Effects of Ejaculation on Levels of Testosterone, Cortisol,
and Luteinizing Hormone in Peripheral
Plasma of Rhesus Monkeys

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Plasma levels of testosterone (T), luteinizing hormone (LH), and cortisol were measured in 10 adult male rhesus monkeys before and shortly after coitus. Mean levels of T and LH did not increase significantly after coitus or in control (no ejaculation) tests, but cortisol levels did in both cases. In 10 different males, no significant change was found in the plasma levels of T after electroejaculation; but in control tests (electric current withheld), the mean level of T was significantly lower at 80 and 140 min, but not at 50 min, after the test. According to present evidence, the effect of ejaculation on T levels differs in primate and nonprimate species. The effects on T levels produced by living with sexually receptive female rhesus monkeys may differ from those produced by intimate but brief contact with them.

In several nonprimate species, the levels of plasma testosterone (T) increase after ejaculation. In some, the mere sight, sound, or odor of the female is sufficient to elicit an increase in T levels within minutes. This phenomenon has been observed in the rabbit (Endröczi, 1962; Endröczi & Lissák, 1962; Halmeyer & Eik-Nes, 1969; Saginor & Horton, 1968), rat (Kamel, Mock, Wright, & Frankel, 1975; Purvis & Haynes, 1974), mouse (Macrides, Bartke, & Dalterio, 1975), hamster (Macrides, Bartke, Fernandez, & D'Angelo, 1974), and bull (Katongole, Naftolin, & Short, 1971). Whether ejaculation affects the levels of luteinizing hormone (LH) in these species is less well documented. Katongole et al. observed increased LH levels in the bull after ejaculation, but Convey, Bentschneider, Hafs, and Oxender (1971) and Gombe, Hall, McEntee, Hansel, and Pickett (1973) did not. Taleisnik, Caligaris, and Astrada (1966) and Kamel et al. (1975) observed that ejaculation was followed by increased LH levels in the male rat, but Spies and Niswender (1971) did not.

Few studies on a relatively small number of primates have investigated the effects of sexual stimuli or ejaculation on T, LH, and cortisol levels. No change in T levels that could be attributed to ejaculation was found in male stump-tailed macaques (M. arctoides, n = 5; Goldfoot, Slob, Scheffler, Robinson, Weigand, & Cords, 1975). But after 2 wk of cohabitation with females, male rhesus monkeys (M. mulatta, n = 4) had higher T levels than when they were caged individually (Rose, Gordon, & Bernstein, 1972). However, the results of these two studies cannot be compared because of differences in procedure.

Masturbation did not affect the plasma levels of testosterone in men (n = 7; Fox, Ismail, Love, Kirkham, & Loraine, 1972), nor did coitus alter the mean serum levels of LH and T (n = 6; Stearns, Winter, & Faiman, 1973). Saginor and Horton (1968) reported (but did not publish the data) that 30 min after coitus the T levels of three
men did not differ from the baseline levels in the morning and evening. According to Fox et al. (1972), the T levels of one subject were the same just before and after coitus but were higher than under resting conditions. The plasma levels of LH and cortisol in human subjects were not altered by coitus (Fox et al., 1972; Ismail, Davidson, Loraine, & Fox, 1972). The mean levels of T in the urine of two men were higher during weeks of sexual activity than during no sexual activity (Ismail & Harkness, 1967). Reports of the effect of sexually exciting movies on T levels are contradictory: one study reported an increase in T levels (Pirke, Kockott, & Dittmar, 1974); another reported no increase in either LH or T level (Lincoln, 1974).

Plasma levels of T increased after ejaculation in all nonprimate species studied so far, but not in any species of primate. If additional studies were to show that primate and nonprimates differ in this respect, important consequences for future research into neuroendocrine-behavioral relations would result.

We undertook this study (a) to determine whether ejaculation affects T, LH, and cortisol levels in the rhesus monkey and (b) to learn more about how and to what extent behavior regulates the endocrine and neuroendocrine events in a species whose brain and behavior are more akin to those of man than to those of most nonprimate species.

Method

Subjects

In Experiment 1, 10 sexually experienced adult rhesus males, weighing between 7.5 and 16.0 kg, and 5 ovariectomized adult females were housed in individual cages in an air-conditioned room and exposed to outside light. During the previous 3 yr, all had been bled on numerous occasions and had been given tests of sexual behavior in the same test cage used in this experiment.

The same animals were used in Experiment 2 except that a male of comparable age, weight, and sexual experience was substituted for a male that had died during the year interval between the two experiments.

Ten different males of comparable age and weight were used in Experiment 3. They had served as sperm donors for 5 yr or more and had been electroejaculated about twice each week. They were housed in individual cages in an air-conditioned room, exposed to outside light, and electroejaculated in the same cage in which they lived.

Apparatus

The stainless steel test cage used in Experiments 1 and 2 measured 1.8 × .71 × 1.22 m and had a Plexiglas front and guillotine doors at each end, which served as entrances. The males in Experiment 3 were tested in their home cage. (The test room, which adjoined the living quarters, was soundproofed, air conditioned, and light controlled.)

Testing Procedure

Ten minutes after 3 ml of saphenous vein blood had been taken at Time 0, a male was placed in the test cage. During the control tests of behavior, no female was present in the cage in which the male remained for 10 min. During the experimental tests (male-female pair tests), an estrogen-primed female (10 mg of estradiol benzoate per day given im for 13 days) was present with the male during the 10-min test. When the test was over and the male had ejaculated, he was removed and 3 ml of blood were drawn from the saphenous vein. On one occasion during Experiment 2, a male was allowed to remain with a female for 13.3 min, at which time he ejaculated. In Experiment 1, blood was drawn 10, 30, and 50 min after the test; in Experiment 2, at 50, 80, and 140 min after the test.

All tests were given between 9:00 and 9:30 a.m. Experiment 1 was carried out in September, and Experiments 2 and 3 were carried out from July to September of the next year. We recorded the frequency of intromission, the number of pelvic thrusts per intromission, and the latency to ejaculation from the onset of the test.

In Experiment 3, a few minutes after the 3 ml of saphenous vein blood had been drawn at Time 0, electrodes were attached to the penis and ejaculation was elicited by electrical stimulation. During the control tests, electrodes were attached to the penis, but current was not applied and the animal did not ejaculate. Blood sampling procedures and the time intervals for drawing blood were the same as in Experiment 2: at 50, 80, and 140 min after the control tests or ejaculation. One week elapsed between tests; on each test day one animal was given a control test, and one was given an experiment test. Time of testing was the same as in Experiments 1 and 2.

Hormone Assay

Blood samples were centrifuged at 5,000 rpm for 20 min at 4 °C; the plasma was then removed and stored at —20 °C until assayed. In all three experiments the T levels were quantified by radioimmunoassay according to the procedure described by Resko, Malley, Begley, and Hess (1973). In Experi-
Experiment 1, serum cortisol levels were also quantified by radioimmunoassay. Antiserum obtained from a rabbit immunized with cortisol-21-succinyl bovine serum albumin were diluted 1:2500 with phosphate-buffered saline, pH 7.0, which contained 1% gelatine. The radioimmunoassay was performed exactly as for testosterone except that [\textsuperscript{3}H]cortisol (95 Ci/mM, New England Nuclear Corp.) was used as the isotope. Eight steroids (cortisol, corticosterone, desoxycorticosterone, testosterone, androstenedione, progesterone, 17\alpha OH-progesterone, and estradiol) found in the plasma of the male rhesus monkeys were checked for cross-reactivity with the antiserum. The percentage of cross-reactivity of 1,000 pg of corticosterone, desoxycorticosterone, progesterone, and 17\alpha OH-progesterone was 21.5%, 15.6%, 19.18%, and 39.19%, respectively. The same amount of cortisol displaced 72.4% of the radioactivity bound to the antibody, but the remainder of the steroids checked had little cross-reactivity. To separate those steroids that interfered with the measurement of cortisol, we chromatographed all samples on Sephadex LH-20 columns in the solvent system hexane:benzene:methanol (62:20:13). Cortisol eluted from the column in the fraction 12-21 ml of eluent. The dimensions of the columns have already been reported (Resko, Ploem, & Stadelman, 1975). Procedural losses averaged approximately 20% throughout.

In Experiment 2, plasma levels of LH were quantified by radioimmunoassay according to the procedure described by Resko, Norman, Niswender, and Spies (1974).

Statistical Analysis

At each time period, the plasma levels of hormone were compared with a single-factor analysis of variance for repeated measures. When significant treatment effects were observed, differences in treatment means were further analyzed with a Newman-Keuls test. All paired comparisons were evaluated by means of the Student’s paired \( t \) test, and the Spearman rank correlation, \( r_s \), was used as a measure of association. The .05 level of probability was set as the critical level of statistical significance. The same statistical analyses were used in all three experiments.

Table 1

<table>
<thead>
<tr>
<th>Hormone measured</th>
<th>Time 0</th>
<th>Minutes after test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Testosterone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No ejaculation</td>
<td>6.64 ± 1.23</td>
<td>5.90 ± 0.99</td>
</tr>
<tr>
<td>Ejaculation</td>
<td>9.42 ± 2.62</td>
<td>8.30 ± 2.12</td>
</tr>
<tr>
<td>Cortisol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No ejaculation(^a)</td>
<td>129.1 ± 11.76 (^b)</td>
<td>219.5 ± 17.89</td>
</tr>
<tr>
<td>Ejaculation(^a)</td>
<td>166.1 ± 4.97 (^b)</td>
<td>240.3 ± 17.18</td>
</tr>
</tbody>
</table>

\( ^a \) \( F(3, 27) = 43.14, p < .0001 \)

\( ^b \) Differs significantly from 0, 10, and 30 min (\( p < .01 \), Newman-Keuls).

\( ^c \) Differs significantly from 0, 10, and 30 min (\( p < .01 \), Newman-Keuls).

\( ^d \) \( F(3, 27) = 20.50, p < .0001 \).

Results

**Experiment 1**

Ejaculation did not produce an increase in the mean levels of plasma testosterone, \( F(3, 27) = .85, p = .473 \), nor did these levels increase when the males were placed alone in the test cage for 10 min, \( F(3, 27) = 1.37, p = .271 \) (Table 1).

Because the highest levels of T were reached at different times after ejaculation in different animals, we compared the plasma levels at Time 0 with the highest level attained regardless of interval. The difference between T levels at Time 0 and the highest point was not statistically significant \( t(9) = .457, p > .05 \). A similar analysis revealed no difference in plasma levels of T in the control series, \( t(9) = 1.721, p > .05 \).

Mean plasma levels of T at each time period during the control series were compared with those at comparable periods during the experimental series. None of the paired \( t \) tests proved to be significant (\( p > .05 \)).

The rank-order correlation of T levels between control and experimental tests at Time 0 was .73 (\( p < .02 \)); at 10, 30, and 50 min they were .64, .69, and .78, respectively. In each case, the probability was less than .05.

The mean T level for each male after ejaculation (Times 10, 30, and 50) was compared with that before ejaculation (Time 0). In four of the males, these levels were higher; in the other six, they were lower. In the control series the mean levels of T...
were higher in five animals and lower in the rest.

In all three experiments testosterone levels did not differ significantly whether the first test was experimental or control \( (p > .05) \).

Cortisol levels increased significantly in both the control and the experimental tests, \( F(3, 27 = 43.14, p < .0001 \) and \( F(3, 27) = 20.50, p < .0001 \), respectively. According to a Newman-Keuls analysis, the level of cortisol at Time 0 was significantly lower than at 10, 30 and 50 min after ejaculation \( (p < .01) \). Moreover, the 10 and 30 min values did not differ from each other, but both were significantly lower than at 50 min \( (p < .01) \). The same pattern of differences and the same levels of statistical significance held for the control series (Table 1).

The percentage of increase in plasma cortisol between the level at Time 0 and the highest level reached regardless of time period was computed for each male in both the experimental and the control series, and the percentage of increase in the two series was compared. A mean percentage of increase of 134.7 ng/ml for the control series was not significantly greater than a mean increase of 90.2 ng/ml in the experimental series, \( t(9) = 1.966, p > .05 \).

Selected items of sexual behavior and levels of T at Time 0 for individual males are given in Table 2. Comparison of T levels and performance in the two experiments are analyzed in Experiment 2, Results. No significant rank correlations between intromission rate, thrusts per intromission, or ejaculation latency and T levels were found at Time 0. Of the five males with ejaculation latencies below the median group latency \( (3 \text{ min}) \), three showed increases in T after ejaculation and two showed decreases. The males with latencies above the group median showed a similar distribution. Three had increased levels, and two had decreased levels of T. Latency to ejaculation was not related to the direction of change in T levels after ejaculation. Using the same kind of analysis, we found that the intromission rate did not predict whether the mean T levels over the three time periods would increase or decrease after ejaculation.

Experiment 2

Because Experiments 1 and 2 were carried out 1 yr apart and because Experiment 1 was carried out in September and Experiment 2 extended from July to September, data from the two could not reasonably be presented as a single time continuum \( (i.e., 10, 30, \text{ and } 50 \text{ min of Experiment 1, and } 50, 80, \text{ and } 140 \text{ min of Experiment 2}) \) and are therefore presented separately.

Ejaculation did not result in a significant increase in plasma levels of testosterone, \( F(3, 27) = 1.50, p > .10 \). An analysis of variance applied to data from the control series also showed no significant changes.

<table>
<thead>
<tr>
<th>Male</th>
<th>Testosterone (in ng/ml) at Time 0</th>
<th>Intromission (rate/min)</th>
<th>Thrusts per intromission</th>
<th>Ejaculation latency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>E 1</td>
<td>E 2</td>
<td>E 1</td>
<td>E 2</td>
</tr>
<tr>
<td>5017</td>
<td>2.94</td>
<td>11.78</td>
<td>1.0</td>
<td>1.2</td>
</tr>
<tr>
<td>1501</td>
<td>3.75</td>
<td>3.23</td>
<td>3.0</td>
<td>2.7</td>
</tr>
<tr>
<td>585</td>
<td>3.80</td>
<td>2.05</td>
<td>.3</td>
<td>.9</td>
</tr>
<tr>
<td>3527</td>
<td>4.34</td>
<td>7.03</td>
<td>1.8</td>
<td>1.5</td>
</tr>
<tr>
<td>4486</td>
<td>5.94</td>
<td>3.88</td>
<td>.8</td>
<td>1.5</td>
</tr>
<tr>
<td>1069</td>
<td>11.47</td>
<td>.56</td>
<td>.6</td>
<td>4.0</td>
</tr>
<tr>
<td>6221</td>
<td>15.38</td>
<td>.75</td>
<td>.4</td>
<td>.5</td>
</tr>
<tr>
<td>1617</td>
<td>17.26</td>
<td>8.66</td>
<td>2.0</td>
<td>.8</td>
</tr>
<tr>
<td>3533</td>
<td>27.27</td>
<td>6.47</td>
<td>.8</td>
<td>2.0</td>
</tr>
<tr>
<td>1068</td>
<td>2.05</td>
<td>.</td>
<td>.2</td>
<td>.</td>
</tr>
<tr>
<td>1067</td>
<td>.</td>
<td>2.25</td>
<td>.</td>
<td>.4</td>
</tr>
</tbody>
</table>

Table 2

Plasma Levels of Testosterone at Time 0 and Performance on Selected Items of Sexual Behavior for Individual Males in Experiments (E) 1 and 2

Note. Only nine animals served in both experiments, which were carried out 1 yr apart.
in T levels, $F(3, 27) = .31, p > .10$ (Table 3).

For each male, the plasma level of T before ejaculation was compared with the highest level after ejaculation. The differences evaluated by means of the paired $t$ test were not significant, $t(9) = 1.802, p > .05$. A similar analysis revealed no differences in plasma levels in the control series, $t(9) = 1.233, p > .05$.

Paired $t$ tests indicated that T levels at comparable intervals did not differ in the control and experimental series. In each case, probabilities were greater than .05. The rank-order correlation ($r_s$) of the males’ T levels between control and experimental series at Times 0, 50, 80, and 140 min were .89, .93, .76, and .79, respectively. Associated probabilities were all less than .05.

During neither the experimental nor the control series was there a significant change in plasma LH levels, $F(3, 27) = 1.91, p > .05$ and $F(3, 27) = 2.00, p > .10$, respectively (Table 3). The mean plasma LH level at Time 0 before the ejaculatory test was significantly higher than at Time 0 before the control test, $t(9) = 3.441, p < .01$, but why, we do not know. At other corresponding times (50, 80, and 140 min, experimental vs. control tests), the mean levels of LH did not differ ($p > .05$).

Intromission rate, number of thrusts per intromission, ejaculation latency, and T level at Time 0 for individual animals are shown in Table 2. Rank-order correlations indicated that performance levels were not associated with plasma levels of T ($p > .05$ in all cases). Plasma T levels at Time 0 were compared with those during the same time period a year earlier for the nine males used in both Experiments 1 and 2. Mean T levels did not differ significantly, $t(8) = 1.726, p > .01$, nor did mean intromission rate, thrusts per intromission, and ejaculation latency change from their respective levels a year earlier, $t(8) = 1.309, 1.353, and 1.085$, respectively, $p > .10$.

### Experiment 3

Electroejaculation did not produce statistically significant increases in T levels, $F(3, 27) = 1.76, p > .10$, but an overall significant difference in T levels was observed in control tests, $F(3, 27) = 4.78, p < .01$ (Table 4). A Newman-Keuls analysis of differences indicated that the mean plasma level of T at Time 0 was significantly higher than at 80 and 140 min ($p < .05$ and $< .01$, respectively). Other differences were not statistically significant.

When the values obtained at Time 0 were compared with the highest level attained after Time 0 for both the control and experimental series, the differences were not significant, $t(9) = 1.304, p > .05$ (regardless of the interval when the highest level was recorded), and the highest levels in the control series were not significantly different from the Time 0 levels, $t(9) = .908, p > .05$.

Testosterone levels at each time period during the control series (0, 50, 80, and 140 min) did not differ from those at the comparable time periods during the experimental series. Paired $t$ tests yielded values

### Table 3

<table>
<thead>
<tr>
<th>Hormone measured and treatment</th>
<th>Time 0</th>
<th>50</th>
<th>80</th>
<th>140</th>
</tr>
</thead>
<tbody>
<tr>
<td>Testosterone</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No ejaculation</td>
<td>4.54 ± 1.41</td>
<td>5.11 ± 1.81</td>
<td>4.02 ± 1.07</td>
<td>4.70 ± 1.34</td>
</tr>
<tr>
<td>Ejaculation</td>
<td>4.67 ± 1.17</td>
<td>6.88 ± 1.80</td>
<td>5.77 ± 1.39</td>
<td>5.03 ± 1.65</td>
</tr>
<tr>
<td>LH</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No ejaculation</td>
<td>.65 ± .05*</td>
<td>.70 ± .07</td>
<td>.78 ± .07</td>
<td>.74 ± .05</td>
</tr>
<tr>
<td>Ejaculation</td>
<td>.92 ± .11</td>
<td>.80 ± .10</td>
<td>.90 ± .11</td>
<td>.78 ± .14</td>
</tr>
</tbody>
</table>

* Differed from LH value at Time 0 in ejaculation series, $t(9) = 3.441, p < .01$.

\[ \text{Note. No significant differences in testosterone or LH occurred across time.} \]
Table 4
Mean (± SE) Concentration of Testosterone (in ng/ml) in Systemic Plasma of 10 Rhesus Males Before and After Control Tests or Electroejaculation in Experiment 3

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Time 0</th>
<th>50</th>
<th>80</th>
<th>140</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control testsa</td>
<td>6.05 ± 1.16a</td>
<td>5.14 ± 1.24</td>
<td>4.47 ± 1.20</td>
<td>4.49 ± 1.09</td>
</tr>
<tr>
<td>Ejaculation</td>
<td>6.29 ± 1.24</td>
<td>7.00 ± 1.35</td>
<td>6.43 ± 1.43</td>
<td>4.64 ± 1.22</td>
</tr>
</tbody>
</table>

* F(3, 27) = 4.78, p < .01, single-factor analysis of variance for repeated measures.

a Differed significantly from control tests, Time 80, and, Time 140, Newman-Keuls, p < .05.

with probabilities greater than .05 for each interval.

Discussion

We found no statistically significant increase in plasma T levels after ejaculation regardless of whether ejaculation occurred during copulation with a female or as a result of electrical stimulation of the penis. These results agree with those of experiments in man that compared plasma levels of T before and after coitus and masturbation. They differ from the results of experiments on nonprimate species.

Testosterone elevations after acute exposure and ejaculation with a female should be distinguished from those after chronic exposure to receptive females with or without accompanying ejaculation. Rose et al. (1972) showed that T was higher in male rhesus monkeys when they lived with females for 2 wk than when they were caged alone for the same length of time. Although T was not measured directly, Vandenbergh (1969) reported that the color of the sex skin, the size of the testicles, and the diameter of the seminiferous tubule increased during the non-breeding season in male rhesus monkeys exposed for an extended period to female rhesus injected with estradiol. These findings do not contradict our observation that ejaculation within a 10-min period to female rhesus injected with estradiol. These findings do not contradict our observation that ejaculation within a 10-min period of exposure to a female did not produce increased plasma levels of T for at least 140 min after ejaculation.

In a 3-hr test, testosterone levels were lower in four adult stumptail monkeys after multiple ejaculations than they were before the test (Goldfoot et al., 1975). However, after a 3-hr period without intervening ejaculation, these levels also declined.

The authors suggested that the effect of copulation was not strong enough to overcome the lowered values associated with the stress of handling. During the 3-hr test, the males ejaculated between 11 and 19 times. When rats copulated to sexual satiety, their plasma T levels declined (Bliss, Frischat, & Samuels, 1972), whereas other studies have shown that fewer ejaculations resulted in increased T levels. Even if a shorter test period and fewer ejaculations by the stumptail males were to result in no change in T, the decline after the control tests would remain unexplained. In our control test (Experiment 3: males not electroejaculated), the mean T levels at 80 and 140 min, but not at 50 min, were significantly lower than at Time 0; why, we do not know. In the control tests in Experiment 1 at 10, 30 and 50 min, and in Experiment 2 at 50, 80, and 140 min, the T levels did not differ from the levels at Time 0.

Plasma levels of LH did not increase after ejaculation either in our males or in man (Fox et al., 1972; Stearns et al., 1973). Because the levels of T did not increase, the failure of LH to do so was not unexpected. Whether LH levels increase after ejaculation in nonprimates remains controversial.

Plasma levels of cortisol increased significantly after both the control and the experimental tests, but T levels did not change significantly. If increases in cortisol indicate stress, then we must conclude that under conditions of acute stress, at least, T levels may remain unchanged. This is not to argue that more severe or chronic stress would not result in reduced testosterone levels. Bliss et al. (1972) have shown that rats forced to swim or shocked intermittently for 1 hr had decreased lev-
els of T, and chronic stress in rhesus monkeys and man also resulted in lower plasma T levels (Kreuz, Rose, & Jennings, 1972; Rose, Bernstein, & Gordon, 1975; Rose et al., 1972).

The increased T observed after ejaculation in some species may be adrenal in origin, although it seems unlikely (Endröcci & Lissák, 1962). Male rabbits injected iv with ACTH did not experience an increase in circulating T (Haltmeyer & Eik-Nes, 1969), nor did adult male rhesus monkeys injected with ACTH (Michael, Setchell, & Plant, 1974).

Experiment 1 was carried out within the month of September, but Experiments 2 and 3 extended from July to September and hence involved different seasons of the year. If T levels varied seasonally in laboratory-housed male rhesus monkeys, the failure to observe a difference in T levels after ejaculation might be due to the time of year when they were tested. A recent report suggested that laboratory-housed rhesus males show seasonal variation in T levels (Robinson, Scheffler, Eisele, & Goy, 1975), but Resko (1967) found no reliable seasonal changes in T levels in males housed at the Oregon Primate Center. Under the same environmental conditions, similar data were obtained for T levels in rhesus monkeys bled in winter months and in the spring (Phoenix, in press).

If, in fact, ejaculation elicited an increase in T in our rhesus monkeys, the increase must have been subtle and of such short duration as to be missed by our bleeding regimen. In any event, increases in T levels after ejaculation are not the striking phenomenon in rhesus monkeys that they are reported to be in rabbits and various rodent species.

References


Phoenix, C. H. Factors influencing sexual perform-
ance in male rhesus monkeys. *Journal of Comparative and Physiological Psychology*, in press.


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