Ovarian blood flow in compensatory hypertrophy in the rat

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Unilateral ovariectomy is followed by compensatory hypertrophy of the contralateral ovary in the rat (Carmichael & Marshall, 1908; Arai, 1920; Slonaker, 1927). This phenomenon is induced by the increased gonadotrophic hormone release resulting from the decrease of the oestrogen level via a negative feedback mechanism (Heller, Heller & Sevinghaus, 1942; Edgren, Parlow, Peterson & Jones, 1965; Johnson, 1966; Peppier & Greenwald, 1970). After hemiovariectomy the contralateral ovary produces the same number of ova as did both ovaries in the control animals (Peppier, 1972). Of the gonadotrophic hormones, LH increases ovarian blood flow and, since ovarian blood flow is known to increase during oestrus (Wurtman, 1964), the present study was to determine whether the elevated gonadotrophic hormone level resulting from hemiovariectomy increased blood flow in the remaining ovary.

Rats of the CFY strain and weighing 170–200 g were randomly allocated to two groups. Under ether anaesthesia, those in Group I were ovariectomized on the left side, and those in Group II were subjected to laparotomy only. On Days 6, 12 and 18 after hemiovariectomy, the animals were anaesthetized with 35 mg sodium pentobarbitone (May and Baker Ltd, England)/kg body wt, and 5 µCi 86RbCl were injected by a thin needle into the jugular vein. The 86Rb method of Sapirstein (1958) was applied to determine the ovarian fraction of cardiac output. This method is useful and accurate to measure blood flow to the ovary (Satchell & Linzell, 1974), but it may underestimate blood flow to the CL in sheep (Brown, Hales & Mattner, 1974) as compared to microsphere techniques. At 30 sec after the injection, 0·3 ml 1 % Evans blue solution was injected by the same route to determine cardiac output (Hamilton, Moore, Kimmsman & Spurling, 1932). Blood pressure was checked in the femoral artery with a Statham’s pressure transducer. The animals were killed by an i.v. injection of a supersaturated KCl solution. The ovary was removed, cleaned, weighed and dissolved in sodium hydroxide. Radioactivity was measured with a Packard Tri-Carb scintillation spectrometer. Analysis of variance was used for statistical evaluation (Scheffé, 1959).

There were no differences in the body weights and blood pressure values of the ovariectomized and control rats. After left hemiovariectomy, the weight of the right ovary significantly increased ($P<0·01$), the increase being most marked on Day 12 (Table 1). However, the fraction of cardiac output and the blood flow of the hypertrophic ovary did not differ from those of the controls (Table 1).

### Table 1. The effect of left hemiovariectomy or laparotomy only on the mean (±S.E.M.) weight of the right ovary, fractional perfusion and ovarian blood flow of rats (nos in parentheses)

<table>
<thead>
<tr>
<th></th>
<th>Ovarian wt (mg/100 g body wt)</th>
<th>Cardiac output (%/g ovarian wt)</th>
<th>Ovarian blood flow (ml/min/g)</th>
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</thead>
<tbody>
<tr>
<td><strong>Control rats</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 6</td>
<td>20.5 ± 1.49 (14)</td>
<td>1.386 ± 0.059 (12)</td>
<td>0.813 ± 0.061 (9)</td>
</tr>
<tr>
<td>Day 12</td>
<td>21.8 ± 0.99 (12)</td>
<td>1.363 ± 0.110 (12)</td>
<td>1.000 ± 0.174 (8)</td>
</tr>
<tr>
<td>Day 18</td>
<td>20.7 ± 1.22 (13)</td>
<td>1.654 ± 0.206 (13)</td>
<td>0.988 ± 0.117 (9)</td>
</tr>
<tr>
<td><strong>Hemiovariectomized rats</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Day 6</td>
<td>25.3 ± 1.23 (12*)</td>
<td>1.275 ± 0.060 (10)</td>
<td>0.777 ± 0.036 (7)</td>
</tr>
<tr>
<td>Day 12</td>
<td>32.6 ± 1.12 (14*)</td>
<td>1.237 ± 0.086 (13)</td>
<td>0.832 ± 0.094 (12)</td>
</tr>
<tr>
<td>Day 18</td>
<td>27.9 ± 1.25 (12*)</td>
<td>1.488 ± 0.114 (12)</td>
<td>1.004 ± 0.083 (10)</td>
</tr>
</tbody>
</table>

* Significant difference between control and hemiovariectomized rats, $P<0·01$.  

409
Our results show that following hemiovariectomy the blood supply of the contralateral ovary expands in proportion to hypertrophy and increases only to the extent necessary to reach the control level of blood flow/unit weight calculated for the organ. The specific increase of blood flow does not appear to be necessary for maintaining hypertrophy and increased ovulation, and indicates that the elevated gonadotrophic hormone level found after hemiovariectomy (Heller et al., 1942; Edgren et al., 1965; Johnson, 1966; Peppler & Greenwald, 1970) does not affect the blood supply of the surviving ovary. Our data do not, however, preclude the possibility of changes in ovarian blood flow immediately after hemiovariectomy that might be correlated with the increased production of gonadotrophic hormones as a general circulatory reaction to ‘surgical trauma’. Wurtman (1964) was unable to obtain an increase in ovarian blood flow in rats by giving FSH, and LH was only effective if given in high doses. In anoestrous dogs both LH and FSH proved to be ineffective (Stark & Varga, 1968). Our results seem to support the suggestion that unilateral ovariectomy increases only the FSH level in peripheral blood and fails to increase the LH concentration (Benson, Sorrentino & Evans, 1969; Howland, Jack & Beaton, 1974).

References


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