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

in which EEG stage 1–REM time and dreaming usually occur. It was hypothesized that alcohol would decrease, and caffeine increase, EEG stage 1–REM time and inferred dreaming.

Seven subjects who were second-year medical students, ages 23 to 28 years, after an initial nonrecorded base-line night, were continuously monitored by EEG and electrooculographic recording on two base-line nights, one drug night, another base-line night, and a second drug night, in that order, with four nights of extra-laboratory sleep interposed between the first drug night and the fourth base-line night. The subjects reported to the laboratory approximately 1 hour before their usual time of retiring. For all subjects this was between 10:00 and 10:30 P.M. After an approximate 30-minute period required for electrode placement, 1 g/kg of body weight of 95-percent ethyl alcohol (the equivalent of 6 oz of 100-proof liquor for a 150-lb man) or 0.005 g/kg of body weight caffeine citrate (between 2.5 and 3.0 cups of coffee for a 150-lb man) mixed in 400 ml of orange juice, was drunk by each subject in approximately 30 minutes. For four of the subjects alcohol and orange juice was the test mixture drunk on the first experimental night and caffeine and orange juice on the second; for three, caffeine and orange juice on the first and alcohol and orange juice on the second. On base-line nights only orange juice was drunk. Immediately after having drunk 400 ml of the test mixture, the subjects retired to a sound-attentuated, temperature-controlled, darkened room, and were not disturbed until their normal waking time.

By the use of Dement's "Manual for Scoring REM Periods" (*8*) with minor modifications, an interscorer reliability of 99 percent was obtained. We scored rapid-eye-movement time during the first 300 minutes of total sleep time, but excluded the initial sleep stage 0 and any short "no record" periods resulting from subject urinating, vomiting, or technical difficulties, and during the balance of sleep time after 300 minutes. This arbitrary selection of the amount of record to be scored made all records comparable to the shortest. The method of determining sleep stages was that described by Dement and Kleitman (*1*).

The transition into the EEG stage 1–REM period is characterized by an activation of the EEG from spindles plus random slow waves to 4- to 7-cy/sec waves which in turn give way to 8- to 12-cy/sec waves which quickly drop in amplitude. This flattening ending with a few alpha frequency waves marks the onset of a low-voltage fast record which is punctuated by rapid eye movements, low-voltage 3- to 6-cy/sec waves [described by Berger *et al.* (*9*) as saw-toothed because they tend to have sharp peaks] and a few alpha frequency waves. We modified Dement's scoring procedure to begin the EEG stage 1–REM period at this easily observed abrupt flattening of the EEG, rather than rely on set time intervals after a body movement.

Further, the continuity of an EEG stage 1–REM period is disturbed occasionally by what appears to be suppressed spindle activity, especially in the first and second EEG stage 1–REM periods in the first half of the night. This activity lacks the amplitude and configuration to be called "true" spindle activity and is not usually visible in later EEG stage 1–REM periods. This suppressed spindle activity gives us the impression that these earlier EEG stage 1–REM periods appear during slightly deeper sleep than later ones. These same waves are sometimes seen as the last half of a "true" spindle burst occurring concomitantly with the onset of the EEG stage 1–REM burst. We scored this activity as spindling only when it obtained an appreciable amplitude (approximately half of that subject's average spindle amplitude); otherwise, they were ignored as indicative of sleep stage 2.

The analyses of records for the five recorded nights are shown in Table 1. An analysis of variance of the three recorded base-line nights for the seven subjects result in an insignificant F associated with nights ($.05 < P < .10$) and a significant F for subjects ($.005 < P < .01$). The means of the base-line nights for the subjects were then compared by t-tests for correlated means with the values of alcohol and caffeine nights. The influence of alcohol was significant ($.01 < P < .025$); the caffeine effect was not significant ($.10 < P < .20$). Therefore, from these results it appears reasonable to conclude that a large dose of alcohol reduces the amount of EEG stage 1–REM time and inferred dreaming in normal subjects and that a moderate dose of caffeine has no significant effect on these parameters of sleep.

Table 1. Minutes of EEG stage 1–REM periods.

Subject	Base-line nights			Mean base-line night	Alcohol night	Caffeine night
	Second	Third	Fourth			
A	60	60	93	71	28	80
B	34	42	39	38	40	60
C	32	56	49	46	28	36
D	*	105	107	106	49	70
E	46	81	132	86	69	119
F	76	71	72	73	65	84
G	72	47	64	61	43	91

* A "no-record period" longer than 20 minutes; data not used.

It appears possible that the negative results obtained from the caffeine may be attributed to the amount or type of drug, that is, the dosage may not have been clinically sufficient or may have lacked the sustained stimulation during sleep that might have been obtained from sustained-release capsules of amphetamine, for example (*10*).

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