Oxygen Consumption of Arterioles and Venules as Studied in the Cartesian Diver

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The metabolism of the aorta and vena cava has been studied in material taken from the rat, rabbit, dog, pig, ox, and man. Aortic slices consume approximately one microliter of oxygen per milligram of dry tissue per hour in Ringer's phosphate medium containing glucose.1 There is evidence for aerobic and anaerobic glycolysis and for operation of Krebs' cycle in these large vessels.1-5

The oxygen consumption of small arterial vessels, 65 to 400 μ in diameter, and of adjacent venous vessels, 110 to 450 μ in diameter, is the subject of this paper. Data presented include the rate of oxygen uptake by normal human and hamster mesenteric arterioles and venules, the relation between oxygen consumption and vascular caliber, the influence of subnormal environmental oxygen tensions, and the effect of an anesthetic agent (pentothal).

Methods

Oxygen consumption was measured by the technique of Linderstrom-Lang. We measured changes in buoyancy of a small, open, gas-filled glass flask (the Cartesian diver) by observation of changes of pressure required to float the diver to a fixed level in its flotation medium. Tissues were suspended in phosphate Ringer as shown in figure 1. The composition of Ringer's fluid was KCl 2.8 mM, CaCl₂ 1 mM, NaCl 142 mM, NaH₂PO₄ 11 mM, and glucose 1.1 mM/1000 ml of H₂O. Above the tissue and its substrate were (1) a gas bubble which provided buoyancy and a source of oxygen, (2) a layer of KOH to absorb CO₂ produced by the respiring tissue, and (3) a layer of mineral oil to retard diffusion of water vapor from the bulb of the diver into the high-density salt solution in which it floated. The flotation medium consisted of NaNO₃ 27 g, NaCl 14 g, Na taurocholate 0.2 g/59 ml of water and had a specific gravity of 1.326.

Each loaded diver was placed in a separate tube containing flotation medium, and maintained in a water bath at 37.00 ± 0.01°C. The flotation tubes were connected one after another with a manometer (fig. 2) containing Brodie's solution.7 Oxygen consumed by the respiring tissue in the diver was replaced by flotation medium entering its open mouth, so that the diver became heavier, and increasing negative pressure was required to float the diver to a fixed reference point, the horizontal hairline in a telescope. The observed, progressive changes of pressure were plotted against elapsed time. The change in volume of gas in the diver, equivalent to the volume of oxygen...
The Cartesian diver is a glass flask, 15 mm from mouth to bottom of bulb, with a volume of about 25 mlters. Bulb contains tissue under study, 0.96 mlter of Ringer's phosphate solution plus glucose in a concentration of 200 mg per cent, and the gas mixture being studied. Neck contains a layer of KOH to absorb CO₂ produced by the respiring blood vessel, and one or two layers of oil to prevent loss of water vapor to the surrounding flotation medium. Tail of the diver, below the bulb, is solid glass and includes four pieces of colored glass for identification, since the volume of each diver is unique. Upper end of diver is open, and is filled with the same medium in which it floats.

Gen consumed by the tissue, V, was given by

$$V = V_o \times P/P_o$$

where \(V_o\) was initial gas volume of the diver, \(P_o\) the initial pressure in the diver (atmospheric) and \(P\) the average hourly change in pressure, determined from the graph of manometer pressure versus time. The apparatus contained six tubes so that five samples of tissue plus a control could be studied at the same time.

**MEASUREMENT OF TISSUE WEIGHT**

After measurement of oxygen consumption was complete, tissue specimens were dehydrated in an oven. Constant tissue weight could be obtained if the sample was air-dried overnight, then heated to 115°C for two hours. The tissues were hydrophilic; it was necessary to weigh the samples promptly when cool on sunny days, there being progressive weight gain if measurements were made on rainy days.

The dried tissue was weighed with a quartz-fiber microbalance made as described by Lowery. Vertical displacements of the fiber were measured by a horizontal telescope mounted on a vertical worm thread calibrated directly in divisions of 0.001 inch. Readings were taken by lowering the telescope and superimposing the cross hair in the eyepiece on the end of the quartz fiber. The average of 10 readings of the displacement of the tare plus sample was subtracted from the average of 10 readings of displacement of the tare alone to find the displacement caused by the tissue. The fiber was calibrated by observing the displacement resulting from known weights of very fine wire (Belden no. 8046). One meter of the wire was weighed on a laboratory analytical balance. Short segments, whose lengths were carefully measured with a stage micrometer and microscope, were used to produce vertical displacement of the quartz fiber, the displacement being measured by the same technique used for tissue samples. Vertical deflection of the quartz fiber was linear with respect to weight over the range of deflection used in this study. The sensitivity of the balance was 4.28 μg/0.01 inch. Although the position of the telescope could be read reproducibly to 0.0005 inch, the width of
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its hairlines and refraction of light by the quartz fiber prevented reproducible estimation of displacement less than 0.005 inch, equivalent to ± 1 μg, or 4 to 15% of usual sample weight. Dried tissue samples weighing less than 5 μg were rejected. The samples reported ranged from 6 to 65 μg in weight, the great majority being 15 to 50 μg.

Tissue
Vascular tissue was obtained from the cheek pouch or mesentery of an inbred strain of albino hamster. The animals were mature (more than 60 days old), and weighed 115 to 170 g. Anesthesia was produced by intraperitoneal sodium pentobarbital (15 mg/100 g body wt).

The diameter of the blood vessel selected for study was measured by means of a reticle in the eyepiece of a stereomicroscope. The tissue was manipulated as little as possible prior to determination of size in order to avoid the well-known contractile response of vessels to mechanical stimulation. After determination of its diameter in situ, the vessel was freed by gentle blunt dissection from surrounding connective tissue and fat. A tubular length of vessel was excised, transferred to a Petri dish filled with Ringer’s solution, and inspected under the microscope. Residual fat was removed with fine forceps and the vessel was stroked gently to express red cells from its lumen. The piece of vessel was then transferred to the Cartesian diver without further alteration.

Human vascular tissue was obtained from a biopsy of the omentum or mesoappendix taken at laparotomy under general anesthesia. An interval of no more than 15 minutes passed from the time of excision of the sample until dissection of the blood vessels was begun.

Primary consideration in this study has been given to vessels free of vasa vasorum (i.e., less than 1 mm diameter). These vessels can presumably achieve adequate nutrition by diffusion alone. Residual fat of the same vessel averaged 0.13 μliter per hour. The total gas volume in the diver of 0.007 to 0.04 μliter per hour. The total gas volume of the smaller value of 25 mm of Brodie’s solution per hour. Tissue samples consumed oxygen at rates producing change of pressure difference in the U-tube manometer of 5 to 25 μm of Brodie’s solution per hour, or change in volume of gas in the diver of 0.007 to 0.04 μliter per hour. The total gas volume in the divers averaged 20 μliters.

Results

I. Reproducibility
Oxygen consumption of a vascular segment, measured in one diver, was compared with that of a contiguous segment of the same vessel, measured at the same time in another diver. As shown in table 1, the difference in oxygen consumption in 13 such pairs of segments of the same vessel averaged 0.13 μliter/mg/hr, or 27% of the smaller value of O₂ consumption.

II. Effect of Variation in Vascular Caliber (Table 2)
Divers averaged 20 μliters.

Results
Oxygen consumption was measured in small arteries with outside diameters ranging from 65 to 400 μ (fig. 3), and in small veins with diameters of 150 to 450 μ. Twenty arterioles with in vivo diameters 150 μ or less consumed oxygen at an average rate of 1.05 μliter/mg/dry tissue/hr. Twenty-four arterioles with diameters greater than 250 μ consumed oxygen at the significantly (P < 0.01) lower average rate of 0.58 μliter/mg/hr. Twenty-four arterioles of intermediate diameter (150 to 250 μ) showed intermediate rates of oxygen consumption averaging 0.85 μliter/mg/hr, not significantly different from the arterioles whose diameters were less than 150 μ.
TABLE 1

Comparison of Oxygen Consumption of Two Contiguous Segments of Same Artery

<table>
<thead>
<tr>
<th>Segment 1</th>
<th>Segment 2</th>
<th>Oxygen consumption</th>
<th>Difference</th>
<th>Difference*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diameter</td>
<td>µ</td>
<td>µ</td>
<td>µ/liter/mg/hr</td>
<td>µ/liter/mg/hr</td>
</tr>
<tr>
<td>330</td>
<td>220</td>
<td>0.64, 0.62</td>
<td>0.02</td>
<td>3.</td>
</tr>
<tr>
<td>290</td>
<td>220</td>
<td>0.77, 0.88</td>
<td>0.11</td>
<td>14.</td>
</tr>
<tr>
<td>260</td>
<td>220</td>
<td>0.24, 0.13</td>
<td>0.11</td>
<td>85.</td>
</tr>
<tr>
<td>250</td>
<td>220</td>
<td>0.28, 0.42</td>
<td>0.14</td>
<td>50.</td>
</tr>
<tr>
<td>242</td>
<td>220</td>
<td>1.31, 1.58</td>
<td>0.27</td>
<td>21.</td>
</tr>
<tr>
<td>220</td>
<td>220</td>
<td>0.67, 0.81</td>
<td>0.14</td>
<td>21.</td>
</tr>
<tr>
<td>198</td>
<td>198</td>
<td>0.45, 0.41</td>
<td>0.04</td>
<td>10.</td>
</tr>
<tr>
<td>154</td>
<td>154</td>
<td>1.13, 0.87</td>
<td>0.26</td>
<td>30.</td>
</tr>
<tr>
<td>260</td>
<td>220</td>
<td>1.27, 1.33</td>
<td>0.06</td>
<td>5.</td>
</tr>
<tr>
<td>290</td>
<td>280</td>
<td>1.46, 1.29</td>
<td>0.17</td>
<td>13.</td>
</tr>
<tr>
<td>260</td>
<td>280</td>
<td>0.39, 0.55</td>
<td>0.16</td>
<td>41.</td>
</tr>
</tbody>
</table>

Table:

* Larger O₂ consumption value minus smaller value X 100.

TABLE 2

Metabolism of Arterioles of Differing Diameters, and of Venules; Pentobarbital Anesthesia, 20.9% O₂

<table>
<thead>
<tr>
<th>Vessels</th>
<th>In vivo diameter</th>
<th>Number studied</th>
<th>Mean oxygen uptake</th>
<th>Standard deviation</th>
<th>0.9 Confidence limits</th>
<th>Extreme values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arteries</td>
<td>µ</td>
<td>µ/liter/mg/hr</td>
<td>µ/liter/mg/hr</td>
<td>µ/liter/mg/hr</td>
<td>µ/liter/mg/hr</td>
<td>µ/liter/mg/hr</td>
</tr>
<tr>
<td>65—150</td>
<td>20</td>
<td>1.05</td>
<td>0.66</td>
<td>0.81—1.29</td>
<td>0.25—2.76</td>
<td></td>
</tr>
<tr>
<td>150—250</td>
<td>22</td>
<td>0.95</td>
<td>0.47</td>
<td>0.79—1.11</td>
<td>0.37—2.25</td>
<td></td>
</tr>
<tr>
<td>250—445</td>
<td>24</td>
<td>0.58</td>
<td>0.38</td>
<td>0.44—0.72</td>
<td>0.12—1.46</td>
<td></td>
</tr>
<tr>
<td>Veins</td>
<td>154—445</td>
<td>29</td>
<td>0.53</td>
<td>0.38</td>
<td>0.42—0.66</td>
<td>0.05—1.58</td>
</tr>
</tbody>
</table>

Arterioles 60 to 100 µ in diameter were the first and second branches actually lying on the intestine of the hamster. Arteries 300 to 400 µ in size were located between the fourth or fifth bifurcation proximal to the gut, and between the third to fifth bifurcation distal to the first division of the superior mesenteric artery.

The metabolism of 24 hamster venules from 13 animals averaged 0.53 µliter/mg/hr, significantly less than that of the arterioles as a group, but in the range of the largest arterioles examined. Rate of oxygen uptake was not related to venular diameter in the size range from 150 to 450 µ. The smallest veins studied were the first branches on the wall of the intestine. Venules located three or four branches proximal to the gut had a diameter range of 250 to 350 µ. The largest veins studied were one to three branches more proximal still, and three to four branches distal to the first bifurcation of the superior mesenteric vein.

III. CHANGES IN VASCULAR METABOLISM INDUCED BY HYPOXIA (TABLE 3)

Both arterioles and venules were highly resistant to hypoxia. (Table 3). No significant change in the metabolism of hamster arterioles 150 to 250 µ in diameter occurred despite reduction, from 150 to 12 mm Hg, in the oxygen tension of the gas to which the arterioles were exposed (Fig. 4). At an oxygen tension of 5 mm Hg, however, arteriolar oxygen consumption averaged 65% of its value at 12 mm Hg (P < 0.1). An oxygen tension of 2 mm Hg was associated with further reduction in metabolism, which averaged 57% (P < 0.05) of the average oxygen uptake at an environmental oxygen tension of 12 mm Hg.

Venules resisted hypoxia to about the same
degree (fig. 4). At an oxygen tension of 5 mm Hg, the rate of oxygen uptake of hamster venules 200 to 400 μ in diameter averaged 78% of its value at 12 mm Hg, the difference being statistically insignificant (0.2 < P < 0.3). At 2 mm Hg, venular oxygen consumption averaged 40% of its value at 12 mm Hg (P < 0.02). Surprisingly, at an oxygen tension of 150 mm Hg, the average oxygen consumption of 29 venules was 40% (P < 0.05) lower than that of venules of similar size at oxygen tensions of 57, 28, and 12 mm Hg.

Neither arterioles nor venules "consumed oxygen," that is, caused volume change, in di-

![Graph](image)

**FIGURE 3**

Relation between arterial diameter and oxygen consumption. Each point represents one arterial segment. Each animal provided two or three segments.

**TABLE 3**

Metabolism of Arterioles and Venules in Environmental pO₂ from 150 to 2 mm Hg

<table>
<thead>
<tr>
<th>Environmental pO₂</th>
<th>Environmental pO₂</th>
<th>Number studied</th>
<th>Mean oxygen uptake</th>
<th>Standard deviation</th>
<th>0.9 Confidence limits</th>
<th>P vs. 12 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>mm Hg</td>
<td>μ</td>
<td></td>
<td>mliter/mg/hr</td>
<td>mliter/mg/hr</td>
<td>mliter/mg/hr</td>
<td></td>
</tr>
<tr>
<td>150</td>
<td>150—250</td>
<td>22</td>
<td>0.95</td>
<td>0.47</td>
<td>0.79—1.11</td>
<td>NS</td>
</tr>
<tr>
<td>57</td>
<td>130—240</td>
<td>17</td>
<td>0.86</td>
<td>0.46</td>
<td>0.71—1.06</td>
<td>NS</td>
</tr>
<tr>
<td>28</td>
<td>130—200</td>
<td>6</td>
<td>0.81</td>
<td>0.25</td>
<td>0.65—0.97</td>
<td>NS</td>
</tr>
<tr>
<td>12</td>
<td>155—320</td>
<td>16</td>
<td>1.08</td>
<td>0.84</td>
<td>0.81—1.32</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>155—220</td>
<td>11</td>
<td>0.89</td>
<td>0.41</td>
<td>0.49—0.89</td>
<td>&lt;0.10</td>
</tr>
<tr>
<td>2</td>
<td>155—250</td>
<td>13</td>
<td>0.81</td>
<td>0.45</td>
<td>0.41—0.80</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>150</td>
<td>150—445</td>
<td>29</td>
<td>0.53</td>
<td>0.38</td>
<td>0.42—0.66</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>57</td>
<td>330—550</td>
<td>15</td>
<td>0.90</td>
<td>0.58</td>
<td>0.86—1.14</td>
<td>NS</td>
</tr>
<tr>
<td>28</td>
<td>267—445</td>
<td>9</td>
<td>0.98</td>
<td>0.72</td>
<td>0.82—1.35</td>
<td>NS</td>
</tr>
<tr>
<td>12</td>
<td>330—660</td>
<td>15</td>
<td>0.94</td>
<td>0.65</td>
<td>0.67—1.22</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>360—440</td>
<td>11</td>
<td>0.70</td>
<td>0.37</td>
<td>0.32—0.87</td>
<td>&lt;0.20</td>
</tr>
<tr>
<td>2</td>
<td>330—540</td>
<td>10</td>
<td>0.36</td>
<td>0.34</td>
<td>0.18—0.54</td>
<td>&lt;0.02</td>
</tr>
</tbody>
</table>

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vers filled with pure nitrogen, a comforting check on the validity of the technique used.

IV. METABOLIC EFFECT OF ANESTHESIA (TABLE 4)

Barbiturates and other anesthetic agents reduce oxygen consumption of liver slices. To determine the influence of sodium pentobarbital on metabolism of small vessels, eight hamsters were sacrificed by decapitation or sudden head trauma without anesthesia. Twenty-one mesenteric arterioles and 13 venules, removed rapidly and studied subsequently by methods identical with those used in anesthetized animals, consumed oxygen at insignificantly lower rates than did similar vessels from animals under barbiturate anesthesia. Oxygen consumption of arterioles from unanesthetized hamsters averaged 0.8 μliter/mg/hr; that of venules averaged 0.42 μliter/mg/hr.

Though the vessels were removed from the unanesthetized hamsters within five minutes after death, marked vasospasm was obvious when the vessels were first inspected. The diameter of arterioles and venules from sacrificed hamsters averaged one-half that of vessels at similar locations in living, anesthetized animals. This decrease of diameter resulted presumably from the hemorrhage accompanying decapitation and head trauma or from change in central nervous control. The change of vascular diameter, absence of intraluminal pressure, and diminished central nervous control were obvious differences in experimental conditions which may have obscured variations of metabolism caused by the anesthetic.

V. METABOLISM OF HUMAN VESSELS (TABLE 4)

Ten arterial and ten venous vessels from five normal mesoappendices, removed during tubal ligation under barbiturate-cyclopropane anesthesia, showed lower rates of oxygen me-
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abolism than did hamster vessels. Human arteries whose in vitro diameters ranged from 65 to 175 \( \mu \) consumed 0.35 \( \mu \)liter of O\(_2\)/mg/hr, about 35\% of the rate of oxygen uptake of hamster arterioles (\( P < 0.01 \)). Human appendicular veins, 135 to 450 \( \mu \) in diameter (in vitro), consumed an average of 0.20 \( \mu \)liter of O\(_2\)/mg/hr, about 30\% of the consumption of hamster venules (\( P < 0.01 \)).

Discussion

The variability of oxygen consumption in each range of arterial sizes (fig. 3) prevents absolute conclusion about the relation between arterial diameter and rate of oxygen uptake. The highest metabolic rates were observed in arteries less than 250 \( \mu \) in diameter, and it seems probable that small arterioles utilize oxygen at greater rates, per unit of tissue dry weight, than do larger arterial vessels.

Of possible explanations for the observed higher metabolic rate of smallest arteries, several seem unlikely. Differences in experimental conditions which would produce erroneous overestimates are not apparent. The increased trauma associated with dissection of the smallest arteries would be expected a priori to diminish their metabolic activity. Longer segments of the smallest vessels were placed in the divers, so that the rates of change of diver volume and the dry weights of the vessels of least diameter fell in the same range as the larger arteries.

The higher metabolic rate of the smallest arterioles could be explained by greater amounts of muscle in the small vessels. We have studied vascular muscle thickness in the hamster by microscopic examination of arteries between each bifurcation from aorta to intestinal wall in two hamsters, using hematoxylin-eosin, Masson trichrome, and Verhoeff-van Gieson stains. Hamster arteriolar vessels with diameters of 65 to 150 \( \mu \) showed proportionately less elastic tissue and more muscle in their walls than did the aorta, but no definite histologic difference was discerned between these arterioles and arteries 250 to 400 \( \mu \) in diameter. Vasa vasorum were present in hamster aortas and superior mesenteric arteries, the latter vessels averaging 900 \( \mu \) in diameter, but these nutrient channels were not found in smaller vessels. Explanation of the higher metabolism of the smallest arterioles on the basis of differences of muscle content or of presence or absence of vasa vasorum therefore seems untenable.

If the surface area available for oxygen diffusion were a limiting factor in oxygen up-

<table>
<thead>
<tr>
<th>Source</th>
<th>Anesthesia</th>
<th>Diameter*</th>
<th>Number studied</th>
<th>Mean oxygen uptake</th>
<th>Standard deviation</th>
<th>0.9 Confidence limits</th>
<th>( P ), vs. line 1</th>
<th>Extreme values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterioles</td>
<td></td>
<td>( \mu )</td>
<td>( \mu )liter/mg/hr</td>
<td>( \mu )liter/mg/hr</td>
<td>( \mu )liter/mg/hr</td>
<td>&lt;0.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamster</td>
<td>Pentobarbital</td>
<td>150–250</td>
<td>22</td>
<td>0.95</td>
<td>0.47</td>
<td>0.79–1.11</td>
<td>0.37–2.25</td>
<td></td>
</tr>
<tr>
<td>Hamster</td>
<td>None</td>
<td>65–150</td>
<td>21</td>
<td>0.80</td>
<td>0.42</td>
<td>0.85–0.95</td>
<td>&lt;0.30</td>
<td>0.37–1.86</td>
</tr>
<tr>
<td>Human</td>
<td>Pentobarbital and cyclopropane</td>
<td>65–175</td>
<td>10</td>
<td>0.35</td>
<td>0.18</td>
<td>0.27–0.45</td>
<td>&lt;0.01</td>
<td>0.16–0.8</td>
</tr>
<tr>
<td>Venules</td>
<td></td>
<td>( \mu )</td>
<td>( \mu )liter/mg/hr</td>
<td>( \mu )liter/mg/hr</td>
<td>( \mu )liter/mg/hr</td>
<td>&lt;0.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamster</td>
<td>Pentobarbital</td>
<td>150–445</td>
<td>29</td>
<td>0.53</td>
<td>0.38</td>
<td>0.41–0.66</td>
<td>&lt;0.05</td>
<td>0.05–1.58</td>
</tr>
<tr>
<td>Hamster</td>
<td>None</td>
<td>150–330</td>
<td>13</td>
<td>0.42</td>
<td>0.25</td>
<td>0.31–0.53</td>
<td>&lt;0.40</td>
<td>0.19–0.99</td>
</tr>
<tr>
<td>Human</td>
<td>Pentobarbital and cyclopropane</td>
<td>130–450</td>
<td>10</td>
<td>0.20</td>
<td>0.15</td>
<td>0.13–0.28</td>
<td>&lt;0.01</td>
<td>0.12–0.26</td>
</tr>
</tbody>
</table>

*Outside diameter, measured before vessels were dissected in all experiments, but after appendix removed from body in human studies. See Results.
take by larger arteries, one would expect a higher ratio of surface area to wall volume in the smaller arterial vessels. In vitro measurements of inside and outside diameters of rings of arteries, ranging from 175 to 275 \( \mu \) in outside diameter, did indicate an increase in surface area relative to wall volume, as arteries of smaller and smaller size were studied. Total (inside and outside) surface area to wall-volume ratio in an artery 260 \( \mu \) in diameter was 1.0; in a more distal branch of the same artery, 175 \( \mu \) in diameter, the ratio was 1.15. Comparable ratios were found in three other arteries. It seems unlikely, however, that surface area represents the critical limiting factor in oxygen transport to cells of resting arterial vessels, in view of Kirk and Laursen's demonstration\(^{15}\) that the thickness of human aortic intimal tissue which can be supplied with oxygen by diffusion is 1000 \( \mu \), twice the total diameter of the largest vessels in our study. Furthermore, there is no reason to suppose that both the inner and the outer surfaces of the vessels in the diver are not available for oxygen diffusion. Biochemical differences that might explain the higher metabolism of the smallest arterioles have not yet been explored.

The resistance of nonworking arterioles to hypoxia is surprising. If these in vitro results are applicable to the intact animal, arterial metabolism would never be limited by general hypoxemia under conditions compatible with survival of the organism. Since small arteries in the hamster have apparently no vasa vasorum, it might be supposed that adventitial layers ordinarily "work" at lower oxygen tension than intimal layers in vivo, and since luminal and adventitial surfaces of arteries in the diver are exposed to environmental oxygen tension, that the observed resistance to hypoxia is the result of the experimental technique. Bronk and colleagues\(^{17}\) observed, however, that oxygen tension on the outer surface of brain arterioles (cat) approached 100 mm Hg, and calculations based on A. V. Hill's\(^{18}\) determination of maximum thickness of tissue through which \( O_2 \) can diffuse suggest diffusion of oxygen through hamster arterial segments 600 \( \mu \) in thickness, over twice the greatest wall thickness found in any of the vessels we studied. This suggests that the \( pO_2 \) of the adventitial surface of arterioles is high in the intact animal, and that the relationship to environmental oxygen tension of arterial segments in the Cartesian diver does not differ greatly from that of arterioles in vivo.

Venular metabolism was about as resistant to severe hypoxia as that of arterioles, appearing more resistant than arterioles at \( pO_2 \) of 5 mm Hg, but less so at 2 mm Hg. At higher environmental oxygen concentrations, on the other hand, in vitro venular oxygen consumption was depressed (fig. 4). There was lower oxygen consumption at an oxygen tension of 150 mm Hg than at tensions between 12 and 60 mm Hg, a finding of interest since venules in the intact animal are exposed to an environmental oxygen tension in the range of 35 to 50 mm Hg, about 50 mm Hg lower than that of arterioles. Further observations on venular metabolism in environments rich in oxygen are needed before firm conclusions can be reached.

Total oxygen consumption (\( Q_o_2 \)) in many species has been shown by others to be related to environmental oxygen tension in a fashion similar to that observed in our experiments on arterioles.\(^8\) \( Q_o_2 \) is constant at oxygen tensions between 150 mm Hg and some critical \( pO_2 \), usually below 50 mm Hg, where sudden reduction in oxygen consumption occurs. These species include some protozoa, annelids (critical \( pO_2 \) 40 mm Hg), Trichinella (8 mm Hg), crabs (4 mm Hg), and "all terrestrial vertebrates."

The higher metabolic rates of hamster vessels, compared to the rates of their counterparts in man, seem best attributed to species difference. Though the diameters of human vessels were measured after excision of the appendix, in contrast to the in vivo measurement of hamster vessels, the latter shrank only 10% on removal from the peritoneum to Ringer's solution. It seems unlikely, therefore, that the small difference in method of estimation of vessel size would explain the large differ-
ence found between human and hamster oxygen consumption. Since metabolic rate was constant for at least four hours in every experiment reported, the 15 to 35 minutes of delay between excision of the appendix and initial measurement of oxygen consumption also seems an unlikely explanation for the observed differences between human vascular metabolism and that of the hamster.

Comparison of our results with data of other workers is not possible for arterioles or venules, nor for larger vessels in the hamster. Average oxygen consumption by rat aortic slices is 1.0 μliter/mg/hr. Human aortic slices obtained postmortem consume 0.37 μliter/mg/hr. In general, aortic metabolic rate shows a rough inverse relationship to body weight. These in vitro vascular metabolic rates contrast with those of rat liver (8-10 μliter/mg/hr), rat kidney (18-24 μliter/mg/hr), and rat heart (10 ± 0.72 μliter/mg/hr).

**Summary**

1. The Cartesian diver microrespirometer has been applied to the measurement of oxygen consumption by mesenteric arterioles and venules from hamsters and human beings. Oxygen consumption by the smallest arterial vessels, less than 150 μ in diameter, averaged 1.05 μliter/mg/hr, in contrast to only 0.58 μliter/mg/hr for arteries 250 to 400 μ in diameter. Hamster venules consumed oxygen at an average rate of 0.53 μliter/mg/hr, nearly equal to the largest arteries studied. Venous oxygen consumption did not vary with vessel diameter.

2. Both arterioles and venules were highly resistant to hypoxia, maintaining normal rates of oxygen uptake at environmental oxygen tensions as low as 12 mm Hg, but not at 5 or 2 mm Hg. Veins consumed less oxygen at an environmental pO₂ of 150 mm Hg than at 57, 28, or 12 mm Hg, whereas arterial QO₂ was unchanged between 12 and 150 mm Hg.

3. Ten human mesenteric arteries, 65 to 175 μ in diameter, and 10 human veins, 130 to 450 μ in vitro, had average rates of oxygen consumption of 0.35 and 0.25 μliter/mg/hr, significantly lower than hamster vessels.

4. Vessels from hamsters sacrificed without anesthesia consumed oxygen at rates similar to arterioles and venules removed from animals under pentobarbital anesthesia.

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**References**


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