onset of the tone coincided with the turning off of the signal light. The subjects were urged to predict to the best of their ability and to attempt to improve their prediction with practice. The small lamps lighted up according to a prearranged schedule. For half the subjects, the left lamp lit up seven times in each block of 10 trials, the right lamp three times. In the other half, these frequencies were reversed.

The acquisition phase involved 50 trials, the last of which was not reinforced. Thirty extinction trials were then given. The CS, a 500–cy/sec tone, had a duration of 2550 msec. The CS-UCS interval during conditioning was 500 msec and the UCS was a 1.0 lb/in.^{2} air puff, 50 msec in duration. Each group contained 25 subjects from an introductory psychology course.

Figure 1 presents the extinction curves for the three groups. Clearly evident are the markedly different rates with which the decrement in conditioned responses occurs during the extinction trials. Group 1, extinguished with no UCS, dropped precipitously to a random level of responding (10 percent) after two nonreinforced trials. The curve of group 2, which was extinguished with a 2500-msec CS-UCS interval, also dropped quite rapidly, but somewhat slower than that of group 1. In terms of the number of conditioned responses in the first ten extinction trials the difference between these two groups was significant at the .01 level. This finding differs, it will be noted, from that of earlier studies (*2*) which obtained no difference between groups extinguished with no UCS and with an extended CS-UCS interval after continuous reinforcement. The only difference in the experimental conditions is that in the present experiment the CS duration was 2550 msec during acquisition, whereas in the previous studies it was increased from 550 msec to 2550 msec in the case of the group extinguished with the extended CS-UCS interval.

In sharp contrast to the findings for groups 1 and 2, the extinction curve of group 3, which was conditioned and extinguished in the context of the probability-learning task, exhibited a very gradual decrease. Thus, by the ninth and tenth extinction trials, this group's performance level had decreased only 14.4 percent from its final acquisition level, whereas the corresponding decrements for groups 1 and 2 were 56 percent and 45.2 percent, respectively. The differences between the performance levels of group 3 and the other two groups during the first ten extinction trials were highly significant, at the .01 level in the case of group 2 and the .005 level for group 1.

After the experiment the subjects were questioned as to whether they noticed any change in the experimental procedure during the course of the experiment. If the subject replied "yes," further questions were asked to ascertain if the change in the CS-UCS interval or the cessation of the UCS in the case of group 1 was noted. The results of these questions indicated considerable differences among the groups. Twenty-one subjects of group 1, 19 of group 2, but only one of group 3, reported noticing the procedural change. Correlated with this absence of recognition on the part of group-3 subjects is the strikingly slower extinction. The slight difference between groups 1 and 2 in the number of subjects that recognized a difference in procedure with extinction indicates that some other factor than difference in proportion of subjects that observed the change must be appealed to in order to account for the difference in mean rate of extinction of these two groups. A possible interpretation is that the lesser change in the case of group 2 than group 1 required, on the average, more extinction trials before the difference in procedure was recognized.

It is apparent from these data that extinction of the conditioned eyelid response in humans is to a considerable degree a function of cognitive factors relating to observation on the part of the subject of procedural changes with the shift to extinction, a finding that suggests support for some version of the discrimination hypothesis. Attempts to infer the quantitative properties of an intervening theoretical variable, for example inhibition, that results from the operation of nonreinforcement, will need to take account of these potent cognitive factors (*4*).

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Testosterone-Induced Incubation Patches of Phalarope Birds

Abstract. *In order to determine the endocrine factors in the production of the incubation patch of phalarope birds* (Steganopus tricolor *and* Lobipes lobatus), *adults of both sexes have been maintained in captivity and injected with estradiol, testosterone, or prolactin, or with different combinations of these hormones. Only testosterone and prolactin in combination produced incubation patches in all birds, both male and female, in the two species.*

The incubation patch is a naked vascular area on the ventral surface of one or both sexes of many avian species. It is found during part of the breeding season. It transfers heat from bird to egg during incubation and is considered analogous in certain functional aspects to the primitive mammary gland (*1*). Furthermore, the hormones required for the development of the incubation patch are said to be "strikingly similar" to those required for the development of the mammary gland, except that the latter requires progesterone (*2*). This conclusion is based chiefly on the work of Bailey (*3*), who showed that estrogen and prolactin are essential for the development of the incubation patch in passerine birds. He suggested that in those species in which only males possess the incubation patch (tinamous, phalaropes, button quail, jacanas) androgen may have the same relation to the patch as estrogen does in the passeriform birds.

To test this hypothesis, we experimented with Wilson's phalarope, *Steganopus tricolor*, and the northern phalarope, *Lobipes lobatus*. In these species the female develops bright nuptial plumage and displays aggressive courtship behavior. Only the male, whose nuptial plumage is a drab counterpart of that of the female, has the incubation patch. Furthermore, only the male builds the nest, incubates the eggs, and broods the young (*4*).

Our observations of wild birds in-

dicate that males have no incubation patch when they first arrive on the breeding grounds in May, although the testes are greatly enlarged and spermatogenesis is in process. The development of incubation patches in these birds occurs during a few days in early June and appears to be concurrent with nest building.

Sixty-one Wilson's phalaropes in nuptial plumage were captured in mist nets in northwestern Montana during May and June of 1961 and 1962. Six northern phalaropes in winter plumage were similarly captured during October 1962. Eleven male and 17 female Wilson's phalaropes and three male and three female northern phalaropes were maintained in the laboratory for experiment for periods ranging from 1 to 5 months. Some of the remaining captive birds served as controls, and others were killed for anatomical studies or died before completion of the experiments.

Because our captive birds were exposed to continuous artificial light, and because the production of gonadotrophic hormone resulting from increased photoperiod may be suppressed by treatment with prolactin (*5*), several male and female Wilson's phalaropes were given daily injections of prolactin beginning immediately after capture and continuing for at least 10 days. These birds were then killed. Their gonads had regressed to a condition anatomically similar to that observed in control birds killed in the fall. Therefore, all 34 experimental birds were injected with prolactin for 10 days before further treatment, and their gonads were then considered to be in the same phase of the annual cycle although the experiments were conducted during the summer and fall.

After preliminary injection of prolactin, experimental birds were divided into six groups with a minimum of four birds per group. Each group included both sexes, and injections were continued for a minimum of 7 days. The first group received continued injections of prolactin, the second was injected with estradiol, the third with testosterone, the fourth with prolactin and testosterone, the fifth with prolactin and estradiol, and the sixth with estradiol and testosterone.

These hormones were administered intramuscularly in the following daily doses: 20 international units (I.U.) of prolactin (*6*) in 0.1 ml of pyrogen-free water, 100 I.U. of estradiol benzoate in 0.04 ml of sesame oil, and 1 mg of testosterone propionate in 0.04 ml of sesame oil. These amounts were not varied whether given alone or in combination.

According to Bailey (*3*), during the development of the true incubation patch the ventral surface must undergo defeatherization, increased vascularity, and edema. By this standard all individuals of the group (four females and two males) receiving prolactin and testosterone developed incubation patches within 6 days after the beginning of treatment. No observable changes could be detected in any other group. Development of an incubation patch was confirmed by gross inspection of all living birds and by microscopic examination of biopsies from the ventral surface of some control and experimental birds. Repluming of the bare area, which is characteristic of the recovery stage (*3*), occurred several weeks after cessation of treatment.

Because most of the male phalaropes were captured in the spring, they already had incubation patches or developed them spontaneously soon after capture. Therefore, incubation patches were experimentally induced in male northern phalaropes which were in winter plumage and in male Wilson's phalaropes whose incubation patches had replumed in captivity. Control females did not develop incubation patches.

These experiments show that in phalaropes, androgen plays the same role in the formation of the incubation patch that estrogen plays in males of some passerine species, and they also suggest that a variety of modes of hormonal control of the incubation patch will be found among different avian species (*7*).

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Alcohol and Caffeine: Effect on Inferred Visual Dreaming

Abstract. *In seven subjects, a large amount of alcohol, taken at bedtime, reduced the total duration of stage 1 electroencephalographic–rapid-eye-movement periods in the first 5 hours of sleep; a moderate amount of caffeine produced no significant change.*

A particular low-voltage and high-frequency electroencephalographic (EEG) pattern, usually accompanied by rapid eye movements (*1, 2*), has been associated with dreaming, predominantly in the visual modality (*3, 4*). Deprivation of 65 to 75 percent of this dreaming, as identified by EEG and rapid-eye-movement correlates for five nights, seemed to produce anxiety, increased irritability, memory lapses, and an increased appetite (*5*). There have been suggestions that hypnotics and alcohol reduce the usual 1 to 3 hours of such dream time achieved nightly by the normal subject (*4*).

As noted above, visual dreaming appears to be identified by an EEG pattern of low-voltage fast activity and rapid eye movements in the electro-oculogram. This low-voltage fast activity is identical to that which has been previously associated with light sleep, and has been called "emergent stage 1" because it appears after so-called deeper sleep patterns.

It has been shown that depressants of the central nervous system decrease the frequency and increase the amplitude of the EEG waves (*6*), whereas stimulants of the central nervous system increase the frequency and decrease the amplitude of the EEG waves (*7*). It would seem to follow, then, that sedatives would increase the depth of sleep and, in turn, result in diminished light sleep, and thereby allow less EEG stage 1–rapid-eye-movement (REM) time and less dreaming. On the other hand, stimulants would tend to decrease the depth of sleep and provide at least part of the setting