pull on the membrane. The top and sides of the box were covered with black plastic to prevent the fish seeing the investigators.

The drains at either end of the box could be set at various levels by sliding them up and down through rubber stoppers. Following the operation the drain at the head end of the box was raised to provide positive pressure in the buccal cavity and facilitate ventilation during recovery. At that time the membrane could be checked for leakage with dye. When the membrane was properly affixed very little leakage was apparent with a pressure differential of 2–3 cm H$_2$O across the membrane.

The trout were allowed overnight recovery following attachment of the membrane. Then the drains in the box were levelled and the fish was left undisturbed for at least 1 h. Ventilation rate ($VR$), ventilation volume ($V_o$), dorsal aortic oxygen tension ($P_{d(O_4)}$), inspired oxygen tension ($P_{i(O_4)}$), and expired oxygen tension ($P_{e(O_4)}$) were then measured at intervals. Ventilation volume was measured by collecting the water spilling over the rear drain as the fish breathed. Water was collected for 1 min and ventilation rate was determined simultaneously by counting the number of mouth or opercular movements. Often these measurements were carried out on the same individual for a period of 2 or 3 days. In a few cases oxygen-deficient water (56–65 mm $P_{O_4}$) was run into the box and the fish's response was studied.

Oxygen tensions in blood and water were measured with a Radiometer electrode system as described by Cameron & Davis (1970) and Davis & Watters (1970). Oxygen uptake was calculated by the Fick principle using the inspired oxygen tension, the expired oxygen tension from the rectangular body-holding chamber, the measured ventilation volume and the oxygen solubility coefficient in water at the test temperature. Cardiac output was calculated from the oxygen uptake, the dorsal aortic $P_{O_4}$ and an assumed venous $P_{O_4}$ of 32 mmHg. We believe this assumption to be justified as the second portion of this study, the results of Cameron & Davis (1970) and those of Holeton & Randall (1967b) all indicate that $P_{d(O_4)}$ in trout remains steady at 30–35 mmHg. Blood oxygen content calculations were based upon unpublished blood
curves for rainbow trout by Beaumont, Holeton & Randall and the relationship between haematocrit and blood oxygen content determined by Holeton & Randall (1967b)

\[ \alpha B_{O_2} = 0.311 \text{Hct} + 0.7, \]

(where \( \alpha B_{O_2} \) and \( \text{Hct} \) are in volumes %). Measurements of \( \alpha B_{O_2} \) in rainbow trout using Tucker's (1967) technique agree with those predicted by Holeton & Randall's formula. It was assumed that venous + arterial blood contained 2.5 and 1.15 mm \( P_{CO_2} \), respectively (Holeton & Randall, 1967b).

**Gill perfusion studies**

To study the effect of varying water flow over the gills it was necessary to devise a method of artificially perfusing the gills of intact trout at different flow rates. Dorsal and ventral aortic cannulae were implanted (Smith & Bell, 1964; Holeton & Randall, 1967a) and a 4 cm length of stiff Tygon tubing (9 mm I.D., 12 mm O.D.) was tied into the mouth. The tube was tied securely so that it penetrated about 1 cm into the buccal cavity and directed a flow of water toward the gills in a plane parallel with the roof of the mouth. The mouth tube eliminated any activity of the buccal pump but the opercular apparatus was free to move.

Following cannulation the trout was placed in a Lucite box similar to the constant-flow system described by Fry (1957). Lucite pegs inserted into holes in a baseplate within the box held the fish upright and kept its body straight. This form of restraint prevented the fish turning its head so that more water could flow over the gills on one side of the body than the other. The top of the box was secured with a gasket and wingnuts and cannulae passed out of the box through needles inserted in rubber stoppers (Fig. 2). Black plastic covered the top and sides of the box.

![Fig. 2. The apparatus for artificial perfusion of trout gills at different water flow rates. A tube is tied into the mouth and the fish is supported with Lucite pegs. Cannulae for blood and water sampling are illustrated.](image)

Oxygen tension in inflow water, outflow water and dorsal and ventral aortic blood were measured with a Radiometer electrode system. All attempted measurements of \( P_{CO_2} \) in water and blood with this system were unsuccessful as \( P_{CO_2} \) was below the 3 mmHg limit of the scale. Blood samples were returned to the animal by backflushing the cuvettes with heparinized Cortland saline (Wolf, 1963). Pressures in the various cannulae were monitored with Statham model P 23 BB pressure transducers and a Beckman RS Dynograph recorder.
Gill water flow in trout

The fish were allowed to recover for 24 h following cannulation since these procedures involve considerable trauma (Houston, DeWilde & Madden, 1969). Following recovery the gills were perfused with water for a period of 1 h at a given rate. At the end of that hour $P_{O_2}$ and blood pressure were recorded from the appropriate cannulae and the flow was reset to a new level. Flows ranged from 45 to 1200 ml/min and were applied at random with the exception that the lowest flows were not used until the end of the experiment. In these experiments 41 trout ranging from 195 to 388 g were studied at temperatures of 10-13-5 °C.

RESULTS

Direct measurement of ventilation volume

Mean ventilation volumes and other parameters from 18 rainbow trout are summarized in Table 1. The number of observations used for each mean $V_O$, $VR$ or $V_{SV}$ is given in column 3 of the table.

Table 1. Circulatory and ventilatory parameters for 18 trout (210-3 ± 2-3 g) at 8-6 °C with oral membranes attached for direct determination of ventilation volume

(All the values are means with the exception of those for minimum and maximum $V_O$ which are individual observations for each fish.)

<table>
<thead>
<tr>
<th>Fish no.</th>
<th>Av. $V_O$ ± s.e. (ml/min)</th>
<th>Av. $VR$ (no./min)</th>
<th>Min. $V_O$ (ml/min)</th>
<th>Max. $V_O$ (ml/min)</th>
<th>Av. $U$ (%)</th>
<th>$Q$ (ml/min)</th>
<th>$V_O$/ $Q$ (mg/kg/h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>48 ± 2-7</td>
<td>9</td>
<td>98-7 ± 0-50</td>
<td>37-0</td>
<td>162-0</td>
<td>39-7</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>38-4 ± 3-3</td>
<td>6</td>
<td>94-7 ± 0-39</td>
<td>30-0</td>
<td>144-0</td>
<td>44-8</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>29-1 ± 3-0</td>
<td>5</td>
<td>68-8 ± 0-41</td>
<td>27-0</td>
<td>147-0</td>
<td>50-8</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>31-4 ± 2-0</td>
<td>7</td>
<td>67-0 ± 0-47</td>
<td>28-0</td>
<td>46-0</td>
<td>47-9</td>
<td>3-7</td>
</tr>
<tr>
<td>5</td>
<td>26-0 ± 0-9</td>
<td>9</td>
<td>68-3 ± 0-34</td>
<td>24-0</td>
<td>27-0</td>
<td>49-5</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>34-7 ± 2-4</td>
<td>4</td>
<td>61-0 ± 0-57</td>
<td>29-0</td>
<td>118-0</td>
<td>51-3</td>
<td>6-0</td>
</tr>
<tr>
<td>7</td>
<td>49-0 ± 2-3</td>
<td>6</td>
<td>108-2 ± 0-46</td>
<td>41-0</td>
<td>94-0</td>
<td>28-5</td>
<td>4-1</td>
</tr>
<tr>
<td>8</td>
<td>33-7 ± 1-9</td>
<td>5</td>
<td>77-0 ± 0-44</td>
<td>27-5</td>
<td>84-0</td>
<td>50-3</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>41-7 ± 1-6</td>
<td>6</td>
<td>86-5 ± 0-48</td>
<td>36-0</td>
<td>129-0</td>
<td>48-0</td>
<td>6-5</td>
</tr>
<tr>
<td>10</td>
<td>44-6 ± 4-0</td>
<td>11</td>
<td>62-4 ± 0-71</td>
<td>40-0</td>
<td>138-0</td>
<td>39-0</td>
<td>3-2</td>
</tr>
<tr>
<td>11</td>
<td>35-3 ± 0-6</td>
<td>5</td>
<td>73-8 ± 0-48</td>
<td>34-0</td>
<td>38-0</td>
<td>41-0</td>
<td>2-7</td>
</tr>
<tr>
<td>12</td>
<td>31-1 ± 2-4</td>
<td>6</td>
<td>67-8 ± 0-47</td>
<td>24-0</td>
<td>43-0</td>
<td>48-9</td>
<td>2-3</td>
</tr>
<tr>
<td>13</td>
<td>27-1 ± 1-8</td>
<td>10</td>
<td>59-1 ± 0-46</td>
<td>22-0</td>
<td>40-0</td>
<td>48-7</td>
<td>—</td>
</tr>
<tr>
<td>14</td>
<td>40-2 ± 2-8</td>
<td>10</td>
<td>61-7 ± 0-65</td>
<td>32-0</td>
<td>57-0</td>
<td>37-9</td>
<td>2-6</td>
</tr>
<tr>
<td>15</td>
<td>49-0 ± 2-3</td>
<td>4</td>
<td>68-0 ± 0-48</td>
<td>27-0</td>
<td>59-0</td>
<td>46-5</td>
<td>3-0</td>
</tr>
<tr>
<td>16</td>
<td>29-1 ± 1-5</td>
<td>7</td>
<td>59-0 ± 0-50</td>
<td>26-0</td>
<td>114-0</td>
<td>49-6</td>
<td>2-8</td>
</tr>
<tr>
<td>17</td>
<td>37-6 ± 2-2</td>
<td>5</td>
<td>71-2 ± 0-53</td>
<td>33-0</td>
<td>76-0</td>
<td>50-8</td>
<td>3-6</td>
</tr>
<tr>
<td>18</td>
<td>39-6 ± 1-5</td>
<td>6</td>
<td>75-0 ± 0-53</td>
<td>35-0</td>
<td>71-0</td>
<td>55-4</td>
<td>5-6</td>
</tr>
</tbody>
</table>

| Av.    | 37-0 ± 2-7              | 9                  | 73-8 ± 0-49        | 30-7               | 88-2        | 46-0        | 3-9                 |
| n      | 18                      | 18                 | 18                 | 18                 | 18          | 12           | 12                  |
| S.D.   | 7-4                     | 14-3               | 0-09               | 5-6                | 43-7        | 6-5         | 1-4                 |
| S.s.   | 7-8                     | 3-4                | 0-02               | 1-3                | 10-3        | 1-5         | 0-4                 |

Mean ventilation volume for the group, weight 210±3-0 g, at 8-6±0-2 °C was 37±1-8 ml/min when the animals were quiet. In one individual $V_O$ fell as low as 22 ml/min and in another rose as high as 162 ml/min. $V_O$ rose sharply when the fish struggled or was disturbed by tapping on the box (Fig. 3).
Ventilatory stroke volume was normally about 0.5 ml/breath and increased markedly as ventilation volume went up. During struggling or when the animal was disturbed in some way $V_{sv}$ was high.

Ventilation rate usually varied little for any one individual despite the fact that observations were often made on the same individual for 2 consecutive days. There was considerable variance in $VR$ between individuals however, and those with high mean $V_{Os}$ tended to have high mean $VRs$ (cf. Tables 1, 2).

Utilization of oxygen from the water passing over the gills, calculated from

$$\% U = \frac{P_{f(lO_2)} - P_{E(lO_2)}}{P_{f(lO_2)}} \times 100,$$

had a mean value of 46.1 ± 1.5% and ranged from 28.5 to 64%. Utilization decreased as $V_{O}$ increased when the fish was disturbed or struggled. The oxygen content of inspired water was virtually constant for any one experiment and ranged between 157 and 163 mm $P_{O_2}$ over the entire study.

Arterial $P_{O_2}$ was quite variable, ranging between 82 and 130 mmHg. This range of tensions represents a small variation in blood oxygen content. Unpublished dissociation curves for rainbow trout blood (Beaumont, Holeton & Randall) show it to be 85–100% saturated with oxygen over that $P_{O_2}$ range.

Calculated cardiac output values for the trout ranged between 2.6 and 6.5 ml/min/fish and had a mean of 3.9 ± 0.4 ml/min/fish. These cardiac outputs, together with the appropriate ventilation volume values for each animal, were used to determine ventilation–perfusion ratios, the ratio of water flow to blood flow over the gills. Ventilation–perfusion ratio ($V_{O}/Q$) had a mean of 10.4 and ranged from 5.8 to 15.5.

Oxygen uptake levels for quiescent trout ranged between 41.4 and 75.9 mg/kg/h and had a mean of 55.3 ± 2.1 mg/kg/h (Table 1).

Simple correlation coefficients between the various circulatory and ventilatory parameters for quiescent fish are given in Table 2. A significant correlation (0.01 level) is shown between $\% U$ and $V_{O}$. There is also a positive correlation between $V_{sv}$ and weight (0.05 level).
Gill water flow in trout

Ventilation volumes and other parameters for four fish subjected to hypoxia are summarized in Table 3. These data were recorded no later than 5 min after the oxygen tension of the inspired water was reduced rapidly to approximately 60 mm \( P_{O_2} \). During hypoxia \( V_G \) rose as high as 276 ml/min in one individual and ventilatory stroke volume went up to 3.7 ml/breath. Averaging the data for the four fish indicates that ventilation volume increased nearly sevenfold in response to hypoxia. Ventilation rate rose only slightly from the level accompanying normoxic conditions. Thus the increase in ventilation volume during hypoxia was largely a result of increased ventilatory stroke volume rather than a large increase in ventilatory rate.

Table 2. A simple correlation analysis on circulation and ventilation data using the mean values for 18 trout with attached oral membranes

(Only correlations which were significant at the 0.05 level or better are given.)

<table>
<thead>
<tr>
<th></th>
<th>( V_G )</th>
<th>( VR )</th>
<th>( V_R )</th>
<th>( %U )</th>
<th>( V_{O_1} )</th>
<th>( \dot{Q} )</th>
<th>( V_{O_1}/\dot{Q} )</th>
<th>( Hct )</th>
<th>Weight</th>
<th>Temp.</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_R )</td>
<td>( 0.68^{*} )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
</tr>
<tr>
<td>( V_{R_R} )</td>
<td>( 0.46^{*} )</td>
<td>( N.S. )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
</tr>
<tr>
<td>( %U )</td>
<td>( 0.67^{*} )</td>
<td>( 0.51^{*} )</td>
<td>( N.S. )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
</tr>
<tr>
<td>( V_{O_1} )</td>
<td>( 0.47^{*} )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
</tr>
<tr>
<td>( \dot{Q} )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( 0.69^{*} )</td>
<td>( 0.72^{<em>}^{</em>} )</td>
<td>( 0.84^{<em>}^{</em>} )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
</tr>
<tr>
<td>( V_{O_1}/\dot{Q} )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( 0.63^{*} )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
</tr>
<tr>
<td>( Hct )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( 0.76^{*} )</td>
<td>( 0.69^{*} )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
<td>( - )</td>
</tr>
<tr>
<td>Weight</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( 0.52^{*} )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
</tr>
<tr>
<td>Temp.</td>
<td>( N.S. )</td>
<td>( 0.49^{*} )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
<td>( N.S. )</td>
</tr>
</tbody>
</table>

* = Significant at 0.05 level. ** = Significant at 0.01 level. N.S. = Not significant at 0.05 level.

Table 3. Ventilatory changes in four trout that resulted when the inspired oxygen tension fell rapidly to approximately 60 mmHg

(The values during hypoxia were determined within 5 min of introducing the hypoxic water.)

<table>
<thead>
<tr>
<th>Weight</th>
<th>Temp.</th>
<th>( V_G )</th>
<th>( VR )</th>
<th>( V_R )</th>
<th>( %U )</th>
<th>( V_{O_1} )</th>
<th>( \dot{Q} )</th>
<th>( V_{O_1}/\dot{Q} )</th>
<th>( Hct )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( g )</td>
<td>( ^\circ C )</td>
<td>s.e.</td>
<td>s.e.</td>
<td>s.e.</td>
<td>s.e.</td>
<td>s.e.</td>
<td>s.e.</td>
<td>s.e.</td>
<td>s.e.</td>
</tr>
<tr>
<td>230.0</td>
<td>7.8</td>
<td>35.8</td>
<td>3.4</td>
<td>58.8</td>
<td>4.9</td>
<td>0.61</td>
<td>190.0</td>
<td>72</td>
<td>2.64</td>
</tr>
<tr>
<td>232.0</td>
<td>7.8</td>
<td>27.5</td>
<td>0.7</td>
<td>72.0</td>
<td>0.8</td>
<td>0.38</td>
<td>276.0</td>
<td>74</td>
<td>3.73</td>
</tr>
<tr>
<td>182.3</td>
<td>7.8</td>
<td>36.4</td>
<td>1.4</td>
<td>70.4</td>
<td>1.7</td>
<td>0.52</td>
<td>194.0</td>
<td>96</td>
<td>2.02</td>
</tr>
<tr>
<td>238.6</td>
<td>7.1</td>
<td>24.4</td>
<td>0.5</td>
<td>72.0</td>
<td>0.6</td>
<td>0.34</td>
<td>185.0</td>
<td>76</td>
<td>2.43</td>
</tr>
<tr>
<td>Av.</td>
<td>220.8</td>
<td>7.6</td>
<td>31.0</td>
<td>68.3</td>
<td>0.46</td>
<td>211.3</td>
<td>79.5</td>
<td>2.71</td>
<td>1.17</td>
</tr>
</tbody>
</table>

Gill perfusion experiments

Inspired oxygen tensions measured in the inflow water to the chamber were fairly consistent over the entire flow range studied, ranging from 153 to 162 mmHg. Oxygen tensions in outflow water from the box reached a low of 85 mm \( P_{O_2} \) at the minimum flow rate applied (45 ml/min) and went up as perfusion rate increased, reaching a maximum value of 154 mm at the highest perfusion rate (Fig. 4). Percentage utilization of oxygen from the water, calculated from the above data, dropped from 44% at the
lowest perfusion rate to a minimum value of 3.8% as the perfusion rate increased (Fig. 4).

Mean dorsal aortic $P_{O_2}$ ranged from 131 to 135 mmHg at gill water flows above 500 ml/min and diminished at lower perfusion rates, reaching 44 mm $P_{O_2}$ at a perfusion rate of 45 ml/min (Fig. 5). Ventral aortic $P_{O_2}$ remained virtually unchanged over the entire flow range at 31.6 ± 0.6 mmHg.

Blood pressure data was converted to area mean pressure according to the formula

$$\text{area mean pressure} = \frac{\text{systolic pressure} + 2 \times \text{diastolic pressure}}{3}.$$  

Area mean pressure gives a good approximation of average pressures that occur in vessels during pulsatile flow (Burton, 1966). Area mean blood pressure in the ventral aorta remained steady at around 35 mmHg over the entire flow range. Dorsal aortic pressure averaged 29.3 ± 1.9 mmHg at a perfusion rate of 80 ml/min and dropped slightly as perfusion rate increased, reaching a low of 21.8 ± 2.0 mmHg at a flow of 1043 ml/min. A $t$-test showed these two values were significantly different at the 0.05 level.
Gill water flow in trout

Oxygen uptake rate, calculated by the Fick principle, rose from 56.2 to 104.9 mg/kg/h as the flow rose to 750 ml/min (Fig. 5). At higher gill water flows oxygen uptake remained around 100 mg/kg/h. Cardiac output, calculated from the preceding data, rose in accordance with the increase in perfusion rate. At a flow of 85 ml/min cardiac output was approximately 4 ml/min/fish while at a flow of 750 ml/min \( Q \) was 6.5 ml/min/fish. At perfusion rates above 85 ml/min heart rate showed little change, remaining around a mean value of 63 ± 3.4 beats/min. At the lowest flow tested (45 ml/min) cardiac output, calculated by the Fick principle using an assumed \( \text{P}_{\text{aCO}_2} \) of 34 mmHg was 15.4 ml/min/fish. This \( Q \) value is considerably higher than those at greater perfusion rates. In addition, heart rate at the minimum perfusion rate was 49 beats/min—a rate somewhat lower than at any other perfusion rate.

![Fig. 5. Circulatory and ventilatory parameters recorded from 41 trout whose gills were perfused at different flow rates. \( \dot{V}_O_2 \) and \( Q \) were calculated using the means for the group. Other values are means ± 2 standard errors.](image-url)
The above data were used to calculate the transfer factor for oxygen across the gills. Transfer factor \( T_{O_2} \) is a measure of the relative ability of the respiratory surface to exchange gases and may be calculated by dividing the oxygen uptake rate by the pressure gradient for oxygen across the gills (Randall et al. 1967)

\[
T_{O_2} = \frac{\dot{V}_{O_2}}{\frac{1}{2}(P_{H_2O} + P_{E_{O_2}}) - \frac{1}{2}(P_{a_{O_2}} + P_{a_{CO_2}})}
\]

Transfer factor was 0.0078 ml O\textsubscript{2}/min/mmHg at the lowest flow rate and increased by about 50\% as the flow rose to its highest level (Fig. 6).

![Fig. 6. Area mean dorsal aortic pressure (means ± 2 standard errors) and the transfer factor for oxygen across the gills of perfused fish.](image)

Opercular closure rate was zero at perfusion rates in excess of 580 ml/min but increased rapidly as flow dropped below this level, reaching a maximum rate of 85 closures/min at a perfusion rate of 95 ml/min (Fig. 5).

\( P_{CO_2} \) levels in both water and blood afferent and efferent to the gills were always below 3mmHg and could therefore not be measured.

**DISCUSSION**

**Measurement of ventilation volume**

Basically, two techniques have been used to determine ventilation volume in fish: (1) the direct technique where all the expired water is collected and (2) indirect methods where \( \dot{V}_G \) is calculated by the Fick principle or is estimated by dye dilution. A summary of literature values for \( \dot{V}_G \) in teleosts and the methods used to obtain these values is given in Table 4.

Recent experiments have indicated that expired oxygen tensions recorded from opercular cannulae in trout (Davis & Watters, 1970) or carp (Garey, 1967) are highly variable. It was felt that this variability could lead to inaccurate estimation of mean expired oxygen tension. Mean expired oxygen tension has frequently been used to calculate ventilation volume by the Fick principle. Owing to these limitations a direct method of \( \dot{V}_G \) measurement was adopted in this study.
Table 4. Ventilation volumes and other parameters measured or estimated for teleosts under a variety of conditions

(The methods used to estimate $V_G$ are: 1-dir. coll. = direct collection of expired water with rubber sacs on the gill outflow, 2-Fick = calculated by the Fick principle from $V_O$, and the oxygen pressure gradient across the gills, 3-van Dam = a rubber van Dam-type membrane stretched over the head, 4-calc. = calculated from theoretical considerations, 5-oral mem. = the oral membrane of the present study.)

<table>
<thead>
<tr>
<th>Species</th>
<th>Reference</th>
<th>Temp. (°C)</th>
<th>Wt (kg)</th>
<th>$O_2$ uptake (mg/kg/h)</th>
<th>% $U$</th>
<th>$Q$ (ml/kg/min)</th>
<th>$V_G/Q$ (ml/kg/min)</th>
<th>$V_G$ (ml/kg/min)</th>
<th>Method of $V_G$ determination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spheroidei maculatus</td>
<td>1</td>
<td>12-22</td>
<td>—</td>
<td>17-103</td>
<td>45-48</td>
<td>—</td>
<td>—</td>
<td>17-117*</td>
<td>1-dir. coll.</td>
</tr>
<tr>
<td>Catostomus commersoni</td>
<td>2</td>
<td>20</td>
<td>0.25</td>
<td>90</td>
<td>2-68</td>
<td>—</td>
<td>—</td>
<td>391-8663</td>
<td>2-Fick</td>
</tr>
<tr>
<td>Ictalurus nebulotus</td>
<td>2</td>
<td>20</td>
<td>0.18</td>
<td>71</td>
<td>8-78</td>
<td>—</td>
<td>—</td>
<td>239-2709</td>
<td>2-Fick</td>
</tr>
<tr>
<td>Cyprinus carpio</td>
<td>3</td>
<td>2-6</td>
<td>28</td>
<td>—</td>
<td>18</td>
<td>12</td>
<td>—</td>
<td>214</td>
<td>2-Fick</td>
</tr>
<tr>
<td>C. carpio</td>
<td>2</td>
<td>2-6</td>
<td>0.174</td>
<td>100</td>
<td>5-85</td>
<td>—</td>
<td>—</td>
<td>331-2891</td>
<td>2-Fick</td>
</tr>
<tr>
<td>Callionymus lyra</td>
<td>4</td>
<td>11-12</td>
<td>0.07-0.14</td>
<td>65</td>
<td>40</td>
<td>—</td>
<td>—</td>
<td>30*</td>
<td>1-dir. coll.</td>
</tr>
<tr>
<td>Mugil cephalus</td>
<td>5</td>
<td>13-21</td>
<td>0.08-0.29</td>
<td>120</td>
<td>66</td>
<td>—</td>
<td>—</td>
<td>20-101*</td>
<td>3-van Dam</td>
</tr>
<tr>
<td>Ictalurus punctatus</td>
<td>6</td>
<td>23-24</td>
<td>0.007</td>
<td>206</td>
<td>47</td>
<td>—</td>
<td>—</td>
<td>925</td>
<td>3-van Dam</td>
</tr>
<tr>
<td>T. tinca</td>
<td>7</td>
<td>17-20</td>
<td>0.05-0.07</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>39-164*</td>
<td>3-van Dam</td>
</tr>
<tr>
<td>T. tinca</td>
<td>8</td>
<td>19-20</td>
<td>0.07-0.13</td>
<td>63-140</td>
<td>67</td>
<td>—</td>
<td>—</td>
<td>20-50†</td>
<td>1, 3</td>
</tr>
<tr>
<td>Anquilla vulgaris</td>
<td>9</td>
<td>16-18</td>
<td>0.41-0.92</td>
<td>57-77</td>
<td>65-80</td>
<td>—</td>
<td>—</td>
<td>90-153</td>
<td>3-van Dam</td>
</tr>
<tr>
<td>Katsuoosus pelamis</td>
<td>10</td>
<td>—</td>
<td>1-67</td>
<td>419</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2014</td>
<td>4-calc.</td>
</tr>
<tr>
<td>Macreral</td>
<td>11</td>
<td>—</td>
<td>—</td>
<td>51</td>
<td>53</td>
<td>120</td>
<td>19</td>
<td>1830</td>
<td>4-calc.</td>
</tr>
<tr>
<td>Salmo gairdneri</td>
<td>12</td>
<td>9-19</td>
<td>0.21-1.1</td>
<td>100-125</td>
<td>55</td>
<td>65-100</td>
<td>2.7-55</td>
<td>274-3560</td>
<td>2-Fick</td>
</tr>
<tr>
<td>S. gairdneri</td>
<td>13</td>
<td>5</td>
<td>0.2-0.4</td>
<td>26-131</td>
<td>10</td>
<td>6-27</td>
<td>95-106</td>
<td>571-2853</td>
<td>2-Fick</td>
</tr>
<tr>
<td>S. gairdneri</td>
<td>14</td>
<td>9</td>
<td>0.21</td>
<td>55</td>
<td>46</td>
<td>3-9</td>
<td>10</td>
<td>37*</td>
<td>5-oral mem.</td>
</tr>
<tr>
<td>S. shasta</td>
<td>9</td>
<td>10-12</td>
<td>0.90</td>
<td>67</td>
<td>80</td>
<td>—</td>
<td>—</td>
<td>148</td>
<td>3-van Dam</td>
</tr>
</tbody>
</table>

References: 1-Hall (1931); 2-Saunders (1962); 3-Garey (1967); 4-Hughes & Umezawa (1968); 5-Cech (1970); 6-Gerald & Cech (1970); 7-Hughes & Shelton (1958); 8-Schumann & Piiper (1966); 9-van Dam (1938); 10-Brown & Muir (1969); 11-Rahn (1966); 12-Holeton & Randall (1967b); 13-Stevens & Randall (1967b); 14-present study.

* = ml/min; † = ml/100 g.
Most estimates of $V_o$ in Table 4 are in ml/kg/min although the studies were not made on 1 kg fish. There is no evidence that $V_o$ is directly related to weight so conversion of $V_o$ estimates to ml/kg/min may introduce an additional source of error. Indeed, metabolic rate and gill surface area increase with body weight to the power of about 0.8-0.9 (Muir, 1969; Muir & Hughes, 1969; Price, 1931) and the growth of other parts of the body is allometric (Prosser & Brown, 1962). Mouth size in fish, and consequently ventilation volume, probably follow a similar pattern.

Table 4 reveals considerable differences among ventilation volume estimates made within and between different species of teleosts. These estimates were made over a wide range of experimental conditions (fish size, temperature, degree of activity, varying oxygen tensions, varying carbon dioxide tensions) and are hence not directly comparable without consideration of these conditions.

Estimates of ventilation volume for rainbow trout (Holeton & Randall, 1967) obtained by indirect means (opercular cannulation) range from 274 to 3560 ml/kg/min. Holeton & Randall’s maximum $V_o$ is for a group of trout under severe hypoxia for a long period (35-40 mm $P_o$, after 1-2 h progressive hypoxia). Stevens & Randall (1967) reported that $V_o$ ranged from 571 to 2855 ml/kg/min for 200-400 g trout at 5 °C in a tunnel-type respirometer with water flowing past the fish. This range represents indirect estimates of $V_a$ for quiescent to moderately active trout in the respirometer. In the present study the $V_o$ of quiescent trout was 371 ± 8 ml/min (171 ml/kg/min), a value lower than the minimum values reported above. The difference may be related to variations in experimental conditions or in part to the techniques used to determine $V_a$. Van Dam (1938), using a direct technique of measurement on a single 900 g trout, obtained $V_o$ values very similar to those of the present study on a per kg basis.

Oral membranes could produce inaccurate measures of $V_o$ if they caused impairment of ventilation. No such impairment was detected as the dorsal aortic blood remained 85-100% saturated and oxygen uptake values were consistent with those measured for similar-sized rainbow trout at 4-8 °C (Stevens & Randall, 1967). Indeed, the membrane was designed so that the tension necessary to produce a seal was exerted laterally from the snout to the corner of the mouth along the maxilla, and from the corner of the mouth to the tip of the mandible. There was virtually no tension exerted on the mouth in a dorso-ventral direction. In addition, fish with oral membranes were capable of a sevenfold increase in $V_o$ in response to hypoxia (Table 3). Since the fish were able to increase $V_o$ to this extent it seems clear that the membrane did not significantly restrict pumping. Tests with dye showed that leakage was not a significant source of error.

In over 200 separate measurements of percentage utilization from fish with oral membranes percentage utilization never rose as high as 80% as reported by Van Dam (1938). Utilization ranged from 23 to 64% and was usually around 50%. It is possible that the tight rubber membrane stretched over the head of Van Dam’s fish impaired pumping to some degree. Preliminary experiments in our laboratory indicated that rubber or plastic collecting bags attached to the operculae of trout produced low $V_o$s and high utilizations compared to fish with oral membranes. In some cases the restriction was severe enough to kill the fish. Van Dam’s trout was large (900 g) and it is hard to say if a serious restriction of breathing existed in a fish of that size. Presumably, the restriction would be greater in small fish owing to the sensitivity of the operculae
Gill water flow in trout

to physical loading and the increased relative tension (Laplace's law). Furthermore, van Dam's $V_O$ estimates compare closely with ours on a per kg basis. Indeed, the 80% utilization reported by van Dam may be related to the large size of his fish. G. F. Holeton (personal communication) observed that larger trout tend to have high utilizations. Table 2, however, shows no correlation between weight and % $U$, possibly because too small a size range of fish was examined.

Oral membranes then, appear to be useful for measurement of $V_O$ and other parameters in quiescent trout providing restraint of the fish can be tolerated. They do not appear to produce any great restriction of breathing in 200 g fish. Oral membranes allow repeated and accurate determination of oxygen utilization and ventilation volume. Fick principle calculations of ventilation volume depend upon accurate determination of mean expired oxygen tension. Ocular catheterization, a technique used to determine mean $P_E(\text{O}_2)$, often produces highly variable results and could lead to errors in determining mean $P_E(\text{O}_2)$. Oral membranes are free of such limitations as they allow direct $V_O$ measurement and provide a mixed sample of all the expired water for determination of mean $P_E(\text{O}_2)$. The authors suggest that the reliability of mean $P_E(\text{O}_2)$ measurements from cannulae should always be checked using an independent method.

The ventilation-perfusion ratio for trout in this study was $10.4 \pm 0.9$. Holeton & Randall (1967b) reported that $V_O/Q$ ranged from 2.7 to 55 (normoxic to severe, prolonged hypoxia) and Stevens & Randall (1967b) reported a $V_O/Q$ range of 95-106 for trout subjected to a range of swimming activity in a respirometer. The large variation in ventilation-perfusion ratio reported for trout in these three studies could have resulted from the different conditions present in each experiment and/or the techniques used to determine $V_O/Q$. In each case $Q$ was determined by the Fick principle, a procedure which requires accurate knowledge of tensions of oxygen and carbon dioxide in arterial and venous blood and a representative oxygen dissociation curve for the blood. If the blood curve was slightly in error or the magnitude of the Bohr shift was not correctly estimated then inaccurate values for $Q$ would result. In the present study it was not possible to measure blood $P_{CO_2}$. Thus our calculated $Q$ depends upon the assumption that venous and arterial blood of quiescent trout contained 2.5 and 1.15 mm $P_{CO_2}$ respectively as reported by Holeton & Randall (1967b). $V_O/Q$ estimates based on theoretical considerations (Rahn, 1966) or determined for other fish species (Baumgarten-Schumann & Piiper, 1968; Hanson & Johansen, 1970; Piiper & Schumann, 1967; Robin, Murdaugh & Millen, 1966; Garey, 1967) range from 8 to 20.

Although there was only a small increase in ventilation rate during hypoxia, Table 2 shows $V_O$ strongly correlated with $VR$. This is because individuals showed very little variation in ventilation rate but some had higher rates than others. Those individuals with high rate also had high $V_O$, hence the positive correlation between these two parameters.

**Gill perfusion studies**

The velocity and pattern of water flow over the gills must have a considerable effect on gas exchange. If the mouth tube directed water over the gills in a pattern of flow that was not optimum for gas exchange then impairment of exchange might result.
Perfused fish were unable to saturate their arterial blood with oxygen at the lowest flow rate tested (Fig. 5). This perfusion rate (45 ml/min) approximates the $V_G$ of quiescent trout with oral membranes attached. Thus it would appear that artificial perfusion of the gills by means of the mouth tube provides less effective conditions for gas exchange than those present during normal ventilation.

It is likely that the restriction of exchange resulted from a poor pattern of water flow over the gills during perfusion. Probably only a portion of the gill sieve received adequate ventilation at the lowest perfusion rate. A flared perfusion tube might have directed the water flow more uniformly over the gills and resulted in a more complete saturation of the blood at the lowest flow. At high perfusion rates the dorsal aortic blood was saturated but utilization was reduced as a result of the high flow rates.

Utilization at the lowest perfusion rate was 44%, a value nearly identical with the mean utilization (46%) of trout with oral membranes at a mean $V_G$ of 37 ml/min. Thus although the dorsal aortic blood was not saturated with oxygen at this low perfusion rate some compensation must have taken place within the fish to maintain a normal utilization when a poor pattern of flows existed at the gills. Fig. 5 shows that cardiac output was high at the lowest perfusion rate in comparison to $Q$ at higher perfusion rates. This high $Q$ value was calculated by the Fick principle using an assumed $P_{\text{a}(O_2)}$ of 34 mmHg and may therefore be subject to error. An increase in $Q$ in response to diminished $P_{\text{a}(O_2)}$ would facilitate utilization and maintain an adequate $V_O$ despite the poor flow conditions at the gills. Rainbow trout increase their cardiac output in response to hypoxia (Holeton & Randall, 1967b) or anaemia (Cameron & Davis, 1970) when environmental oxygen is low or the oxygen carrying capacity of the blood is reduced.

Opercular movements appeared at perfusion rates below 700 ml/min and increased in frequency as flow rate declined (Fig. 5). There are two possible sources for initiation of this opercular activity. Ventilation could be controlled by information from receptor sites on the gills sensitive to changes in water flow. Sutterlin & Saunders (1969) described proprioceptors associated with gill filaments and rakers of the sea raven and Atlantic salmon. These receptors responded to mechanical displacement. Alternately, the opercular activity could be related to arterial oxygen tension. Opercular movement was initiated at roughly the same flow rate as that when dorsal aortic $P_{O_2}$ began to decline (Fig. 5). It is possible that trout possess a receptor system capable of adjusting ventilation in response to changes in arterial $P_{O_2}$. The pseudobranch could be the site of such a system as it receives an arterial blood supply and has been shown to exhibit chemoreceptor and baroreceptor activity (Laurent, 1967).

Cardiac output rose approximately 50% as perfusion rate increased from 85 ml/min to the highest rates tested. As mentioned earlier, Fick principle calculations of $Q$ in this study may be somewhat inaccurate due to a lack of $P_{\text{a}(O_2)}$ data. In addition, the $Q$ calculations may have been affected in perfused fish by ‘washing out’ of blood CO$_2$ owing to the high solubility of CO$_2$ in water and the persistent gradient between blood and water at high perfusion rates. It appears that inaccuracies in calculated $Q$ were present for the perfused fish as $V_O$ approximately doubled over the perfusion rate range while calculated $Q$ increased by only about 50%. Since arterio-venous oxygen difference did not change appreciably over the flow range (Fig. 5), except at the lowest flow rate, the only means of increasing $V_O$ was by elevation of $Q$. Thus a doubling
of $\dot{V}_{O_2}$ should have been accompanied by a doubling of $Q$. These results show that Fick principle calculations of $Q$ are subject to the same limitations as Fick principle calculations of $V_O$. Reliable Fick $Q$ requires accurate knowledge of arterial and venous $P_{O_2}$ and $P_{CO_2}$ levels and a representative blood curve. Errors in $Q$ in the present study are probably a result of lack of $P_{CO_2}$ data.

Oxygen uptake increased as perfusion rate went up (Fig. 5) despite the fact that utilization fell (Fig. 4) and anatomical deadspace in the gills had probably increased (Hughes, 1966). Assuming that our calculations of $Q$ are sufficiently accurate to show large changes in $Q$, the rise in $\dot{V}_{O_2}$ can be attributed to two factors: (1) an increase in cardiac output and (2) a vasodilation of the gills. Cardiac output rose with perfusion rate but there was no elevation of dorsal aortic blood pressure (Fig. 6). In fact, blood pressure decreased slightly as perfusion rate went up. Since blood pressure did not rise in the presence of elevated $Q$ there must have been a vasodilation of blood vessels in both the gills and peripheral circulation. Vasodilation of the gills could open additional vascular exchange areas (Steen & Kryusse, 1964; Richards & Fromm, 1969) and lead to a reduction in physiological deadspace (Hughes, 1966). Also, the 50% increase in transfer factor that occurred as flow went up suggests that adjustments favouring the transfer of oxygen across the gills had occurred. An increase in transfer factor could be the result of increased surface area of the gills, a decrease in diffusion distance for oxygen, an increase in the diffusion coefficient for oxygen or a combination of these factors (Randall et al. 1967).

The rise in $\dot{V}_{O_2}$, $Q$ and vasodilation could result from the fish becoming disturbed or excited at high flows. During excitement catecholamines liberated into the blood (Nakano & Thomlinson, 1966) act on $\alpha$-adrenergic receptors and may cause dilation of gill blood vessels (Randall & Stevens, 1967). These catecholamines could also elevate cardiac output by increasing the rate and force of contraction of the heart (Randall, 1968).

Another possibility is that ventilation and circulation may be controlled so that cardiac output and $\dot{V}_{O_2}$ are matched. It does not appear that $\dot{V}_{O_2}$ increases in response to elevated cardiac output alone in trout since $\dot{V}_{O_2}$ does not rise during anaemia despite a tenfold increase in $Q$ (Cameron & Davis, 1970). However, the capacity-rate ratio (Hughes & Shelton, 1962) was maintained in anaemic fish. The present study showed that $Q$ increased as perfusion rate went up which would result in a tendency to maintain capacity-rate ratio. This rise in $Q$, accompanied by a vasodilation of the gills, would facilitate gas exchange at high $\dot{V}_{O_2}$ and would meet the increased exchange requirements associated with activity or excitement.

Utilization of oxygen from the inspired water was exceedingly low at high perfusion rates (Fig. 4). This is a result of the high flow rates and short residence time of the water in the gills and may also reflect poor exchange conditions at the gills at high flows. When flow rate is high adjacent gill filament tips are drawn apart and a portion of the respiratory flow spills past the tips without contacting the lamellae (Saunders, 1961; Pasztor & Kleerekoper, 1962). Hence, a large proportion of the total flow would follow a non-respiratory pathway and low utilization would result. Also, interlamellar flow rate could be high enough to prevent water in the centre of each interlamellar space from exchanging gases at all (see fig. 3 in Hughes, 1966). Active fish with high $\dot{V}_{O_2}$ have more closely spaced lamellae than sluggish fish (Hughes, 1966).
Closely spaced lamellae would reduce interlamellar anatomical deadspace at high flow rates. It is also possible that interlamellar flow decreases at high flows owing to distortion of the lamellae and abduction of the gill filaments to allow non-respiratory spillage of water. The fusion of gill filaments in tuna (Muir & Kendall, 1968, 1969) may reduce lamellar distortion and keep the lamellae close together to facilitate utilization at high flow rates.

Artificial perfusion of the gills by means of a mouth tube appears to provide a less than optimum flow pattern over the gills. The perfusion studies do provide useful information on the control of circulation and ventilation and emphasize the importance of efficient distribution during normal ventilation. In addition they show that gas exchange is not drastically inhibited at high perfusion rates even though anatomical deadspace may be high.

The limitations of mouth perfusion should be considered by those contemplating its use in experiments or surgical procedures. A flared perfusion tube might distribute water more uniformly to the gills. Care should always be taken to keep perfusion rates high to ensure saturation of the arterial blood of perfused fish.

**SUMMARY**

1. Ventilation volume was measured directly in rainbow trout using a rubber membrane attached to the mouth which separated inspired and expired water and allowed collection of the latter.

2. Mean ventilation volume at 8.6 °C for 18 trout weighing approximately 200 g was \(37 \pm 1.8\) ml/min/fish. Mean ventilation rate and ventilatory stroke volume averaged 74 breaths/min and 0.5 ml/breath respectively.

3. Ventilation volume could be increased nearly sevenfold during moderate, short-term hypoxia as a result of a large increase in ventilatory stroke volume and a small increase in ventilation rate.

4. The ratio between the flow rates of water and blood through the gills was approximately 10.

5. Percentage utilization of oxygen from inspired water had a mean of 46±1.5% and ranged from 23 to 64%.

6. Artificial perfusion of the gills with water at different flow rates was achieved by tying a tube into the mouth of trout.

7. Perfused fish could not saturate their arterial blood with oxygen at a perfusion rate of 45 ml/min but could do so at rates ranging from 85 to 1200 ml/min.

8. Low arterial tensions at a perfusion rate approximating the mean \(V_o\) of fish with oral membranes are probably the result of a poor pattern of water flow over the gills during perfusion.

9. Opercular movements occurred only at perfusion rates below 700 ml/min and increased in frequency as perfusion rate dropped. This ventilatory activity may have resulted from receptors sensitive either to water flow over the gills or to arterial \(P_{o_2}\).

10. As perfusion rate went up cardiac output and oxygen uptake increased. These changes were accompanied by a drop in dorsal aortic pressure which reflected vasodilation of the gills and peripheral circulation. This change in the pattern of blood
flow through the gills contributed to a 50% increase in oxygen transfer factor across the gills.

11. At the highest perfusion rates there was no apparent impairment of gas exchange even though anatomical deadspace was probably high.

We would like to thank Dr D. J. Randall who provided guidance during this study and assisted greatly in the preparation of this manuscript. The comments of Dr G. Holeton and Mr C. Wood are greatly appreciated. Miss S. Bourque provided technical assistance during the hypoxia experiments. Dr Murray Newman and the Vancouver Public Aquarium Association generously provided research space for this study. The work was supported by grants from the National Research Council of Canada and the British Columbia Heart Foundation and an NIH Postdoctoral Fellowship award (1-FO2-HE36334-01) to J. N. C. from the U.S. National Heart Institute.

REFERENCES


18

J. C. DAVIS AND J. N. CAMERON


