Oxygen Consumption of the Thoracic Aorta of Normal and Hypercholesteremic Rats

By F. J. Loomeijer, Ph.D., and J. P. Oostendorf, B.Sc.

The respiratory activity of the thoracic aorta wall of hypercholesteremic rats was shown to be 30 to 40 per cent higher than in normal animals. The addition, in vitro, of serum of different cholesterol content did not affect the oxygen consumption.

According to Christie and Dahl¹ the abdominal portion of the rat aorta shows a significantly lower respiration than its thoracic segment. The divergence increases with aging. Briggs, Chernick and Chaikoff,² in an earlier study, did not detect this phenomenon. The first authors suggest that the preferential sites of development of atherosclerotic lesions might show a lower rate of respiration. This suggestion prompted a study of the oxygen uptake of aortic tissue under conditions favorable for the development of atheromatosis.

**METHOD**

Albino rats, 3 to 4 months old, of both sexes, and weighing 180 to 280 Gm. were killed by decapitation. The thoracic aorta was rapidly removed and transferred at room temperature to a Ringer-phosphate solution of pH 7.4, containing 0.2 per cent glucose and saturated with oxygen. Adhering tissue and blood were removed, the aortas were cut open and the resulting sheets cut into an upper and a lower half. The thoracic aortas of five rats were prepared in this way. Two portions of tissue, each weighing 60 to 100 mg., were collected by assembling three upper and two lower parts and the remaining two upper and two lower parts. The two portions were blotted between filter paper, weighed and immediately transferred to two Warburg flasks containing 2.7 ml. of the Ringer-phosphate medium. Every Warburg flask ("experiment" in table 1) thus contained the mixed thoracic aortas of 2½ rats. Sex was disregarded in these experiments.

The flasks were flushed with oxygen in the constant temperature bath at 37.0 C. for 5 min., followed by a 15 min. period to attain temperature equilibrium. Carbon dioxide was absorbed by 0.2 ml. 10 per cent potassium hydroxide. Serum or other agents were added in 0.3 ml. amounts from the side arm after the respiration had been measured for 60 min. The first reading was made 60 to 70 min. after the decapitation of the first rat. Subsequent readings were taken every 60 min. QO₂ was calculated as microliters of oxygen consumed per milligram of dry tissue per hour. Dry tissue weight was estimated at 29.9 ± 0.2 per cent of the wet weight. No difference in water content between the aortas of control and those of hypercholesteremic rats was found.

The cholesterol level of the plasma of rats was raised by adding to the stock diet 2 per cent cholesterol and 1 per cent cholic acid. Great individual differences were noticed in the cholesterol values of the hypercholesteremic rats. Sera of normal and hypercholesteremic animals were pooled separately and stored at a temperature of −40 C.

**RESULTS**

The results of seven series of experiments are summarized in table 1. It was found, in agreement with Briggs et al.,² that the oxygen uptake remains fairly constant for at least three hours after the start of the experiment, although in nearly all cases a slightly increased respiration rate was shown during the second hour. The aorta of hypercholesteremic rats (series G and H) showed a remarkably increased oxygen consumption. The addition of serum at the end of the first hour of incubation did not affect the respiratory activity of the arterial tissue at all, consequently no influence of the cholesterol content of the serum could be detected.

**DISCUSSION**

Several authors¹,³ have reported that the QO₂ of arterial tissue is inversely proportional to the age of the animal. The anatomic
### Table 1—Oxygen Consumption of Normal and Hypercholesteremic Rat Aortas Under Different Conditions

<table>
<thead>
<tr>
<th>Series</th>
<th>Number of experiments</th>
<th>Aortas of*</th>
<th>Substrate added after 60 min. incub.</th>
<th>Cholesterol level in substrate</th>
<th>( \text{QO}_2 )† 1st hour</th>
<th>( \text{QO}_2 )† 2nd hour</th>
<th>( \text{QO}_2 )† 3rd hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>8</td>
<td>N</td>
<td>Ringer-phosph.</td>
<td>0</td>
<td>1.37 ± 0.02</td>
<td>1.91 ± 0.03</td>
<td>1.29 ± 0.04</td>
</tr>
<tr>
<td>B</td>
<td>13</td>
<td>N</td>
<td>rat serum</td>
<td>60</td>
<td>1.43 ± 0.06</td>
<td>1.54 ± 0.06</td>
<td>1.45 ± 0.07</td>
</tr>
<tr>
<td>C</td>
<td>12</td>
<td>N</td>
<td>rat serum§</td>
<td>160</td>
<td>1.37 ± 0.04</td>
<td>1.53 ± 0.06</td>
<td>1.43 ± 0.05</td>
</tr>
<tr>
<td>D</td>
<td>4</td>
<td>N</td>
<td>rat serum§</td>
<td>700</td>
<td>1.34 ± 0.04</td>
<td>1.48 ± 0.04</td>
<td>1.40 ± 0.02</td>
</tr>
<tr>
<td>E</td>
<td>12</td>
<td>N</td>
<td>rabbit serum</td>
<td>125</td>
<td>1.37 ± 0.04</td>
<td>1.48 ± 0.04</td>
<td>1.36 ± 0.04</td>
</tr>
<tr>
<td>F</td>
<td>12</td>
<td>N</td>
<td>rabbit serum</td>
<td>1000</td>
<td>1.34 ± 0.04</td>
<td>1.56 ± 0.03</td>
<td>1.35 ± 0.03</td>
</tr>
<tr>
<td>G</td>
<td>8</td>
<td>C</td>
<td>rat serum</td>
<td>65</td>
<td>1.88 ± 0.15</td>
<td>1.86 ± 0.13</td>
<td>1.84 ± 0.11</td>
</tr>
<tr>
<td>H</td>
<td>7</td>
<td>C</td>
<td>rat serum§</td>
<td>160</td>
<td>1.74 ± 0.10</td>
<td>1.87 ± 0.07</td>
<td>1.74 ± 0.02</td>
</tr>
</tbody>
</table>

*Normal rats, serum cholesterol level 60-70 mg. per cent; Hypercholesteremic rat, serum cholesterol 150-180 mg. per cent.
†Mean microliters \( \text{O}_2 \)/mg. dry weight/hour.
§Two per cent cholesterol and 1 per cent cholic acid added to stock diet.

Site of the artery used in the experiment also affected the \( \text{QO}_2 \) and the suggestion was made that a decreased rate of respiration of the arterial wall and the sensitivity of the tissue for the development of atherosclerosis might be related.

From the study presented here it is obvious that the animal responds to a diet rich in cholesterol and cholic acid administered for 6 to 7 weeks with an increase of its aortic respiratory activity of 30 to 40 per cent over its original value. This finding does not confirm or contradict the views of the authors mentioned. Further research is necessary to show that the increase of the \( \text{QO}_2 \) is a result of a real defense mechanism of the arterial wall against injury by the high cholesterol level of the serum.

**Summary**

The oxygen consumption of the rat thoracic aorta was measured under different conditions. The arterial tissue of hypercholesteremic animals showed a significantly higher respiration rate than aortas of normal individuals. The oxygen uptake was not affected by the addition of hypercholesteremic serum in vitro.

**Summario in Interlingua**

Le consumption de oxygeno del aorta thoracica de ratos esseva mesurata sub varie condiçones. Le histos arterial de animalia hypercholesterolemic exhibeva un significativamente plus intense respiration que le aortas de individuos normal. Le aceptation de oxygeno non esseva afficite per le addition de sero hypercholesterolemic in vitro.

**References**

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