A COMPARATIVE EEG STUDY OF SLEEP

A COMPARATIVE ELECTROENCEPHALOGRAPHIC STUDY OF SLEEP

By

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This thesis is concerned with the variations which occur in the electroencephalograms, eyemovements and neckmuscle potentials of three species, the pigeon, rat and chicken during prolonged recording under normal conditions, under conditions in which the animals were fatigued, and after drugs had been administered. While the recordings from the rat showed the two stages of deep sleep typical of mammals, no distinctive "sleep" patterns were observed in the records from birds, except after nembutal had been given. The results seem to support the idea that birds do not sleep.

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CHAPTER ONE

INTRODUCTION

In a sense, sleep is everyone's problem. As Kleitman has stated, "sleep is a subject on which almost every individual considers himself competent to write because of personal interest and first hand experience." (1939, p. 6) Yet, despite this truly voluminous literature on the subject, the mechanisms of sleep still remain a mystery to us.

Early theories of sleep centred on the notion that somehow fatigue substances accumulate in the organism during wakefulness, and the action of these substances on the nervous system causes sleep. Although these ideas are over one hundred years old we are still compelled to accept some variation of them today. Modern electrophysiology has suggested that the cite of action of these substances may be in the hypothalamus or so-called activating system. However, why sleep is necessary at all is still a puzzle.

Nauta has emphasized the difficulties that are a result of our lack of knowledge:

"The essential role of the nervous system in the regulation of the sleep-waking rhythm of higher animals is now widely recognized. No less a person than von Economo, however, drew attention to the fact that the phenomenon of sleep cannot be accounted for by a mere functional change of the central

nervous system from its condition during the waking state. It is indeed an important fact that the function of sleep, instead of being characteristic of higher animals, is also observed in several organisms which do not possess a central nervous system, and even in several vegetable species. It is therefore impossible to attribute this mysterious function to any special organ. Since all experimental work on sleep has hitherto been confined to mammals, chiefly to cats and monkeys, practically no data concerning the comparative physiology of this phenomenon are available." (1946, P. 285)

It is to this latter statement of Nauta's that the research to be reported in this thesis is directed. While, since 1946, several comparative studies have appeared, none have employed submammalian species as subjects. This fact is especially remarkable, since, as we shall see, considerable emphasis has been placed on the role of mammalian cortex by several modern sleep theorists. If, as these authors seem to suggest, different stages of sleep are a function of the activity of the neocortex, what would one expect from an organism lacking such a structure?

The present research represents an exploratory attempt to answer some of these questions. To achieve this, electroencephalograms were recorded from different species over long periods of time and under several different experimental conditions. Specifically, we were interested in whether the typical "sleep-type" EEG patterns which appear in mammals, occur naturally or could be experimentally produced in avian species. The study is incomplete since no behavioral observations were made. However, it was felt that finding out the extent of variations in the EEG records of birds would be of sufficient theoretical

interest to warrant such a design.

CHAPTER TWO

HISTORY

Ancient Theories

The oldest theories of sleep centre around the circulation and distribution of the blood. Alcmeon, who lived in the sixth century B.C., is credited with the first theory of sleep (Kleitman, 1939). In this time when the importance of the brain was unknown, Alcmeon hypothesized that sleep was a result of the retreat of blood into the veins. Awakening was thus due to venous disgorgement. Aristotle, who also emphasized the heart as the centre of "nervous" functions, saw sleep as a result of the digestive process. During digestion, evaporation occurred, and the heat from this process rose through the body causing drowsiness and sleep. Fatigue acted as a solvent in the body, therefore yielding the same result. Aristotle also suggested that the proportionately longer time spent in sleep in infants was a result of infants having larger heads compared with their body size.

These ancient theories are essentially limited in nature, since more importance is attached to the mechanism of sleep than to the problem of why sleep occurs. However, they are the forerunners of the 17th and 18th century "cerebral

anemia" theories. With a better understanding of both the function of the brain and of circulation, numerous authors proposed that sleep resulted from either a change in the blood composition or a change in the blood flow to the brain. With the improved techniques of the last 50 years or so, most of these early notions have been experimentally tested and the results have either failed to show the appropriate relationship or have shown the exact opposite. Thus, while these early theories showed considerable imagination on the part of the authors, few have been borne out by modern facts.

With the many gains made in the latter half of the 19th century in the understanding of both physiology and neurology, numerous new theories of sleep were proposed. Kleitman (1939) has classified these into two groups- humoral and neural. We shall therefore follow Kleitman's example.

Humoral Theories

Kleitman ascribes the following characteristics to theories in this group: "The common feature that characterizes the humoral theories of sleep is the production and accumulation of certain substances, usually end products of metabolism, either in the tissues, in general, or in certain organs, such as the brain." (p. 479) A number of these theories emphasize the importance of the oxygen balance in the brain. One factor leading to the attenuation of the oxygen concentration included

accumulation of lactic acid. Conversely, the accumulation of carbon dioxide and its adverse effect on the brain (autonarcosis) has been noted.

Several theories have postulated the existence of certain fatigue substances (toxins). In these theories, the fatigue toxin accumulates during the waking hours and causes sleep. During sleep the fatigue toxin dissipates or is counteracted by an anti-toxin.

Similar to the toxin theories are those theories which emphasize endocrine function. Here, hormones are released into the blood and consequently affect certain "sleep centres." Typical of the hormone theories is Salmon's (Kleitman, 1939). Salmon proposed that the anterior lobe of the hypophysis produced an antitoxic hormone that prevented the sleep toxins from operating during wakefulness. The posterior lobe produced a vasoconstrictor hormone which inhibited the activity of the sleep centres and lead to sleep. The support for Salmon's theory came from three sources, as Kleitman (1939, p. 485) has pointed out: "(1) the production of sleep by the injection of posterior-lobe extracts into the brain ventricles; (2) the favorable effects of these extracts in cases of hypophyseal syndromes with insomnia; and (3) the increase in sleepiness in hypertrophy and hyperemia of the hypophysis (pregnancy, incipient acromegaly, castration, hypothyroidism, and infections

such as influenza and encephalitis)." While Kleitman concedes that Salmon's sleep centre may be located in the diencephalon he argues that this cannot possibly be the dominant control of the sleep-wakefulness rhythm. The burden of proof for Kleitman lies in the "well-known fact" that lesions in the hypophysis do not result in continuous wakefulness or sleep.

Kleitman lists five reasons why both the toxin and hormone theories are unacceptable: (1) They cannot explain why the same concentration of the "sleep-producing substance" in a single individual can at one time result in sleep but at another - wakefulness. This might depend entirely on the circumstances. In other words sleep, at least in adult humans, is a voluntary act. (2) Babies, despite a minimum amount of activity, sleep most of the time. (3) Parabiotic monsters, babies with two heads and a single body and circulatory system, have been observed when one of the heads was sleeping and the other nursing. This observation has also been made on several sets of Siamese twins. (4) Cross-blood circulation studies on dogs also showed that they sleep independently. (5) Kleitman's own observations on humans that have been kept awake for several days have shown that they do not get continuously sleepier, but rather follow a periodic cycle of sleepiness. His subjects were much less sleepy on the third day than they were on the second night. Kleitman also suggests that if the toxin-hormone theories were valid, one

would rise in the morning at the peak of efficiency, whereas in fact, this does not occur until several hours after rising.

The exponents of this group of theories could undoubtedly answer Kleitman's criticisms, some more easily than others. For example, Kleitman's observations that sleep is a voluntary act does not preclude the possibility that toxin substances exist. It may be that the fatigue elements predispose certain neural patterns and that sleep results from the integration of a number of factors, including neural activity involved in the "voluntary desire." The cyclic day-night pattern common to human adults may be well within the critical fatigue level which would result in "compulsory" sleep.

Thus, the fatigue substance theories are not a dead issue but are implicit in much of the present day theorizing. Failure to discover the appropriate substance does not prove the theory false. It might mean instead that the analytic technique used is inappropriate, or that the crucial substance is rapidly dissipated in the blood stream, after affecting a "sleep centre." However, despite these possibilities, scientific interest has generally shifted away from the hormone-toxin theories to the group to be described in the next section - neural theories.

Neural Theories

The earliest strictly neural theories of sleep appeared toward the end of the last century. These theories generally

pertained to some change which occurred within the individual neuron or in the dendrites of the neuron. Duval (Kleitman, 1939) originally proposed that during wakefulness, the dendrites of one cell in the cortex gradually moved away from the connecting cell body. As this ameboid movemement reached a certain limit the circuit was broken and sleep resulted. During sleep, the shrunken dendrites again became elongated and the broken contacts were reestablished causing the organism to awaken. A modified and somewhat more complicated ameboid movement theory was proposed by Cajal. However, since there is no evidence of histological changes during waking and sleep it does not seem profitable to go into detail.

A second group of neural theories which have proven infinitely more popular are those involving the concept of inhibition. While several inhibition-type theories have been proposed, none have reached the prominence of the one proposed by Pavlov (1927, Lectures XV & XVI). Pavlov's basic assertion is that "sleep and what we call internal inhibition are one and the same process." (p. 251) An example offered by Pavlov might demonstrate this relationship:

> "An animal has conditioned reflexes established to different stimuli, including one to a definite musical tone. During the whole period the animal remains alert. The development of a differentiation of a tone close to the positive one is now started, and it is noticed that during the process, the animal gets drowsy. The drowsiness gradually increases, and often culminates in a deep sleep with a complete relaxation of the skeletal muscles, and snoring, so that when no other positive conditioned stimuli

are administered and reinforced by food it is necessary to stir up the animal and even to introduce the food forcibly into its mouth to initiate the act of eating." (1927, p. 251)

In this "typical" experiment, he argues that excitation of various cortical units was developed through reinforcement, i.e., after each stimulus element, food was delivered to the animal. While initially only the food, (unconditioned stimulus, US), elicited salivation, (unconditioned response, UR), later the stimuli preceding the food, (conditioned stimulus, CS), brought forth the conditioned response (CR). Following this, a non-reinforced discriminatory stimulus was presented for a number of trials. This procedure, according to Pavlov, resulted in widespread cortical inhibition (neural description) or sleep (behavioral observation). Pavlov's explanation for this was: (1) "...the cortical elements, which represent the highest point of development of the nervous system, are extremely sensitive and are therefore functionally exhausted with comparative ease," and (2) "The progressively developing inhibition ... assumes the role of a protector of the cortical elements, preventing any excess fatigue or dangerous functional destruction of this highly sensitive structure." (p. 250) Thus, in the preceding example, conditioning had resulted in establishing an <u>excitatory</u> state in numerous cortical elements to a point (near exhaustion?) where any discriminatory stimulus caused the antithesis of excitation - inhibition. Since a number of cortical elements had been

originally excited, this suppression of the activity of neural units (inhibition) was considerably more widespread than the type of inhibition which occurs during the alert state. Thus, sleep resulted!

Pavlov cites several experiments in which various techniques were used to achieve the same results. For example, if a delay procedure was used (where the onset of the CS occurs prior to the US and continues until the presentation of the US) and the CS-US interval (time between the onset of the CS and the onset of the US) was extended beyond 45 seconds, an inhibitory state occurred during the early stages of the CS presentation. In other words, when the CS was first presented and there was a "quick transition from full alertness into true physiological sleep" no salivation was elicited. (p. 253) Toward the end of the CS-US interval, the dogs suddenly awakened and salivated copiously. (See Figure, p. 262)

While Pavlov's behavioral observations are convincing ("The eyes close, the head droops, the whole body relaxes, and hangs on the loops of the stand, and the animal emits an occasional snore." p. 261) some serious theoretical questions can be asked concerning his explanation of this phenomenon. Although localized inhibition of a cortical area adjacent to one in which excitation is occuring is commonly accepted, Pavlov states that during sleep, inhibition "applies to all the cellular structures

of the cortex equally." (p. 250) Thus, the dynamic state of suppressed inhibitory activity pervades the entire cortex.

If, in fact, Pavlov were correct, Kleitman (p. 272-4) suggests some obvious problems for inhibition theory. (1) If the entire cortex is inhibited, where does this inhibition come from? (2) What is responsible for maintaining this inhibition? (3) If inhibition is a result of exhaustion, how is it that the animal falls asleep with each stimulus presentation (employing the long delay CS)? (4) How could dreaming be possible while in a state of complete inhibition? While this list could be extended ad infinitum, let us instead consider some direct evidence in the form of electrophysiological recordings from the cortex. Evarts (1960, 1961, 1962) has recently done microelectrode recording from single units in the visual cortex of the cat during wakefulness and sleep. His results clearly show that the majority of cells from which he has recorded spontaneously fire at a higher rate during sleep. It is doubtful whether these results would have been predicted on the basis of Pavlov's inhibition theory. In fact, Evarts has suggested, on the basis of recordings from Betz cells in the motor cortex, which show very rapid bursts during sleep, that sleep involves the fatigue of inhibitory Renshaw cells. He feels that this idea fits in with such phenomena as the increased susceptibility to seizures during sleep of epileptic patients.

Thus, it can be seen that none of these "early" theories of sleep have proven to be entirely satisfactory. Many were based on propositions which at the time of their presentation could not be proven, or were not capable of incorporating newer discoveries. In the 1930's there was a tendency to move away from attempts at complex theories and to apply the increasing electronic technology to elucidate the mechanisms of sleep. The following section is devoted to the early electroencephalographic studies of sleep and the changes that these brought in the theoretical outlook.

Modern Theory

> "That slow potentials appear in the record when a subject is asleep was one of the first observations to be established in electroencephalography; and since Berger's (1932, 1933-34, 1935) original demonstration of this change, it has been plentifully confirmed by subsequent workers." (Brazier, 1949, p. 195)

The significance and importance of this observation of Berger's was quickly realized by a number of workers in the '30's. For the first time an effective monitoring device was available to measure the electrical activity of the brain. Blake and Gerard (1937, p. 692) have stated: "Electrical potential of the human brain, led from the intact scalp, afford an objective measure of the activity of this organ which may be correlated with other physiological or psychological states." Thus, a considerable number of papers were reported at this time that showed the various changes which occurred in EEG patterns during wakefulness and sleep.

Since the individual papers are not particularly important for our present purposes, only a summary of the findings based on review papers will be described (Blake and Gerard, 1937; Blake, 1937; Loomis, Harvey and Hobart, 1937; Davis, Davis, Loomis, Harvey and Hobart, 1938; Blake, Gerard and Kleitman, 1939).

It is probably generally agreed that the EEG represents "massed synaptic potentials of apical dendrites of mainly pyramidal cells, becoming synchronous and oscillating as fields of maximal amplitude." (Glaser, 1963, p. 6) Normal human EEG, in the waking state but relaxed, usually has a dominant rhythm, called alpha, which varies between 8 and 13 cycles per second (cps). The amplitude of these waves is in the range from 30 to 100 microvolts (uV), and they appear most prominently in the parietal and occipital regions of the brain. As well as the alpha, other frequencies may be present in these areas, but most often in reduced amplitude. When the relaxed subject is presented with sensory stimuli, the alpha rhythm is "blocked" or diminished in amplitude and there remains "beta" activity (15-30 cps) which is faster than the alpha and of lower amplitude. During the early stages of sleep there is an increasing tendency for the alpha to diminish and the gradual appearance of irregular activity in the 4-7 cps range, along with sharp positive waves. Interspersed with this activity may be "spindling" (bursts of symetrical waves) at 12-14 cps or low voltage, fast activity. As sleep deepens the dominant rhythm appears as large, slow, and

irregular waves. Periodic alteration also appear during this phase of sleep. Waking does not necessarily follow the mirror image of the sequence. It generally occurs as a sudden transition from quite slow high amplitude waves to normal alpha. These changes are very dramatic, and occur in almost all subjects.

The effect of the EEG findings was to cause a theoretical reorientation. Several functional aspects of the brain were now obvious. The spontaneous activity of the nervous system does not cease during sleep: what apparently changes is the synchronizing mechanism controlling populations of neurons. Neither is sleep an homogenous process. Different stages and depths of sleep occur, and these are dramatically reflected in the EEG patterns. EEG recordings during sleep show as much variation as do records from the awake subject. Thus interest was shifted to more detailed studies of the controlling mechanisms which lead to alterations in the electrical rhythms of the brain. Along with this, the entire question of the sleep-wakefulness cycle changed from "Why do animals sleep?" to "Why do animals stay awake?" This latter change was largely due to Kleitman's influence (1939, chapter XXXVI). Kleitman's emphasis on the wakefulness portion of the diurnal cycle lead him to propose a two-factor "evolutionary" theory which will now be discussed.

Kleitman's Evolutionary Theory

From Kleitman's view, it is not sleep which requires

explanation, but rather wakefulness. He states, "In spite of sleep being frequently designated as an instinct, or global reaction, an actively initiated process, by excitation or inhibition of cortical structures, there is not a single fact about sleep that can not be equally well interpreted as a let-down of the waking activity." (1939, p. 502) Having thus emphasized the wakefulness part of the cycle, Kleitman describes two kinds of wakefulness- (1) wakefulness of necessity, which is under the direct control of subcortical structures, and (2) a supplementary wakefulness of choice, which along with the typical diurnal sleep wakefulness cycle of adult humans, is a cortical function. Wakefulness of necessity, which has as its counterpart, primitive sleep, is demonstrated in decorticated dogs and human neonates. In these examples, (primitive) sleep is the dominant mode of existence, and is only disturbed by strong external stimuli or unpleasant internal stimuli. The latter might be represented by hunger pains or excretory functions: when the state of the organism is returned to its normal equilibrium (by feeding or elimination) the interoceptive stimuli are reduced, and the organism returns to the sleeping state. Similarly, decorticate dogs can be aroused by strong sensory stimulation, but quickly return to sleep after the termination of the stimulus (Kleitman and Camille, 1932).

In contrast, wakefulness of choice is an associative

function resulting from "a social acculturation to the family and community pattern of living." (Kleitman, 1957, p. 365) This is a major point when comparing Kleitman's theory to earlier models. The early toxin and hormone theories implied that sleep is regular and unavoidable. However, conventional sleeping habits of humans are probably a convenience dictated by our day-night cycle. Kleitman (1952) has shown that daily metabolic rhythms can be altered by avoiding daylight and artificially creating "days" of different lengths. Similarly, Oswald (1962, p. 172) reports that two of his colleagues "adapted" to a 48 hour "day". While they originally took 7.5-8 hours of sleep daily, they only required 11-12 hours of sleep every two days at the end of the experiment. Thus, Kleitman avoids many of the earlier pitfalls encountered when trying to explain the "why" of sleep. The primitive form of sleep requires no special mechanism other than the cessation of external and internal stimuli. Wakefulness of choice and the diurnal sleep-wakefulness cycle are accounted for by social influence and day-night alterations.

The Sleep-Wakefulness Centre Problem

Central to Kleitman's evolutionary theory is the concept of a wakefulness centre, i.e., a sub-cortical area or nucleus responsible for the maintainance of "cortical" and behavioral wakefulness. Kleitman's own work with decorticate dogs suggested that incoming sensory stimuli passed to or through such a wake-

fulness centre. This resulted in arousal. However, without this afferent stimulation the dogs could not stay awake. After reviewing the evidence, Kleitman (1939, chapter XXXV) concluded that the location of such a centre must be in the thalamus of hypothalamus. It is significant that this also represents the location of the last synaptic relay station to the cortex. An important role of this wakefulness centre in Kleitman's original formulation was to "turn off" afferent sensory stimulation to the cortex. However, several lines of research have shown that disturbance of the classical pathways is not crucial.

Bremer (1935) was the first worker to show the effects of various brain stem sections in the cat. If the section was made below the medulla (encéphale isolé), the EEG showed normal alternating patterns of sleep and wakefulness. However, if the upper midbrain of the cat was sectioned (cerveau isolé), the animal showed continuous sleep-like slow rhythms. Bremer felt that this preparation had reduced the total afferent inflow to the cortex to such a degree that the cortex lacked sufficient input to maintain normal wakefulness patterns. This interpretation was maintained until the discovery of the activating properties of the brain stem reticular formation (BSRF) by Moruzzi and Magoun (1949).

Moruzzi and Magoun (1949), measured EEG change from the surface of the cortex in anesthetized cats, while rapidly stimulating (up to 300 pulses/sec.) the BSRF with a low voltage (1-3

volts) pulses. This resulted in immediate desynchronization of the EEG rhythm, comparable to the change that occurs when the cats awoke from sleeping. This change could not be accounted for by direct afferent fibres, since single stimuli to the BSRF did not elicit evoked potentials at the cortex. As well, reticular stimulation did not effect the classical pathways. The authors concluded from their observations: "that a background of maintained activity within this ascending brain stem activating system may account for wakefulness, while reduction of its activity either naturally, by barbituates, or by experimental injury and disease, may respectively precipitate normal sleep, contribute to anesthesia or produce pathological somnolence." (1949, p. 472) This latter thesis was confirmed in a subsequent study by Lindsley, Bowden, and Magoun (1949). Various lesions were made throughout the reticular activating system and EEG recordings were made after recovery. Recordings after making lesions in the hypothalamic regions of the diencephalon and through the tegmentum showed sleep-like EEG patterns or patterns resulting from barbituate anesthesia. Large, slow waves predominated, with recurrent spindle bursts. Lindsley, Schreiner, Knowles, and Magoun (1950) subsequently demonstrated that midbrain lesions of the classical afferent pathways, sparing the tegmentum, left the EEG alterations undisturbed. Normal patterns of sleep and wakefulness still appeared. Thus, the results of Magoun and his

co-workers clarified the findings of Bremer, and indicated the presence of a sleep-wakefulness control centre in the brain stem which is independent of classical afferents.

The role of the BSRF in sleep and arousal has subsequently been confirmed in behavioral studies. Segundo, Arana, and French (1955), for example, demonstrated that the normal sleeping monkey could be awakened by reticular stimulation. Thus, it can be seen that the BSRF is functionally close to Kleitman's concept of a wakefulness centre. There is, however, one important difference (other than location) that should be noted. Kleitman viewed the (hypothalamic) wakefulness centre as a blocking device on the classical afferent paths. Moruzzi and Magoun (1949) suggest the BSRF diffusely projects to the entire cortex thus maintaining "brain tone". It is apparent by his later review (Kleitman, 1957) that Kleitman accepts these modifications to his theory.

Despite the intense interest shown in the BSRF and its possible function as a wakefulness centre, a second group of workers have concentrated on a complex of ascending structures in the diencephalon. Reminiscent of Kleitman's analysis these workers have experimentally demonstrated the importance of the hypothalamus and certain non-specific nuclei of the thalamus. Gellhorn (1957) has shown that generalized cortical excitation can be achieved by direct stimulation of the posterior hypothalamus

in the cat in a manner similar to the results from reticular activation. Similarly, he demonstrated that stimulation of this same area during the presentation of an auditory stimulus augmented the response in the auditory cortex. He concludes from his numerous studies that this "hypothalamic-cortical discharge" is necessary in the maintainance of wakefulness and attention.

Nauta (1946) has also emphasized the importance of the hypothalamic structures. In an extensive series of experiments, he has shown that various lesions and transections in this area of the rat caused significant alterations in the occurrence of behavioral sleep. Bilateral transections or lesions in the hypothalamus resulted in a decrease in the waking capacity. Unilateral lesions in the same area disturbed wakefulness to a lesser degree. However, control sections between the anterior and posterior commissures, 5mm wide, and extending to the ventral border of the thalamus failed to produce variation in the sleep-wakefulness pattern. Nauta concluded from these experiments: "The location of lesions which cause disturbances of the function of waking indicated the existence of a structure in the caudal hypothalamic region and in the adjacent part of the midbrain tegmentum, which is of specific importance for the capacity of maintaining the waking state during the absence of external stimulation ("waking centre")." (1946, p. 314)

These and other observations (Hess, 1957) on the functional

role of the hypothalamus in wakefulness may in fact not be inconsistent with the previous discussion of the reticular formation. Lindsley (1960) points out that "the reticular substance extends in to the posterior hypothalamus and the upward efferent projections from it extend to and beyond the hypothalamus." (1960, p. 1561) Gellhorn himself recognizes this relationship for he states: "Both structures have certain functions in common, and the excitability of the posterior hypothalamus seems to depend on the reticular formation." (1957, p. 4) It appears fairly clear from these connections that the activating system of the brain stem includes this part of the diencephalon.

Modern Comparative Studies on Sleep Mechanisms

By far the most intensively studied laboratory animal during sleep and wakefulness has been the cat. A number of studies have reported that the changes which occur during the transition from wakefulness to sleep are similar to human changes (Clark and Ward, 1945; Dement, 1958; Horovitz and Chow, 1961; Hess, Koella, and Akert, 1953; Hubel, 1960; Jouvet, 1961a, 1961b). The earlier studies (Clark and Ward; Hess et al) noted that during the onset of behavioral sleep there was a tendency toward a general slowing of the EEG pattern and an increasing number of spike and slow wave complexes. Hess et al studied both normal cat sleep and artificially induced sleep. The artificial sleep was induced by stimulating non-specific thalamic

nuclei. This latter observation suggests a possible relationship between the activating system of the brain stem (BSRF) and the diffuse thalamic projection system. However, the details of this interaction are still unclear.[1]

Horovitz et al and Hubel described a deeper phase of sleep as well as the "slow wave" phase. In this phase, low voltage fast patterns appeared similar to those recorded from an alert animal. During this phase the behavioral arousal threshold was increased considerably. These observations have been plentifully confirmed by Jouvet (1961a, 1961b) whose extensive research has suggested a possible controlling mechanism for these different phases.

Jouvet (1961b) analyzed the EEG records of 60 chronically prepared cats. Of this number, there were 15 normals, 11 totally or partially decorticate, 20 with total or partial brain stem section, 12 with partial coagulation of the brain stem, and 2 in which there was total ablation of the cerebellum. Each animal was studied from 6-12 hours a day for a minimum of one week. As well as EEG, eye movements and neck muscle potential were recorded from each cat. The results of the normal controls showed two distinct "types" of sleep varifying the earlier observations. In the first phase (slow sleep) there was a general tendency for the EEG to slow down. As this phase continued, large spikes appeared

[1] For a full discussion of this relationship the reader is referred to Lindsley (1960).

(300-500 uV) in the limbic system. Heart rate and neck muscle potential both dropped "discreetly." During the second phase of sleep, (rapid, paradoxical, or rhombencephalic) low voltage, fast activity appeared in the cortex and mesodiencephalon. These EEG patterns were indistinguishable from wakefulness. At the same time, 6-8 cps activity was recorded from the region of the pontile reticular formation. This phase was also marked by the complete disappearance of EMG activity from the neck muscle and the occurrence of rapid eye "jerks." Behavioral arousal tests showed that in this latter phase, the threshold was raised 2-3 times. Similarly, auditory stimulation insufficient to cause behavioral arousal would return the patterns to the former "slow" phase. Jouvet concluded from these various measures that the rhombencephalic phase represented a much deeper and "profound" sleep than the "slow" phase.

The results of the various lesion and section preparations showed the following patterns: Cats with complete removal of neocortex showed considerable alteration in the EEG patterns recorded from subcortical structures. There was an apparent lack of slow wave and spike activity, 90% of the record being dominated by rhombencephalic sleep rhythms. Sections at the base of the diencephalic formations (cerveau isole, Bremer, 1935) showed continuous slow wave and spike activity when records were made in the cortex above the section. If the recording was from below the section, however, there was rapid activity during

wakefulness and spindles during sleep. Also during sleep, there was a distinct loss of neck muscle potential.

Jouvet concluded from these observations that (1) the slow phase of sleep is a result of the activity of the cortex and/or diencephalic structures, and (2) rapid or rhombencephalic sleep is triggered by the action of structures within the BSRF, probably in the region of the pontile nucleus. This latter hypothesis was tested by making 3-4mm lesions in the pontile regions. These animals exhibited typical slow sleep and arousal patterns, but no trace of rhombencephalic sleep.

The general conclusion from Jouvet's work is that there are two distinct "phylogenetically" different phases of sleep. Slow sleep Jouvet calls "neosleep" since it is probably under the control of neocortical structures. Rhombencephalic sleep, being triggered by subcortical structures is classified as "archisleep."

This classification system is somewhat reminiscent of Kleitman's (1939) two-fold theory. It will be recalled that Kleitman described a primitive sleep along with its complementary state, wakefulness of necessity. This he held to be of a subcortical nature. In contrast, wakefulness of choice and the diurnal cycle were cortical events. However, it is clear from Kleitman's examples (p. 14) that these phases were representative of different experimental conditions or developmental stages. The studies of humans and cats (as well as other species, as we shall shortly see) show that archisleep and neosleep appear in

the normal intact subject during one sleep session. This criticism is not serious since the nervous system of mature mammals represents both higher and lower brain structures. However, a further problem is created by Kleitman's example of human neonates. If Kleitman's primitive sleep and Jouvet's archisleep are analogous, one would predict a typical rapid or rhombencephalic sleep pattern from human neonates. In fact, recordings from the scalp of neonates do not show these patterns but rather show slow activity more similar to "slow" sleep (Glaser, 1963, pp. 6-9). One must, however, be cautious in the interpretation of these results since the functional development of the neocortex in neonates is low and ascending transmission may be sparse. Possibly one crucial test of this Kleitman-Jouvet analogy would be sub-cortical recording from neonates!

Dement (1958) has also recorded EEG activity from cats along with measuring rapid eye movements (REMs). His results showed complete agreement with Jouvet's. Both phases of sleep patterns were clearly recognizable. During the rapid phase (which Dement termed "activated") the low voltage, fast activity was accompanied by considerable REMs.

These observations of the cat have been subsequently confirmed for other species. Weitzman (1961) recorded EEG from the monkey during a continuous eight hour sleep. The results were similar to those of the cat in that both forms of sleep were

apparent. Associated with low voltage fast activity were REMs. These two phenomenon occurred together 16% of the time during a night's sleep.

The electrical activity of the brain of the rat measured over long periods has been reported by two workers. Swisher (1962) measured EEG activity from the surface of the brain while making behavioral observations. He showed two distinct "types" of sleep. The first, slow sleep, was similar to that found in other mammals. The "activated" pattern, however, appeared as a remarkably synchronized 6-8 cps rhythm. This rhythm always followed slow sleep and was characterized by increased arousal threshold. While this phase appeared to be similar to the "archi-sleep" of Jouvet and others, Swisher reported that there were behavioral "shifts" in muscle tonus instead of EMG inactivity. Later, Hall (1963), in reply to Swisher's paper, reported that muscle potential, when recording EMG from permanently fixed electrodes, did disappear during activated sleep. According to Hall, Swisher's observations could be explained by the fact that toward the end of the activated sleep period, the animal exhibited sudden "jerks." These, however, were short lasting and apparently did not effect the level of the neck EMG activity.

It is of interest to note that these 6-8 cps spindles have been recorded in the cat during rhombencephalic sleep only in the BSRF, they appear on the surface of the cortex in the rat, while EEG records from the cat cortex are considerably faster and

of lower amplitude. These differences will be considered in later discussion.

As well as the animals previously mentioned, Ruckebusch (1963) has recently reported a study of EEG activity during sleep in sheep. Both phases of EEG activity were noted, similar to those reported by Jouvet and others.

Thus, as we have seen, there are two distinctly separate phases of deep sleep in the mammals represented.[2] The first of these, "slow wave" sleep is characterized by a pronounced slowing of the EEG pattern, slight attenuation of neck muscle potential and heart rate, and regularity of breathing. The deeper sleep, (rapid, paradoxical, activated, or rhombencephalic) is associated with complete attenuation of neck muscle potential and REMs. The EEG during this latter phase varies from low voltage, fast activity similar to that found in the alert waking state (thus, "paradoxical") in most of the animals studied, to a synchronous 6-8 cps activity in the rat. Jouvet has attempted to show that the first of these stages is under cortical control, while the latter is triggered in the pontile reticular formation.

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[2] It should be noted that rapid sleep has also been recorded from human subjects. This phenomenon was first reported by Blake and Gerard (1937) but attracted little interest until it was shown to be associated with dreaming by Dement and Kleitman (1957). These latter authors demonstrated that when low voltage, fast activity occurred with REM's, awakened subjects reported that they had been dreaming. Since 1957, a considerable number of clinically oriented studies have appeared using EEG and eye movement measures as monitors for dreaming.

The Present Problem

The present status of our information about sleep has now been reviewed, and it is quite obvious that there are large gaps in our knowledge. One such gap, as pointed out by Nauta at the beginning of the paper, is our total ignorance about sleep and the neural mechanisms of sleep, particularly as seen in submammalian forms of life.

This fact is most surprising when one considers the emphasis placed on the function of the cortex in sleep. As we have seen, Jouvet, has attributed one phase of sleep to this structure, and much of his evidence comes from decorticate preparations - certainly a messy and truamic procedure at the best of times. Might it not be instructive to look at alterations in EEG rhythm in an intact animal, such as the bird, which lacks a neocortex?

Furthermore, contemporary theorists all appear to make the assumption that sleep is a biological necessity, for homiothermic animals at least. This assumption is so implicit, in fact, that the question is seldom raised. While it has been shown that sleep deprivation in some mammals can be fatal (Kleitman, 1927) there is little information concerning the problem in lower species, especially birds. In fact, what little observation there is, seems to suggest that perhaps certain members of this class do not sleep! Take, for instance, certain behavioral

observations which have been reported in the literature about the pigeon. Wendell Levi, an eminent authority on the pigeon, writes, "In forty years of pigeon breeding I have been unable to come upon any pigeon at any time that was sleeping, although everybody seems to understand that they do." (1937, p. 365) This observation is largely based on the fact that pigeons rarely, if ever, close their eyes for longer than a fraction of a second (Levi, 1957). While this phenomenon has been noted in mammals, including humans, its incidence is generally quite rare.

Skinner (1960) has informally reported that pigeons in his laboratory have maintained pecking behavior on various schedules for periods up to 1500 hours. In a more detailed paper, Skinner and Morse (1958) reported that by using alternating DRL, FR, and VI schedules they could maintain pecking behavior for over 100 days. Detailed analysis, however, showed the birds did, in fact, take numerous short breaks from their routine. There still remains several runs in which a high level of activity was maintained. One bird, for example "worked" for 12 days with only two pauses greater than 10 minutes. No analysis for pauses less than 10 minutes were reported. Although it is possible that these birds may have slept during the short pauses which occurred, it was certainly not the type or quality of sleep found in mammals. The cumulative records show that during the DRL schedule, the animals typically took a break which was only sufficient to produce another reinforcement.

Further, while the pigeon has proven to be an invaluable subject for behavioral and genetics studies, there are no data concerning its normal EEG patterns. Only one study appears in the literature which reports chronic EEG recording for any bird.[3] Silva, Estable, and Segundo (1959) studied EEG activity in various structures of the "tero" bird (Belonopterus chilensis lampronotus) in the normal state and during "hypnosis." This was achieved by suddenly inverting or rotating the animal while, at the same time, firmly restraining all movement. In their very brief report on this bird, the normal EEG was measured from the corticoid surface of the hemispheres, corpus striatum, cerebellum, optic tectum and mesencephalon. These records, when subjected to electronic frequency analysis, showed a 4-12 cps range suggesting a lack of the synchronization apparent in mammals. During hypnosis, there was a tendency toward slower frequency and increased amplitude. Although these authors attempt to establish a relationship between the records obtained during hypnosis and the sleep state, they admit in a foornote: "So far, the authors have not encountered a situation in which it (the tero bird) exhibited behavior and EEG conforming to a satisfactory picture of sleep for a prolonged period. At night time, in a quiet environment and with little illumination, preparations tended to whow

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[3] Peter, Vonderahe and Powers (1958) have reported the EEG and eye movement records for day old chicks and chick embryos. These workers were, however, primarily interested in the response to photic stimulation and therefore did not report any changes over time.

intermitently large amplitude, slow (1.5-3.5, 8-12 cps) waves."[4] (1959, p. 175) This work thus suggests that EEG rhythms appear quite different than those of the mammals discussed earlier both in wakefulness and sleep. There is also the suggestion that if, in fact, the tero does sleep, it may well be that there exists only one stage in contrast to mammals. It is of some interest that this stage, if it exists, is slow sleep, rather than "archisleep" as Jouvet's theory would seem to suggest.

Thus, prompted both by this scanty previous evidence, and the theoretical importance of information from submammalian animals, three experiments were designed employing the pigeon. In each experiment, electroencephalograms were recorded in order to determine the extent of variation that occurs. As well as the EEG, which has proven to be the most sensitive indicator of the state of awareness, two other physiological measures were made, eye movements and neck muscle potential. Wherever possible, the chicken and the rat were used as controls. The rat was selected because of its convenience, and because not much information is available about its sleep patterns. The brief reports in Science by Swisher (1962) and Hall (1963) are to some extent contradictory, and do not give any evidence about eye movement patterns. The chicken was used, again, because it

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[4] Italics ours.

is easy to get and to keep in the laboratory, as well as the fact that it is known to be susceptible to "hypnosis." Since Silva *et al* believe the EEG of the hypnotized tero bird shows a pattern similar to sleep, it was felt that it would be instructive to see the pattern in another bird.

This thesis then, is an exploratory study of long term EEG recording in three animals with chronically implanted electrodes, and is preliminary in nature. The first part of the work, which is not discussed here, consisted of developing suitable recording methods which could be applied to birds. The principal aim of later phases was to find out what the EEG's of birds looked like, and whether there were any patterns of activity which seemed related to sleep. Though visual observations or behavioral tests of the bird were not carried out, it was felt that recordings of eye movements and muscle activity would give some indication of their behavioral state. In addition, the investigation aimed at giving further data on the mechanisms of sleep in the rat, and on hypnosis in the chicken.

CHAPTER THREE

METHOD

Four separate experiments were carried out. In Experiment 1, samples of the EEG were taken at regular intervals over a 72 hour period to see whether characteristic EEG patterns appeared. In Experiment 2, the effects of experimental "fatigue" were investigated by observing how a 48 hour period of wakefulness effected the EEG. Experiments 3 and 4 were designed to study the effects on the EEG pattern of nembutal and "hypnosis" respectively. As the same general method was used for all experiments, it will be outlined below. The specific procedures followed in each experiment will be described in the next chapter.

Subjects and Apparatus

While the major interest in this study was the common pigeon (Columba domestica), two other species were employed for control purposes. The hooded rat served as a mammalian control and the domestic chicken (Hyline) as an avian control. In all, 14 animals were used: nine male pigeons weighing between 325 and 410 grams, 3 male rats weighing between 320 and 345 grams and two hens 860 and 1100 grams.

The apparatus used for the electrophysiological recording was a four channel Model IV Grass electroencephalograph,

which was fitted with an electronic sampling timer which controlled a set of ST relays between the last amplifier stage and the recording pens. This adaptation allowed the amplifiers to be running continuously and thus avoided a "warm-up" period at the beginning of each sample. The paper drive was also disconnected when the pens were inactive. In Experiments 1 and 2, the timer was set to record 48 seconds every 12 minutes. For EEG recording, the low filters were set at 1.0 and the high at 35 cps; for muscle potential and eye movements, 5 and 70 cps. In several instances it was necessary to lower the high band pass filter in order to attenuate 60 cps interference. The paper drive speed was either 15mm or 30mm/sec.

The EEG machine was located in the hallway near the soundproofed recording room. During recording, Ss were kept in a plywood and wire cage, comparable in size to their home cage, and food and water were available at all times. The outside walls of the cage were covered with grounded wire sereening to reduce interference. Since the door of the recording room was closed and since much of the recording was done in the dark, E could not observe the animals behavior during Experiments 1 and 2. The arrangement gave E the advantage, however, of being able to record for several days without disturbing the Ss.

In Experiment 2 which dealt with "fatigue" a treadmill was used. This consisted of a box 18" x 5" and 14" high with a

plastic window along one side. (For a photograph of this apparatus see Appendix I). The floor of the box was a motor driven continuous canvas belt moving at the rate of 36"/min. This was a relatively slow rate and the box was sufficiently large for most Ss to turn around. No apparent discomfort on their part was noticed during the walking period. Recording during this phase of the experiment was not attempted since it was felt that 1) most of the record would have been dominated by movement artifact, and 2) interference from the motor drive would have been picked up by the recording units, thus rendering any analysis abnormally difficult.

Preparations

All Ss were prepared for recording with intraperitoneal injections of pentobarbital sodium anesthetic (nembutal). When, on occasion, it was found that the initial dosage was insufficient, the anesthesia was maintained with ethyl chloride. Nembutal dosage for the pigeon was 12 mg regardless of weight, and both chickens received 42 mg. The rats were injected with 60 mg/kg body weight.

The electrodes were made from .010" enamelled nichrome alloy wire (Driver-Harris Company, Harrison, N.J.). The wire was mounted on Amphenol "Subminax" (27-9) connectors by scraping 2 or 3 mm of the enamelled finish away and soldering to the connectors using 10% phosphoric acid as flux. Two wires were soldered

on each connector, twisted, and cemented in place using dental cement (Nuweld; Caulk). The electrodes were then cut to the desired length and the bared tips were separated to ½mm.

Following anesthesia, Ss were placed in a headholder for implanting. A standard stereotaxic instrument was used for the rat. For the pigeon and chicken, it was found that the head could be rigidly held by clamping phone jacks in the ears and supporting the beak. A small amount of skin (10mm x 20mm) was then removed from the skull and the skull scraped clean and dried. Burr-holes were made for both the electrodes and supporting jeweller's screws. The screws and electrodes were then inserted and the entire incision filled with dental cement. It was found that by spreading the dental cement to the open edges of the wound, infections were rare. Electrode placements in the rat were in the cortex. In the chicken and pigeon, they were situated in the neostriatum and at the base of the cerebrum slightly posterior to the optic chiasm. The electrode placements were checked at the termination of the experiments by examining histological sections.

Two different techniques were used for recording eye movements in the birds. At first, a twisted bipolar electrode was inserted through a burr-hole in the skull situated directly above the orbit, so that the tips entered the orbit and lay just above the eyeball. In several subjects, two such electrodes were

implanted so that the eyes could be recorded independently. In later subjects, the wires were separated, and one wire was implanted in each orbit. The second technique was used for recording eye movements in the rat. In this case, the wires of the electrode were separated and 2-3mm insulation removed from the ends which were then bent to form a hook. Each end was then inserted under the skin and hooked into the orbit. The Amphenol connectors in each case were cemented in place on the skull.

The electrodes for measuring neck muscle potential were made in the same way as those for the eye. The insulation was removed 3mm from the ends of 25-30mm wires, and these were then embedded in the neck muscle 15mm apart on either side of the midline and sutured into place. The skin over this area was then sutured, and Sulfa powder was applied to the edges of the wound to prevent infection. Following implantation, the Ss were returned to their home cages and allowed to recover for a period of 5-6 days.

The electrodes for these three measures were not implanted in all Ss, since many of the techniques involved were developed during the study. A summary of the implantations and experimental use of Ss is given in Table I.

TABLE I

Summary of Subjects, Preparations, and Experimental Usage.

Subjects		Bipolar Electrodes			Experiment Used			
		EEG	Neck	Eye	1	2	3	4
PIGEON	1	2			x			
	2	2			x	x	x	
	3	2			x			
	4	2			x			
	5	2			x			
	6	1	1	1	x	x	x*	
	7	2		2	x	x		
	8	2	1	1	x	x		
	9	2		2	x	x	x	
CHICKEN	1	2		2	x	x		x
	2	2	1	1	x	x	x	x
RAT	1	2			x			
	2	2	1	1	x	x		
	3	2	1	1	x		x	

* Died from overdose of nembutal

CHAPTER FOUR

THE EXPERIMENTS

Experiment 1

It was felt that if the birds sleep, they should show some distinctive pattern of EEG activity at certain times. To find this out, it was decided to record from the animals over prolonged periods. Also, as we were interested in finding out whether there was any difference in the EEG during the light and during the dark, the following procedure was adopted:

Five or six days after the operation Ss were placed in the experimental cage and observed by E for one-half hour while recording. Following this, samples were taken (48 seconds every 12 minutes) over a three day period. During the first 48 hrs. the room was completely dark. On the last day the room lights were on all the time.

All nine pigeons were used in this experiment and each of the physiological measures (EEG, eye movement, neck muscle) was represented in at least 2 birds. (For complete record of implantations see Figure I.) As well, two chickens and three rats were used as controls.

The results from this and other experiments are illustrated with samples from a few animals. However, the records

from all animals of each type used are similar, and the records shown are representative.

Pigeon: The first most striking feature of the records obtained from the normal relaxed pigeon was the lack of synchronous activity - that is, it was almost impossible to detect any occasion on which any one rhythm was dominant. The normal pattern showed a mixture of frequencies, varying from about 4-12 cps.[5] This observation is in agreement with the findings of Silva et al (1959) on the EEG of the tero bird. There were extensive fluctuations in the pattern from second to second; the relatively prolonged persistence of a particular pattern which is typical of higher mammals (and which can even be seen in the rat) was not observed.

Figure 1 shows the EEG rhythm from the normal relaxed animal. The amplitude varies from 80 to 140 uV., and the mixture of frequencies is apparent. Large slow waves (3-5 cps) occur, as do faster frequencies, a condition not usually observed in the waking normal mammal. It can also be seen that apparent synchrony never persists for longer than a few cycles.

The "arousal reaction", however, is frequently observed.

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[5] The exact determination of the distribution of frequencies is impossible without the benefit of an electronic frequency analyzer. Although Silva et al reported that they had used such an instrument in analyzing the EEG of the tero bird they only reproduced the frequency histograms for their mammalian subjects. This ommission probably suggests that the results of the analysis were particularly variable.

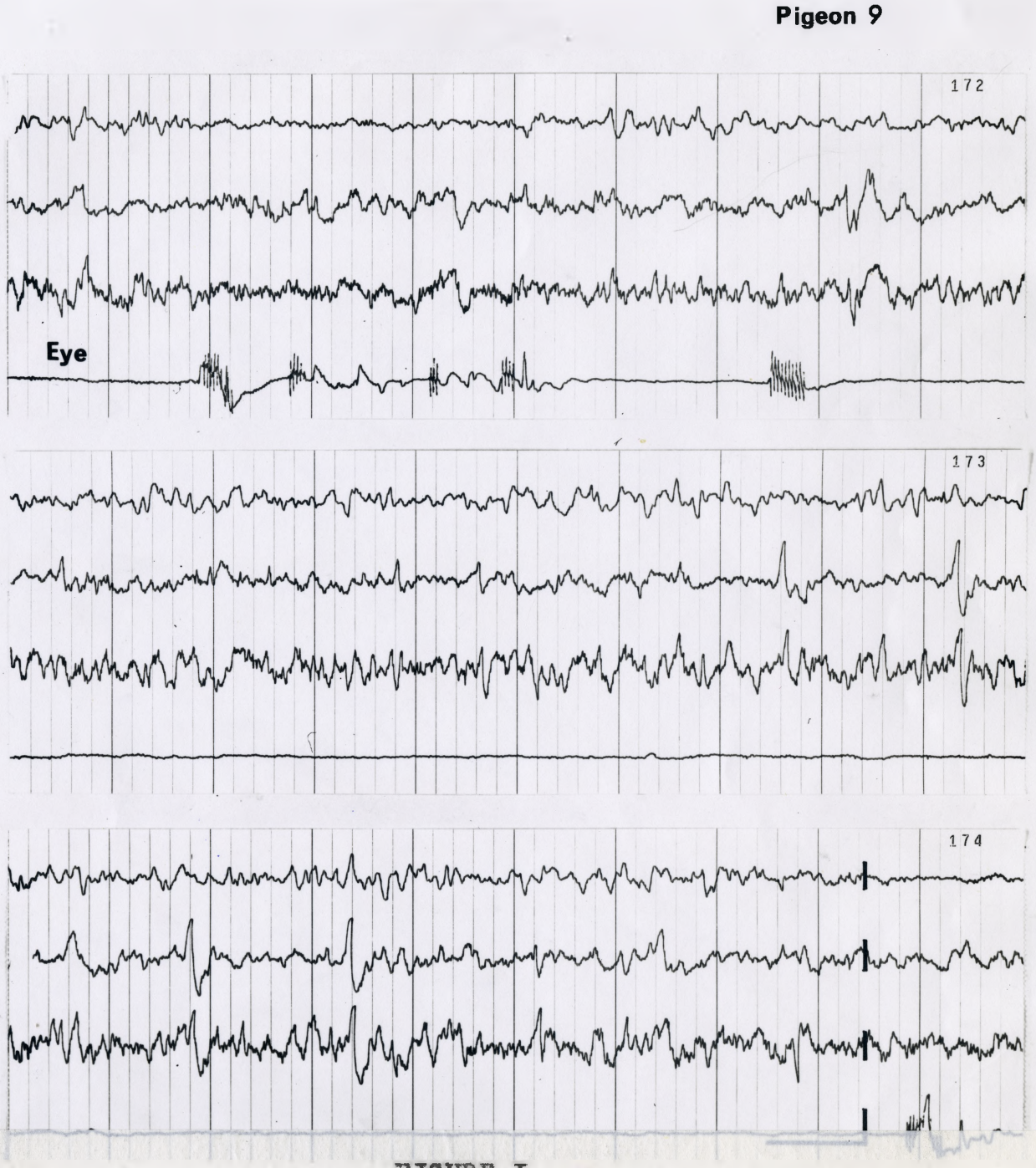


FIGURE I

EEG and Eye Movement Recordings Showing Normal Patterns for Pigeon 9 (Channel 1 - neostriatum; Channel 2 - base of cerebrum; Channel 3 - neostriatum - base of cerebrum; Channel 4 - eye. All calibration signals 100 uV and 1 second unless otherwise noted)

Any strong stimulation of the bird produces the appearance of low voltage, fast activity, and the relative blocking of the slower frequencies, thus causing aflattening of the tracing. An example of this is shown in Figure 2. The change is most apparent on channel 1, where both electrodes are located in the neostriaum. Such blocking could be persistent and was observed to last for a matter of minutes. This flat EEG pattern is also seen to occur "spontaneously" (without any apparent stimulation).

Over the 72 hour sampling period, further variations in the EEG patterns were observed. Besides the types of records shown in Figures 1 and 2 there were periods in which slower rhythms predominated. Figure 3 shows a typical record of this. In this example there is an increase in prominence of the lower frequencies in the range of 2-6 cps and amplitude of the waves is larger (about 200 uV).

Of the three records presented showing range of variation of the EEG, Figure 3 most closely resembles the records obtained from sleeping mammals. However, it should be pointed out that trains of slow waves may also be present in the bird who is observed to be awake. This pattern can be seen in Figure 4. The record in this figure was made before the sampling procedure was begun, while E was observing the bird. The pigeon in this case had just been put into the recording box a few minutes before. There were movements of the head, and, as

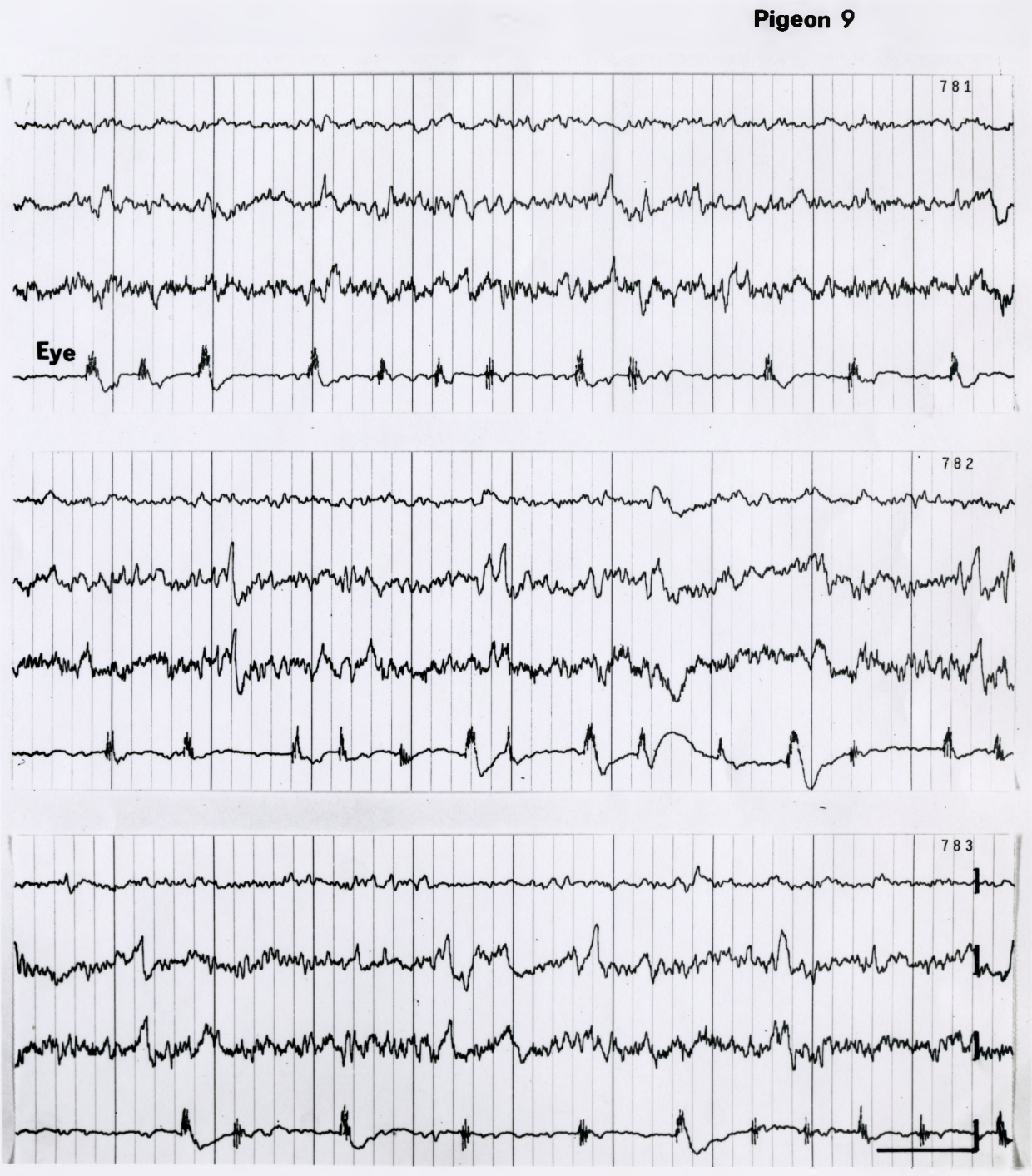


FIGURE 2

EEG and Eye Movement Recordings
Showing Arousal for Pigeon 9

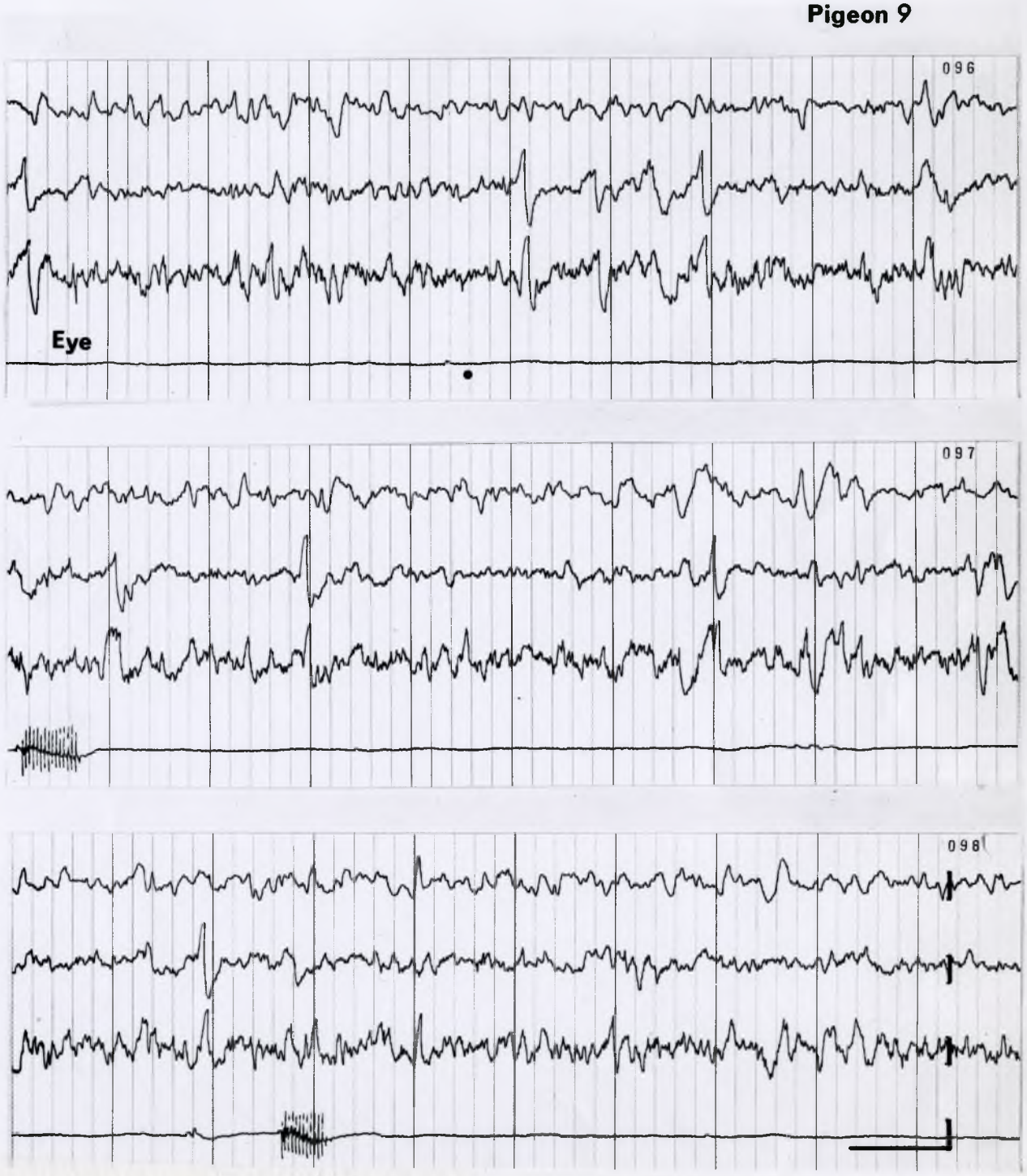


FIGURE 3

EEG and Eye Movement Recordings
Showing Slow Pattern for Pigeon 9

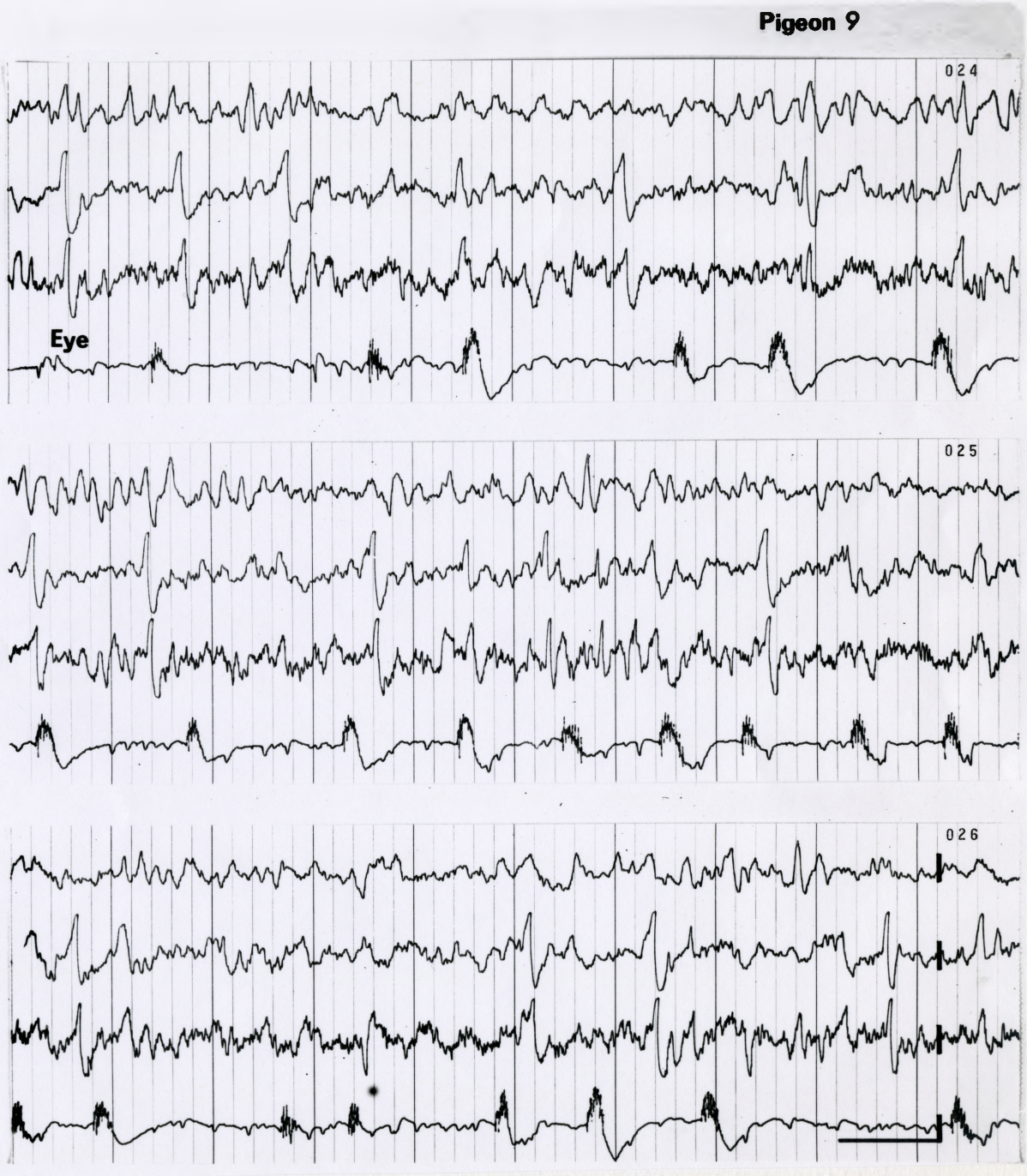


FIGURE 4

EEG and Eye Movement Recordings
While Alert for Pigeon 9

the record shows, a high rate of eye movements. The prominent artifacts in the second and third EEG channels are due to twitching of the tail feathers.

There did not appear to be any differences in the EEG patterns recorded in the light and in the dark, at least not of the magnitude that could be detected by visual analysis. The range of variation seen in Figures 1-3 was evident under both conditions.

As we have seen in the introduction, one of the most prominent indicators of what Jouvet has called "archisleep" in the mammal is the appearance of large rapid eye movements. In our experiments, therefore, electrodes for recording the bird's eye movements were implanted in the orbits. The records obtained from these electrodes, we can arbitrarily say, indicate three different types of waves. The first and most distinctive of these, the tremor or blink, consists of a rapid oscillation varying from 25-40 cps and lasting for .08 to 1.0 second. This process probably results from closure of the nictating membrane and eyelids. When the eye was carefully observed, it could be seen that the oscillations in the record always occurred when the bird blinked, or on the rare occasions when the nictating membrane alone could be seen sweeping across the eye with the lid open.

The second wave is a slow wave (which we will call the "O" wave) which only occurs during some tremors and varies

greatly in amplitude. This phenomenon can be seen in Figure 4 where the tremors occur. The origin of this wave is obscure; it may indicate movement of the eyes, or be related to the basic blink process.

The third type of wave (M-wave) occurs independently of the tremor, and presumably reflects eye movements. These are irregular in form and examples can be seen at the beginning of Figure 1. The records suggest that these movements occur much less frequently in birds than they do in mammals. (This is in line with the unpublished observations of Pritchard and Heron.) This probably reflects the fact that the pigeon moves its head instead of its eyes a good deal of the time.

There is some evidence to suggest that these different types of eye movement may be related to specific EEG patterns, and as well, to general level of arousal. Generally, when slow EEG patterns are evident, the tremors occur less frequently, the rate of oscillation is slower, the duration is longer, and the O wave tends to be small or absent. They also occur at irregular intervals. This can be seen in Figures 1 and 3. When the record is flat, as in Figure 2, the tremors occur frequently, the rate of oscillation is high and they tend to be superimposed on O waves. The correlation between the EEG pattern and the eye movements however, is not a perfect one, since, as we can see in Figure 4, a high rate of tremors is associated with large slow

waves. During this record, as we have indicated previously, the bird was wide awake and alert. Thus, it appears that the recordings from the eye may be a better indication of the level of arousal of the pigeon than the EEG.

In one pigeon, bipolar electrodes were implanted in each orbit to find out whether then eyes moved independently of each other. The records show (Fig. 5) that the tremors usually occurred in both eyes simultaneously and were of the same duration. The M waves, however, were observed to occur either simultaneously (Figs. 5 and 6) or independently in each eye. (Record B, Fig. 6).

A dramatic difference in the rate of occurrence of eye tremors was observed between recordings made in the light and those made in the dark. The change occurred within 10 seconds of altering the conditions. By turning on the lights in a dark room, the rate of occurrence of eye tremor shifted from once every 5 seconds to once every 2 seconds. At the termination of the light, the rate shifted back again to the pre-light level. This change in the tremor rate due to the light conditions of the room did not appear to habituate. During the 72 hour sampling period (48 hours dark followed by 24 hours light) a shift in the tremor rate occurred at the beginning of the light period which persisted to the end of the session. This shift was plotted for three pigeons and is shown in Figure 7. As can be seen from this figure, the percentage distribution of eye tremors for

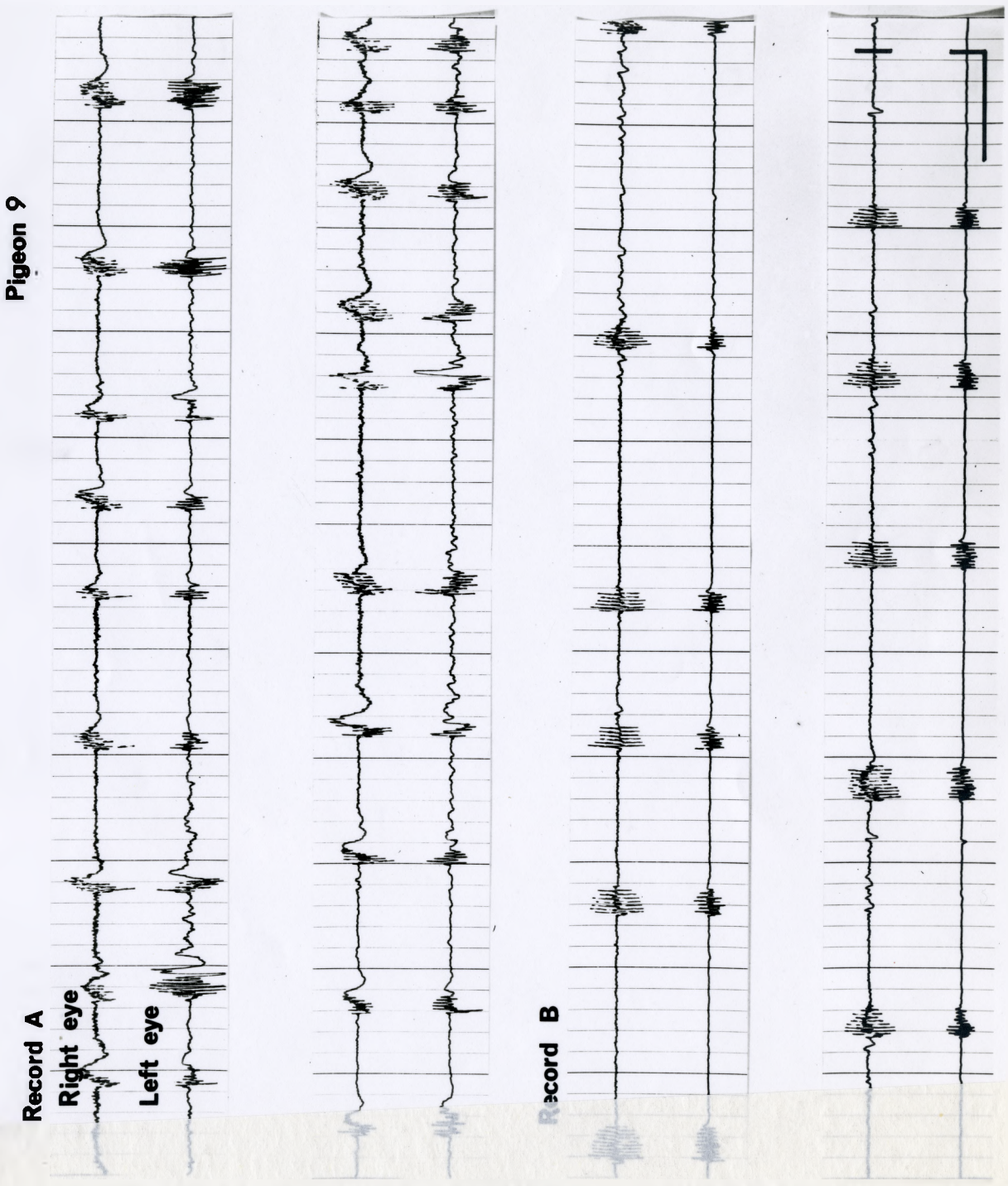


FIGURE 5

Right and Left Eye Recordings

Under Light and Dark Conditions for Pigeon 9

(Record A - light; Record B - dark)

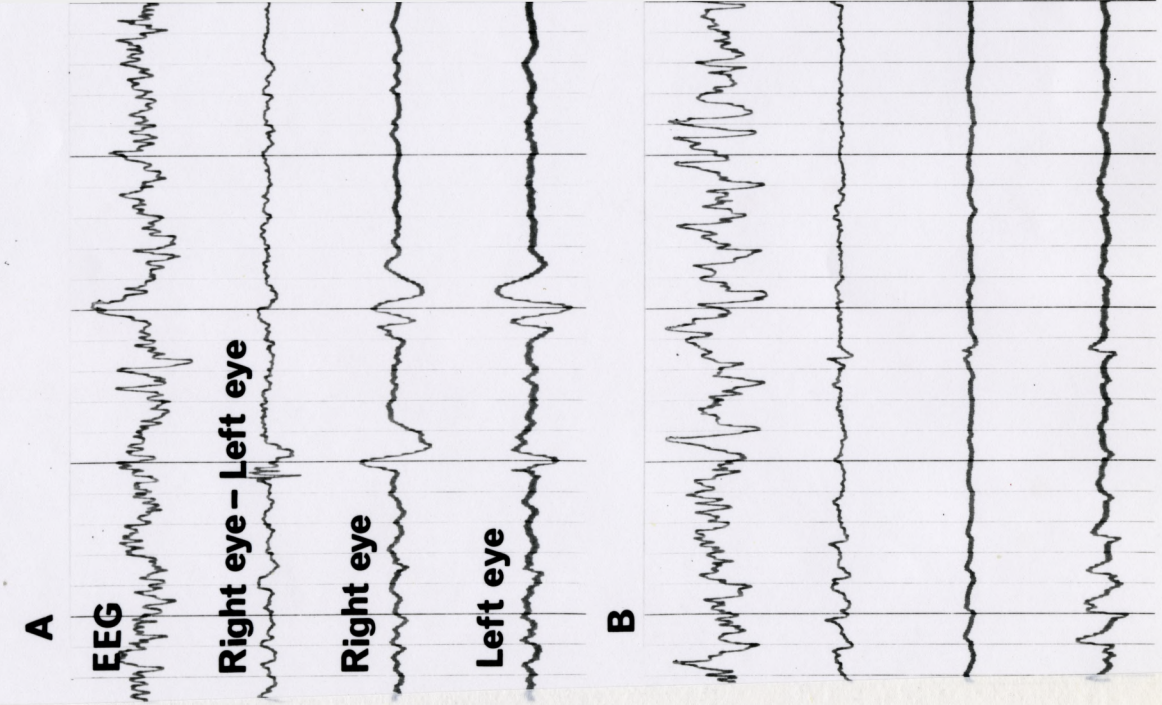
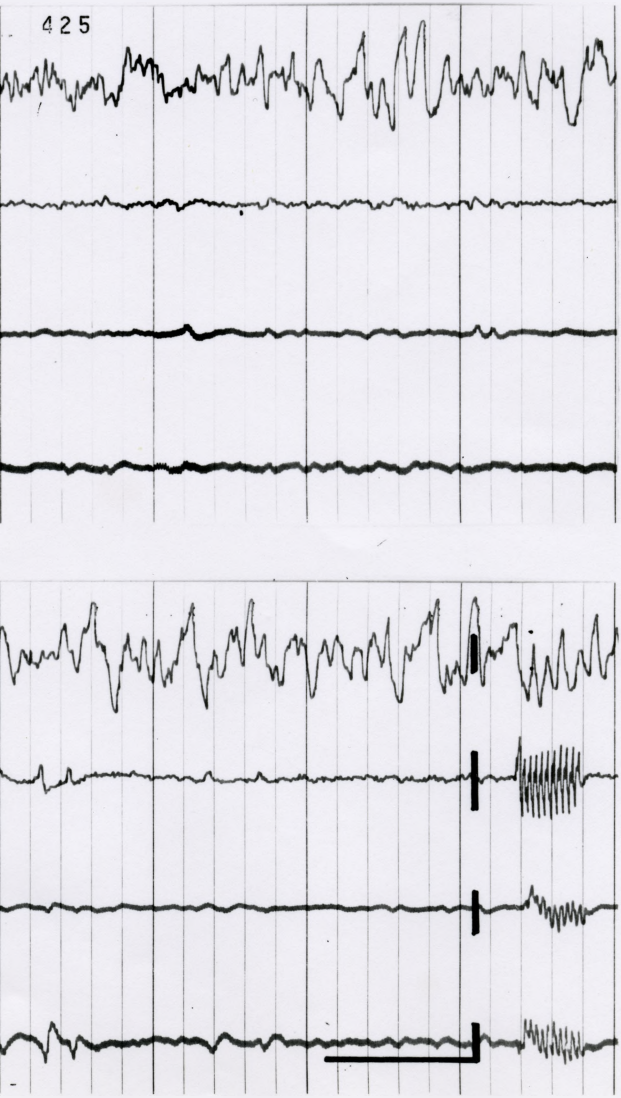


FIGURE 6

EEG and Eye Movement Recordings
Showing Different Eye Movement Patterns for Pigeon 9
(Channel 1 - neostriatum - base of cerebrum)

Pigeon 9



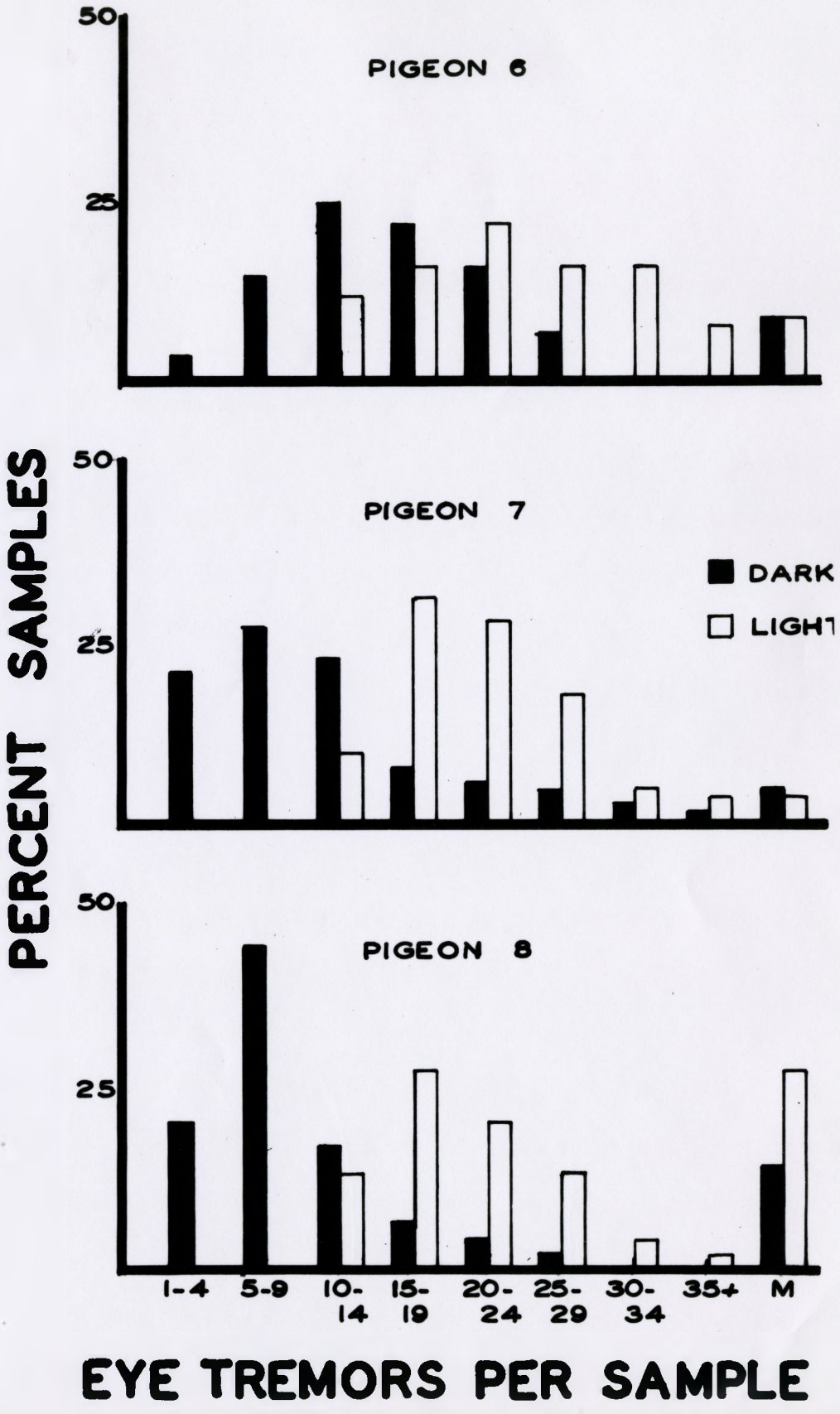


FIGURE 7

Distribution of Samples as a Function of Eye Tremor Frequencies under Light and Dark Conditions for Three Pigeons

different frequencies is slightly skewed for both light and dark samples, but the mode shifts to the higher frequencies during the light period. This change was not due to the initial effect of the light; the 24 hour light period was broken down into 6 hour periods and the pattern was identical for each period. These results suggest that the eye tremors are associated with perceptual functions. However, that they are also related to the degree of arousal in the bird, is indicated by the fact that records taken in the dark, in which the EEG shows a typical arousal pattern, show a fast rate of occurrence of the tremor.

A pattern of activity which bears some resemblence to Jouvet's "archisleep" in the cat was occasionally detected in the pigeon. In records made in the dark M waves occurred at the same time that the EEG pattern showed low voltage activity. An example to demonstrate this phenomenon is presented in Figure 8. In this continuous recording low voltage EEG activity replaces the slower waves during the presence of the eye movements. Shortly after the eye movements cease, the slow activity returns. This pattern of eye movement was not normally seen during slow activity of the EEG, but was observed when the pigeon was known to be awake (from movement artifact or direct observation) and in the light.

From Jouvet, we know that the crucial characteristic of archisleep in the mammal is the drastic reduction of activity

FIGURE 8

EEG and Eye Movement Recordings

Showing Slow Eye Movement and Fast EEG Patterns

for Pigeon 9 (EEG - see Fig. 1)

in the neck muscles. In the case of the pigeon, however, the only time that the neck muscle activity was seen to change was when the bird moved. At this time it showed an increase. Never, when M waves and flattening of the EEG occurred, was there a decrease in muscle potential. Thus, if we take this evidence into account it appears that the pigeon pattern differs greatly from that of the mammal.

Chicken: The range of variation in the chicken EEG recordings was similar to that seen in the pigeon. The lack of synchrony and mixture of frequencies covered approximately the same range. In the normal relaxed bird, waves appeared varying in frequency from 4-12 cps. There is however, a greater tendency for slower rhythms to occur.

The range of variation of the EEG records is presented in Figure 9. Record A (Fig. 9) shows the EEG rhythm of the normal relaxed bird. The amplitude of the waves varies from 80-140 uV. Large waves (3-5 cps) can be seen intermixed with higher frequencies.

The arousal pattern is also observed in the chicken. When this occurs slower waves are attenuated and the record appears to flatten. This pattern can be seen in Record B (Fig. 9). Here the amplitude is reduced to 80 uV.

Besides these two patterns, slow rhythms appeared which showed a high proportion of waves under 6 cps whose amplitude

FIGURE 9

EEG and Eye Movement Patterns for Chicken 1
(Record A- Normal; Record B- Arousal; Record C- Slow Pattern.
EEG- neostriatum - base of cerebrum)

often exceeded 230 uV. These waves were considerably slower and of greater amplitude than those observed in the pigeon records.

The eye movements of the chicken were characterized by the same eye tremor as that of the pigeon. The oscillations, however, were slower (16-25 cps). The duration of the tremor was as much as 2 seconds, which was double the maximum length seen in the pigeon. The same change in the rate of occurrence of eye tremors during light and dark conditions was seen. This is shown in Figure 10. As in the pigeon, there was a tendency for a greater number of eye tremors to occur when the bird was in the light than when it was in the dark.

The M waves occurred even less frequently than they did in the pigeon, and were almost entirely absent. As well, O waves seemed to be less prominent. This can be seen in all three samples of Figure 9.

Rat: The electrophysiological records from the rat are quite different from those of either pigeon or chicken. The extent of this variation is presented in Figures 11 and 12. Record A (Fig. 11) shows the EEG, neck and eye recordings in the normal awake animal.. The amplitude of the EEG varies from 80 to 150 uV. During arousal the amplitude decreases to 50-80 uV with less frequent large waves (Record B, Fig. 11). Light sleep or slow sleep (Swisher, 1962) is characterized by the high voltage slow waves as seen in Record A (Fig. 12). The deeper form of sleep or

FIGURE 10

Distribution of Samples as a Function of Eye Tremor Frequencies under Light and Dark Conditions for Chicken 1

FIGURE 11
Electrophysiological Recordings Showing Normal and Arousal Patterns for Rat 3 (Record A- Normal; Record B- Arousal. EEG 1- deep - shallow cortex; EEG 2- shallow cortex)

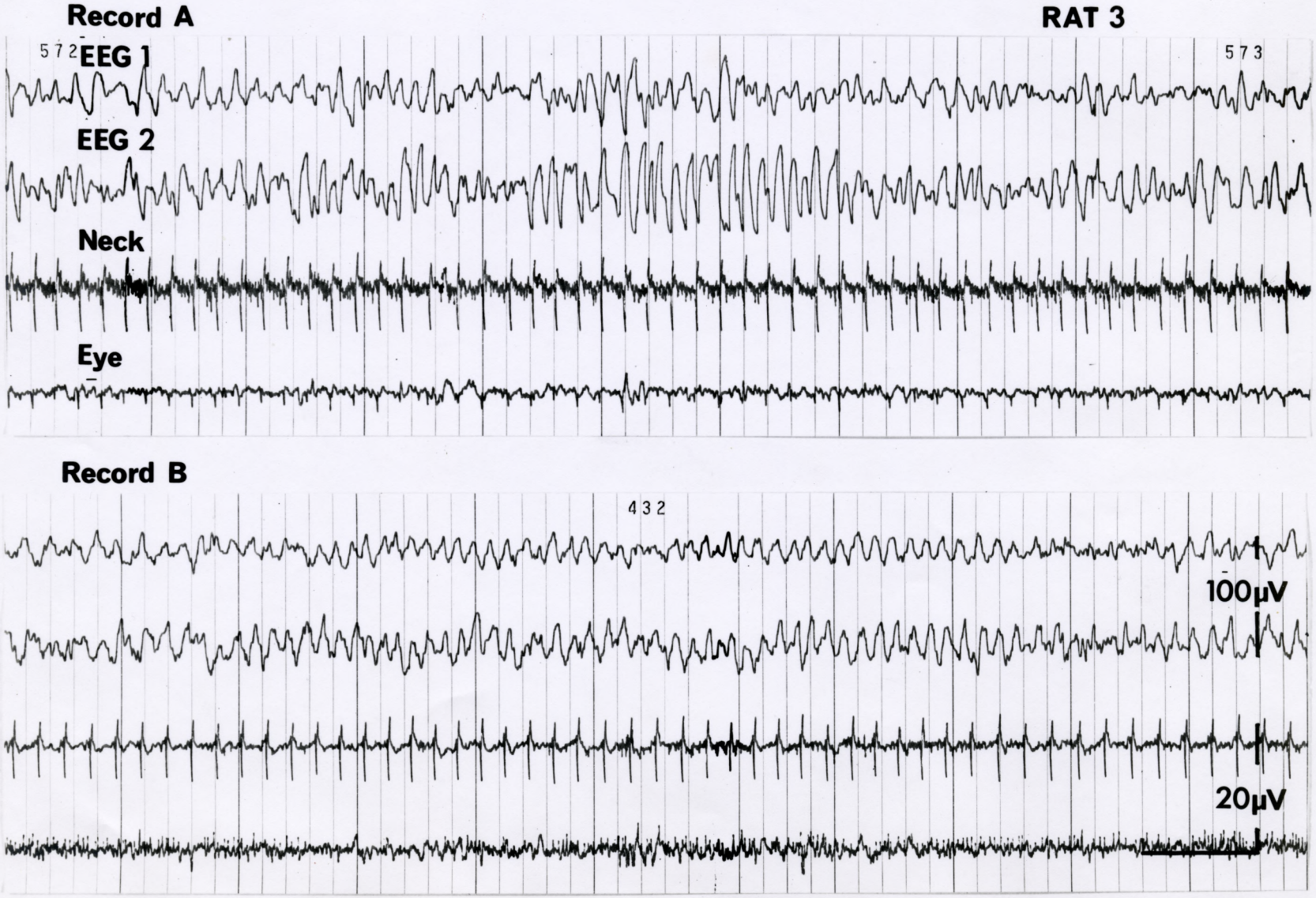


FIGURE 12

Electrophysiological Recordings Showing Two Stages of Sleep for Rat 3

"activated" sleep appears in the EEG patterns of Record B (Fig. 12). As can be seen from this record, the EEG forms a highly synchronized 7 cps rhythm ranging in amplitude from 100 to 130 uV.

Recordings of the neck muscle potential also show large changes. During the aroused state (Record B, Fig. 11), the amplitude of the neck potential ranges from 50-70 uV. During the light stages of sleep (Record A, Fig. 12) the amplitude is reduced to 10 uV. Finally, during activated sleep, there is a dramatic disappearance of all neck muscle activity (Record B, Fig. 12). This observation is in agreement with Hall's findings (1963). As well as the muscle activity, it will be noted that this implant also allowed some estimation of the changes in heart rate during the two phases of sleep. The fast vertical deflections of the pen on "neck" channel represent heart beats. During slow sleep, the heart rate varies between 300 and 320 beats/minute. While this is considerably slower than in the normal alert animal, a full analysis was impossible since much of the data was lost due to the neck muscle potential. During activated sleep, the rate drops to 280-290 beats/minute.

The results of the eye movement recording were less clear-cut. While the eye electrodes for the birds were implanted in the skull, in the rat they were in muscle over the eye. This and the fact that the bilateral eye electrodes tend to pick up EEG rhythms resulted in some interference on the record. However,

in most samples, the eye movements were gross enough to distinguish them above the noise level.

The most obvious eye movement appeared on the records as an oscillatory wave. Frequently, only one or two of these waves appeared together. However, on some occasions, "trains" of these were apparent over several seconds (Record A, Fig. 11). During arousal (Record B, Fig. 11), large waves frequently occur. Trains of these waves may appear at any time except during slow sleep, when eye movement is greatly reduced (Record A, Fig. 12).

Earlier research (Dement, 1958; Jouvet, 1961a,b; Weitzman 1961) presented evidence that eye movements occur during activated sleep in the cat and monkey. During this phase of sleep in the rat these movements are also observed. Although the dominating "activated" sleep pattern in the rat appears as a highly synchronous 7 cps rhythm, on some occasions this rhythm is blocked and low voltage, fast EEG activity occurs. This change is frequently associated with rapid eye movements. The effect is demonstrated in the continuous 30 second sample shown in Figure 13. While there is a complete absence of neck muscle activity for the duration of this sample, the 7 cps rhythm is blocked several times, and low voltage fast waves appear. On each occasion eye movements are observed. Thus, it may well be that these brief intervals of low voltage, fast

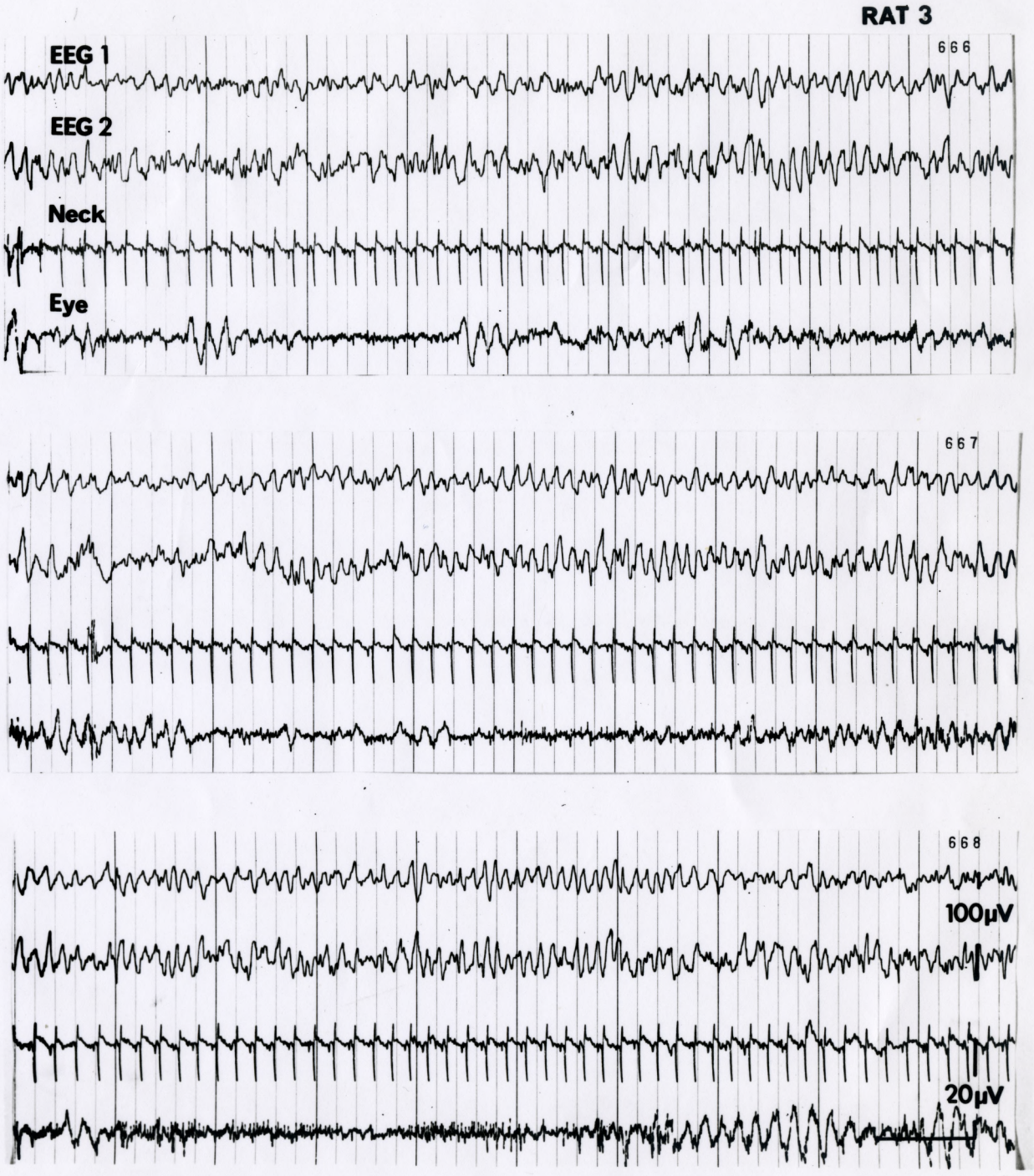


FIGURE 13

Electrophysiological Recordings Showing Blocking During Activated Sleep for Rat 3

activity are the true homologues of rapid or rhombencephalic sleep in higher mammals. If, as Jouvet suggests, the triggering mechanism for "archisleep" is subcortical, the evidence from the rat suggests the 6-8 cps pontile rhythms may undergo considerable modification at the cortical level. This hypothesis will be examined further in the Discussion.

On the basis of the three electrophysiological monitors used, an attempt was made to determine the amount of time spent in the various stages of wakefulness and sleep. Given the present sampling method and lack of behavioral observation, these figures are, at best, only crude estimations of the total picture. However, they have proven to be reasonably reliable within a single rat. Table II shows the percentage of time during two 24 hour periods (24 hours light followed by 24 hours dark) spent in the different stages of wakefulness and sleep for Rat 3.

TABLE II

Percentage of Samples Representing Different Stages of Wakefulness and Sleep in Rat 3

	24 hrs dark	24 hrs light
AWAKE		
Movement	30	19
Alert	21	32
Transitional (Drowsy?)	13	14
SLEEP		
Slow sleep	28	30
Activated sleep	8	5

(The criteria for this classification are essentially the "typical" records shown in Figures 11 and 12. Occasionally medium amplitude waves appear which we have called "transitional".) As can be seen from this table, total time sleeping in light was 35% while the total time sleeping in the dark was 36%. Activated sleep accounted for 5-8% of the total time. The proportion of time spent in activated sleep in two other rats was in the range from 5-10%.

It is interesting to note that the total sleeping time of the rat is not dissimilar to that of the human. The pattern, however, is radically different. The rat tends to sleep frequently and for brief intervals; the longest duration of sleep-type records was 4 consecutive samples, equivalent to 36 minutes. There is still no way of knowing, of course, whether the animal was sleeping between samples.

Experiment 2

The first experiment, then, has not given us any clear indication that birds sleep. On the other hand, there was a slight suggestion in some of the records from the pigeon that brief patterns of eye movement activity, associated with low voltage EEG did occur, and it is possible that this may be related to activated sleep. Since the muscles potential records showed no change during these periods, however, no conclusions could be drawn.

It seemed reasonable to suppose that if we were to keep the birds "awake" and working at some task for a prolonged period, a comparison of the records before and after this treatment might differ.

Five pigeons, two chickens and one rat were used in this phase of the study. All Ss had electrodes for EEG and eye movements, the rat and one chicken had electrodes for recording neck muscles as well. The procedure was to put the Ss in the recording box for one day's adaptation. Recordings were then taken for 24 hours in the dark (Pretest). Following this, they were put on the treadmill for a 48 hour period. (The chicken was only subjected to this procedure for 24 hours since it tended to lie on the treadmill.) A half-hour break was given at the end of 24 hours for feeding and watering. Immediately on removal from the treadmill, Ss were replaced in the recording box and records taken for 12 hours, in the dark (Test). A comparison was then made between the Pretest and Test periods.

The effects of this treatment on the pigeon were generally quite limited. All of the electrophysiological measures taken in the Test period seemed normal and there appeared no extensive change due to the "walking." Test records were similar to the Pretest records, and of course, to those reported in Experiment 1. Since gross inspection of the records seemed to show no differences, various forms of analysis were carried out. As all of these were

based on the oscillograph tracings, and because of the variation apparently typical of the avian brain, they were necessarily crude.

First, an attempt was made to find out whether walking had any effect on the percentage of time that low voltage activity dominated the EEG pattern. This analysis revealed no differences between the Pretest and Test records. In the Pretest period, blocking in the pigeon occurred between 16 and 27% of the time, while in the Test records the variation was from 17 to 30%. Within an individual bird, the change was never greater than 3%.

Next, a wave count analysis of the type suggested by Engels et al (1944) was employed. This was done by counting the number of waves in each one second interval, and calculating the percentage of the total time that intervals with the various number of waves occurred. This was done for samples within the first four hours of the Test period and compared to an equal interval at the corresponding time of the Pretest day. The results of this analysis for a typical bird are shown in Table III. It is apparent that this technique showed no differences beyond those expected from random variation. The distributions of wave counts before and after walking are very similar. It is possible, of course, that differences may have been obscured by the fact that pigeon EEG patterns contain a mixture of frequencies, and this technique is most powerful

TABLE III

Percentage of Wave Counts at Different Frequencies in Pretest and Test Conditions for Pigeon 2

	FREQUENCY							
	6	7	8	9	10	11	12	L
Pretest (HOURS 1-4)	4	10	23	33	10	3	3	13
Test (HOURS 1-4)	4	9	26	33	10	3	2	14

when applied to records which show a dominant rhythm.

It will be recalled from earlier that M waves accompanied by blocking of the EEG occurred in the pigeon (Fig. 8), and that it was felt that this might be similar to the activated sleep pattern of mammals. Although these data were only available for one pigeon, the results of our procedure tended to show a slight decrease in the occurrence of the eye movement and flat EEG complex. In the first 8 hours of the Test period, this phenomenon was seen only three times for a mean duration of 4.0 seconds. During the corresponding Pretest period, it was present 12 times for a mean duration of 5.4 seconds. Whether this change is significant, will have to await varification from further experimentation, but it strongly suggests that, at any rate, the walking procedure had little or no effect on the length of time this pattern appeared. On the basis of the duration of the pattern, it seems obvious this phase is not similar to activated sleep in the mammal. In the rat, for example, the

activated pattern frequently exceeded 48 seconds.

An analysis of the pattern of eye tremors between the Pretest and Test periods was also made. This analysis showed little difference between the two patterns.

Thus, the general impression gained from these results is that the walking procedure had little effect on the pigeon EEG or eye movement patterns. If, in fact, any combination of these factors does represent a form of sleep, the lack of change between Pretest and Test suggests that the experimental treatment had not effected the normal pattern. By way of conclusion, there still appears to be no convincing evidence for a highly differentiated sleep pattern in pigeons. The results of this experiment suggest, in fact, that this is especially true of the activated sleep found in mammals.

This conclusion is supported by the chicken data. The chicken records were analyzed in the same way as were those of the pigeon. Wave count frequency distributions showed no differences between Pretest and Test periods. A summary of the percentage wave counts for the two periods is presented in Table IV.

Similarly, the percentages of time that low voltage EEG was present were compared for the two periods. In Chicken 2, for example, flat records appeared for 14% of the time in the Pretest period and 15% during the Test. Counts of the

incidence of eye tremors were also made for the Pretest and Test periods and no difference was seen. As in the pigeon,

TABLE IV

Percentage Wave Counts at Different Frequencies in Pretest and Test Conditions for Chicken 2

	FREQUENCY								
	6	7	8	9	10	11	12	13+	L
Pretest (HOURS 1-4)	5	11	21	20	18	11	8	6	
Test (HOURS 1-4)	2	11	18	18	20	14	8	7	

the Test records of the chicken appeared similar to both the Pretest samples and to those shown in Experiment 1.

The EEG records from the rat were analyzed by eye for two periods, the first 20 hours during the Pretest and the first six hours of the Test. The results of this analysis are shown

TABLE V

Percentage of Samples Showing Different Stages of Wakefulness and Sleep During Pretest and Test Conditions in Rat 2

	20 hr Pretest	6 hr Test
Awake		
Movement	40	17
Alert	27	43
Transitional (Drowsy?)	10	17
Sleep		
Slow sleep	13	17
Activated sleep	10	6

in Table V. It can be seen that the walking procedure caused a drastic reduction in the amount of movement of the animal, and that there is an increase in the amount of the "drowsy" type record. Little change, however, occurs in the total percentage of records showing slow and activated sleep.

Experiment 3

As attempts to modify the EEG patterns of the pigeon and chicken by the walking procedure seemed to have no effect, it seemed reasonable to see what results would be produced by a barbituate anesthetic. As this is known to have potent effects on brain stem mechanisms in mammals and causes large slow waves (which are somewhat similar to those seen in sleep) to appear in cortical and subcortical structures, it seemed of interest to find out how the pattern of the bird's EEG would be affected.

The procedure was as follows: The animal was put in the recording box and a sample record taken. It was then injected intraperitoneally with nembutal (40-60 mg/kg for the birds, 60 mg/kg for the rats) and the experimental recording was begun, and continued for one hour. Three pigeons, one chicken, and one rat were used.

The records for a pigeon are shown in Figure 14. It can be seen that slow waves begin to appear after 2½ minutes. After 10 minutes, frequencies in the range of 3-6 cps are most prominant. However, it should be noted that even under these conditions,

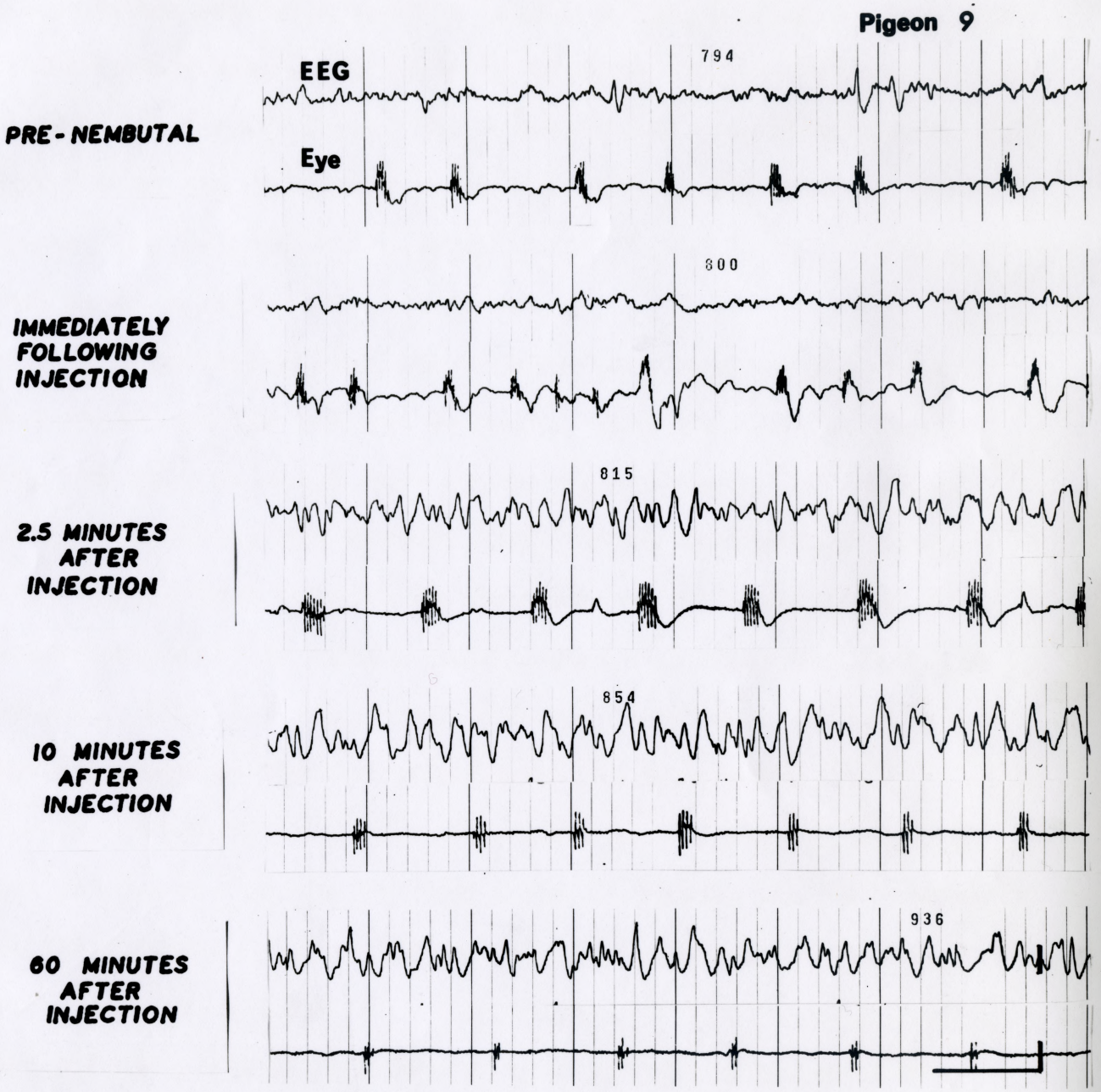


FIGURE 14

EEG and Eye Movement Recordings
Showing Effects of Nembutal on Pigeon 9
(40 mg/kg. EEG --neostriatum)

the pigeon's EEG shows a considerable lack of synchrony.

The eye movements of the pigeon show several changes. The tremors attain a greater degree of regularity as the anesthetic progresses, and after 10 minutes show a decrease in both duration and amplitude. The O wave shows a similar decrease. The M wave eye movements which are present immediately following the injection are practically absent by 2½ minutes, and by 10 minutes have disappeared.

Recordings from the chicken show similar patterns to the pigeon in all but one respect. This is shown in Figure 15. It will be noted that in the final record (60 minutes after injection) eye tremors have become exceedingly rare. (The rhythm that appears in this channel is a result of EEG interference.) By actual count, they had decreased to 1 per minute. This was the lowest rate of occurrence of the eye tremor recorded for the chicken in all experiments.

Alterations in the EEG, neck muscle potentials, and eye movements for the rat are shown in Figure 16. The effects of the nembutal on the EEG rhythms are apparent 2½ minutes after injection and consist of a large increase in the amplitude of the pattern. This is followed by a general slowing of the waves, 20 minutes after injection. At this time too, there was a complete disappearance of neck muscle potential.

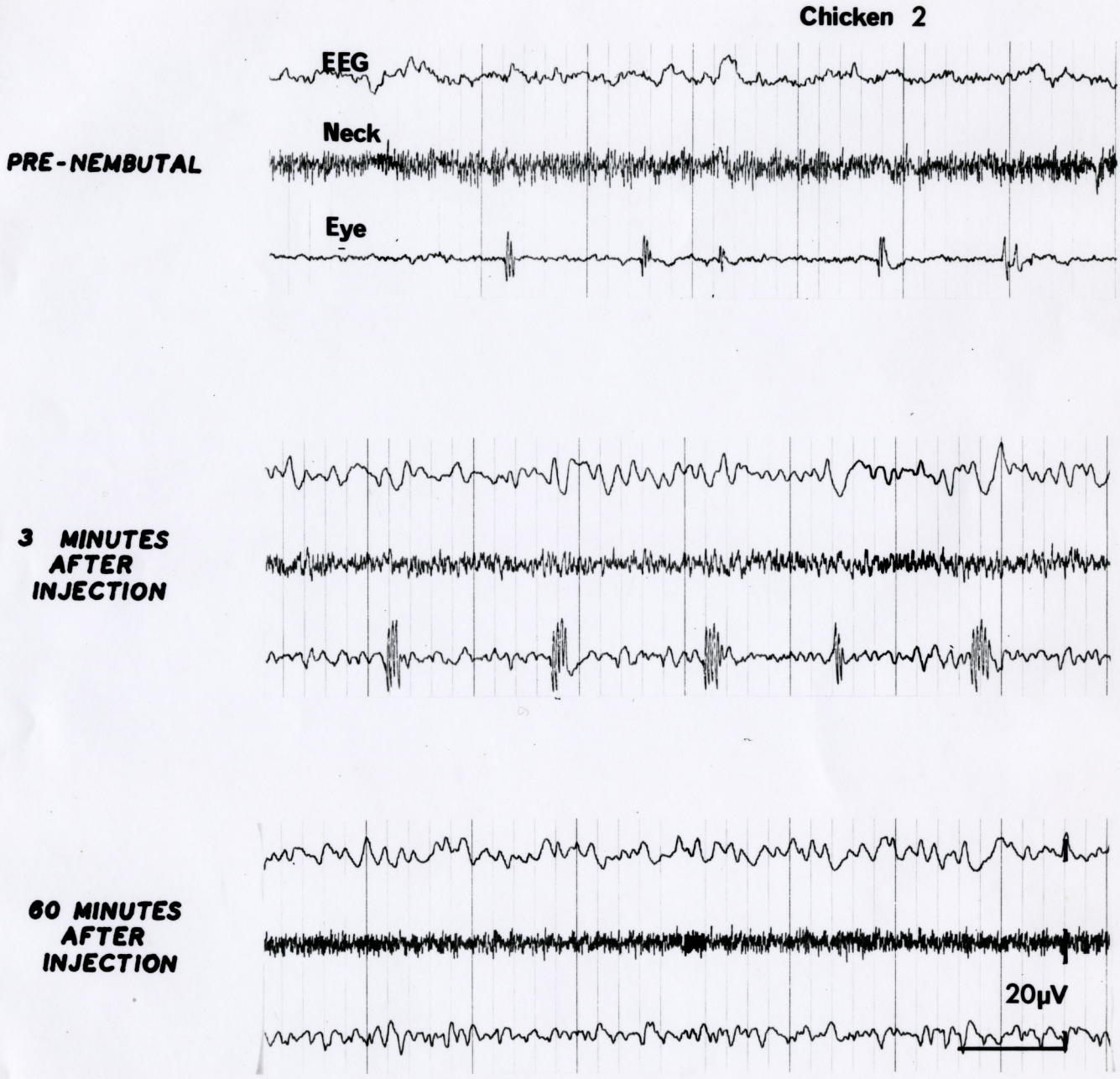


FIGURE 15

EEG, Eye Movement and Neck Muscle Recordings
Showing Effects of Nembutal on Chicken 2
(40 mg/kg. EEG - neostriatum)

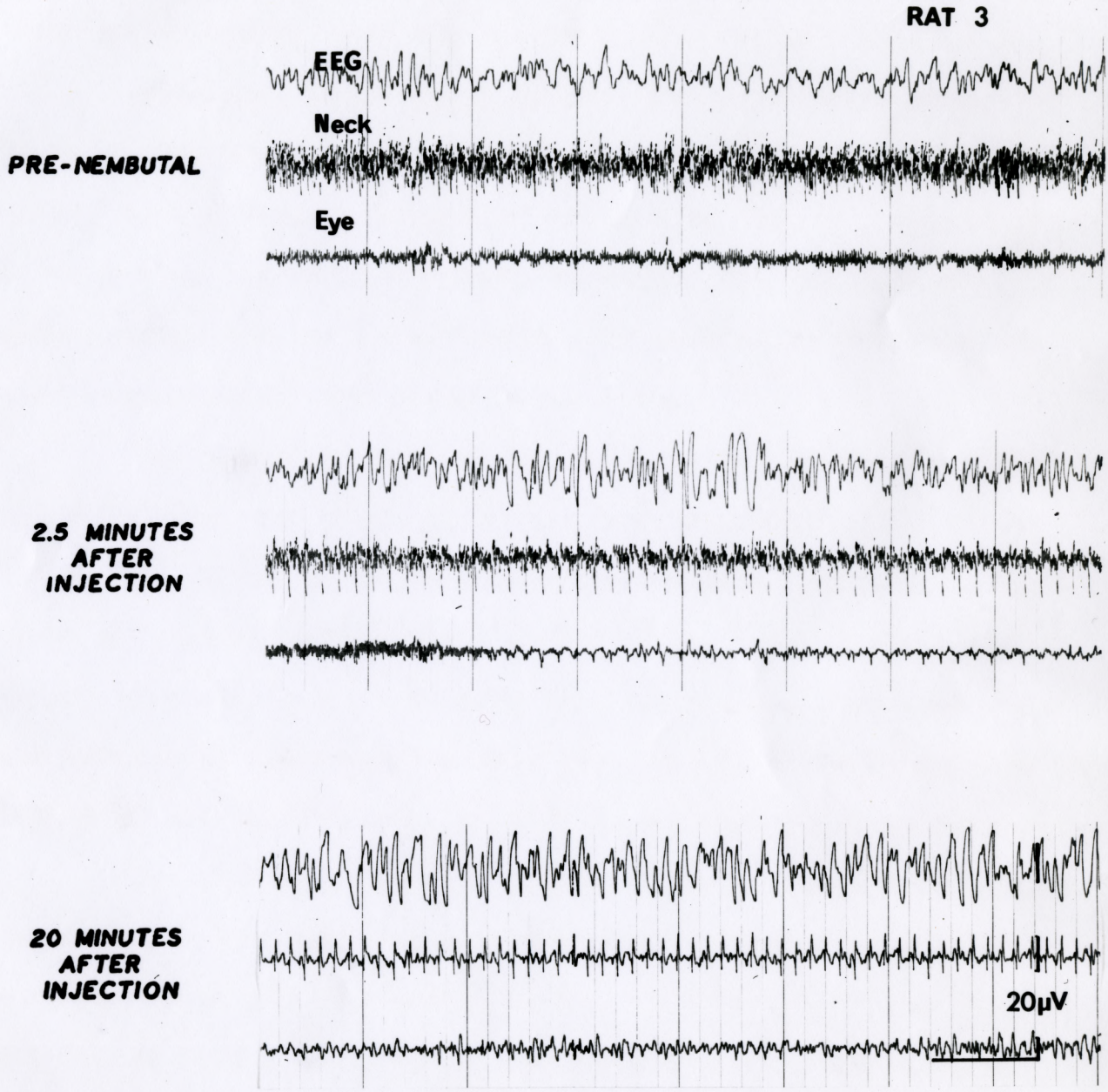


FIGURE 16

EEG, Eye Movement and Neck Muscle Recordings
Showing Effects of Nembutal on Rat 3
(60 mg/kg. EEG - shallow cortex)

Experiment 4

The previous experiment has shown that nembutal causes the appearance of high amplitude, slow frequency rhythms in birds which differ from any observed under normal conditions.

Recently, Silva et al (1959) have shown that structures in the brain of the tero bird show slow patterns of activity during "animal hypnosis" which they feel are similar to sleep waves. As other attempts to modify the EEG without giving drugs had been ineffective, we decided to see what effect "hypnotizing" the birds would have.

The procedure adopted was to hold the head of the bird under its wing and place it on its back restraining all movement. The pigeon, however, was not very susceptible, and only a light state of immobilization could be achieved. The bird's eyes stayed open and there were many head movements. The procedure was completely unsuccessful when the EEG electrode leads were attached to the bird's head, so that no records could be obtained.

The chicken, however, proved to be an ideal subject for "hypnosis." The bird was apparently not effected by the recording box or the connecting leads, and "hypnosis" was successful in better than 80% of the attempts. The duration of the hypnotic sessions ranged from 3-15 minutes, and no doubt would have been longer if there had not been test stimuli presented. Examples of the changes which occur during "hypnosis"

are presented in Figure 17. Immediately on release of the bird (at first arrow), the EEG pattern appears generally flat (Record A, Fig. 17), and the eye tremor rate is about 1 per second. This flat record continues for about 30 seconds when large slow (3-5 cps) waves begin to appear (Record B, Fig. 17). With this slowing of the EEG, the eye tremor rate drops slightly. After one minute, the EEG is dominated by the slower waves (Record C, Fig. 17). Record D (Fig. 17) offers an example of arousal under hypnosis. The arrow represents the time at which E clapped his hands. This action resulted in a sudden attentuation of the slower waves and a slight increase in the rate of eye tremor. Record E (Fig. 17) is a continuation of Record D. The duration of this "blocking" pattern was from 60 to 80 seconds. At the end of this period, the slow wave pattern returned, and remained until blocked by stimulation or until the bird came out of immobilization.

Behavioral observations show that changes occur only during the early stages of hypnosis. During the flat EEG pattern, the eyes remain open, claws are stretched out rigidly, and the head undergoes shifts in position. After the first 60 seconds, however, the animal appears to reach a deeper state. The eyes close, the claws curl under the breast and there appears to be a general relaxation of muscle tone. During the early stages, twitching of the head and legs often occur in

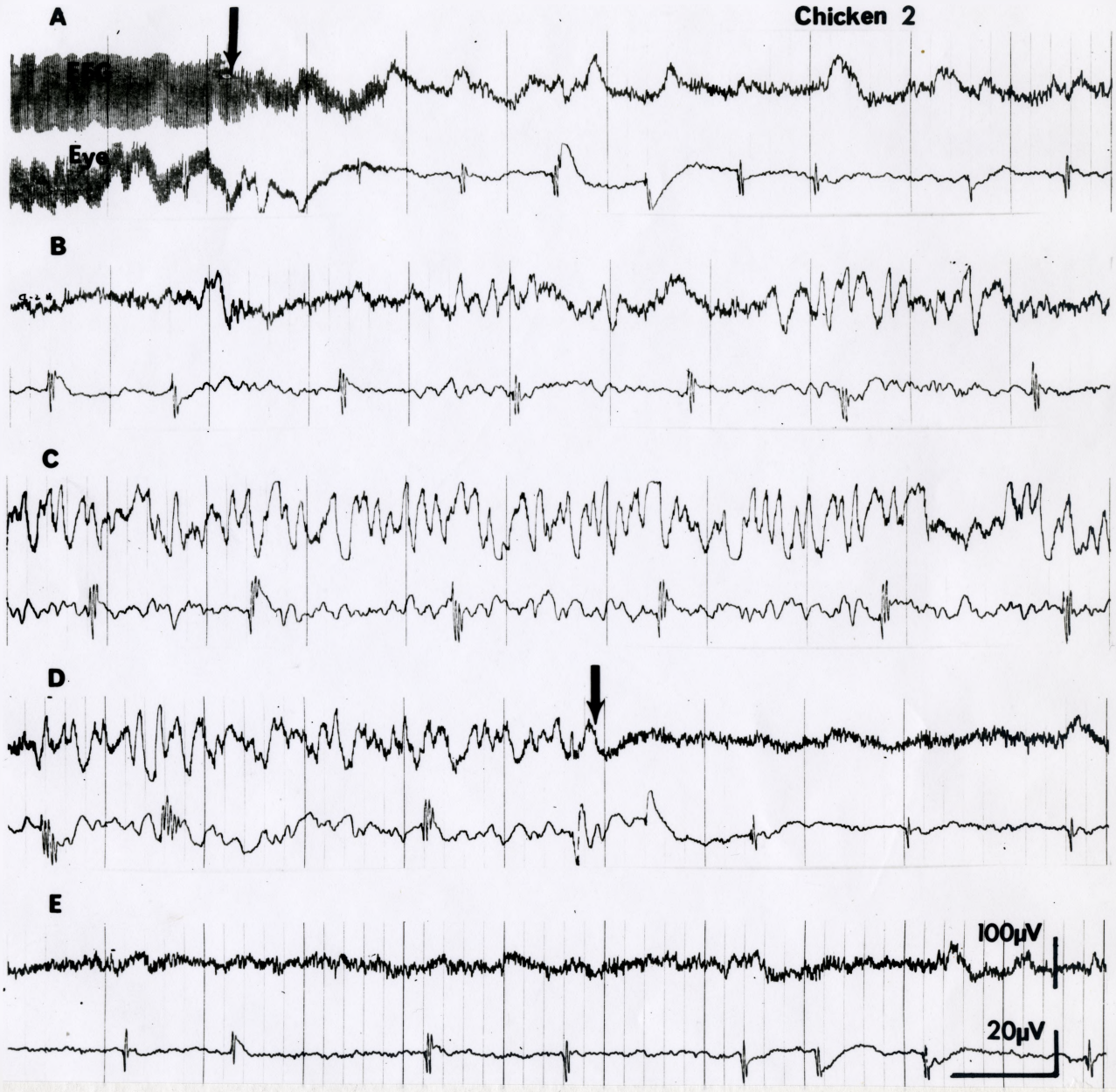


FIGURE 17

EEG and Eye Movement Recordings
Showing Changes During Hypnosis in Chicken 2

(Record A - Chicken released by E at arrow; Record B - 30 seconds after release; Record C - 60 seconds after release; Record D - arousal caused by hand-clap at arrow; Record E - continued from Record D. EEG - neostriatum - base of cerebrum)

response to auditory or visual stimuli. However, during deeper stages as characterized by the slow EEG patterns, there is little behavioral reaction to these same stimuli. In Record D (Fig. 17) for example, the stimulus which caused such a profound change in the EEG patterns failed to evoke any behavioral response.

Recovery from hypnosis was sudden and usually occurred naturally. E rarely woke the bird by presenting auditory or visual stimuli. However, the slightest tactual stimuli (touching the bird or knocking the cage) had the effect of immediately arousing the bird to full wakefulness.

CHAPTER FIVE

DISCUSSION

This study, then, has described the EEG and eye movement pattern of two kinds of birds over long periods of time, and no evidence was found that indicates that sleep occurs. In addition the study has suggested that the activated sleep pattern of the rat may be closer to that of other mammals than previous workers had believed. Finally, some peculiar records associated with "hypnosis" in the chicken have been obtained.

The most salient characteristic of the birds' EEG is the striking lability of patterns and the simultaneous occurrence of a wide range of frequencies which appear in records obtained from most structures in the brain. Although the records presented are limited to structures in the neostriatum and to structures in the base of the cerebrum, other placements in preliminary experiments have shown the same picture: At no time do highly synchronized rhythms appear. It seems, in fact, that the sychronizing mechanism which must be responsible for the relatively high rhythmic patterns of activity observed in the mammal are absent or do not operate in the same way in birds. Alternatively, it is conceivable that there are fewer interconnections between functional cell groups and consequently less reciprocal influence. In any case, the results

suggest that it would be of great interest to carry out investigations similar to those which were done on mammals to demonstrate the recruiting and augmenting responses, to see whether there are equivalent synchronizing mechanisms in the bird.

As we have said, the EEG and eye movement patterns do not give any indication that sleep occurs. There were no general changes in EEG activity noticed over the 72 hour sampling period, nor could any changes be detected after the birds had been wakling for 48 hours. However, slow waves do occur, and there are periods in which they are more prominent than in others. In addition, these periods are frequently associated with the long, multioscillation eye-blink pattern not superimposed on the O waves, a pattern which is atypical of the alert animal. However, it should be noted that slow EEG patterns also occur in the alert bird (see Fig. 4), and that O wave "tremors", accompanied by high voltage slow waves in the EEG are found in the "hypnotized" chicken. The suggestion is very strong that in this case the bird is awake, since very weak stimuli cause a dramatic blocking of the EEG. Thus, like Silva et al, we are forced to conclude that there is no clear electrophysiological evidence for sleep in birds.

If they do sleep at all, the results suggest that the state is probably associated with slow waves. The "activated"

sleep pattern which presumably would be predicted by Jouvet, occurs rarely, and then only for periods of a few seconds.

As far as the rat is concerned, the study shows that the activated sleep pattern is accompanied by eye movements, though these are apparently less frequent and prominent than in the case of higher mammals, as far as can be gathered from published accounts. The 7 cps rhythm reported by Swisher and by Hall is very prominant, and persists for longer periods of time than our samples (48 seconds). The results agree with Hall in finding that muscle tone disappears during this period, and do not fit in with Swisher's observations.

The mechanisms involved in causing the 7 cps rhythms which appear at the cortical level in rats but not in higher mammals is still obscure. It is possible, of course, that they are triggered by the pontile system as Jouvet suggests, and are in synchrony with this system. One observation which was made which is of interest was that frequently, during the 7 cps rhythm, blocking occurred for brief periods of a few seconds. Thus, it may be that the higher brain stem systems responsible for cortical blocking are not as effective under these conditions in the rat, but are still operating.

The action of the nembutal was somewhat different for the birds than for the rat. In both the chicken and the pigeon the EEG waves appeared both slower and of greater amplitude than

those seen at any time in the normal state. The rat EEG under the barbituate was quite similar to that seen during slow sleep. Thus, these results suggest that different controlling mechanisms for normal and barbituate induced slow wave EEG patterns exist for birds and mammals.

* * * * * * * *

The results of this study had indicated several directions for future research. First, since the electrophysiological measures did not give clear indication of sleep patterns in the birds, it would be interesting to study any variations in the arousal threshold and attempt to correlate this with the EEG patterns. Possibly a programme similar to that of Horowitz and Chow might be informative. These workers tested the cortical arousal threshold during different EEG patterns to varying levels of brain stem stimulation.

More interesting, perhaps, is the problem of brain control mechanisms. What structures in the bird's brain, for example, are responsible for the extraordinarily large slow waves that appear in the chicken during hypnosis? What does the activity of the brain stem activating system look like during hypnosis? If knowledge of the changes in these structures were available, greater understanding of the avian brain and its relation to the mammalian brain would be forthcoming.

SUMMARY

Electrophysiological recordings of EEG, eye movements, and neck muscle potential were made from three species- the pigeon, chicken and rat. These measures were sampled over 72 hour periods, after experimental "fatigue", and during nembutal anesthesia. As well, records were taken from the chicken during animal hypnosis. Certain characteristic differences were noted between the EEG of birds and those of the rat. While the rat showed two distinctly different types of sleep (slow and activated), the bird data gave no indication of any EEG patterns which could be related to mammalian sleep, or any suggestion that the birds slept at all.

Eye movement results also showed differences between the birds and the rat. In the pigeon slow eye movements appeared both independently of and simultaneous with a faster oscillating tremor. In the chicken, slow waves only appeared superimposed on eye tremors. The rat showed typical mammalian eye movement patterns.

Neck muscle recordings showed no change in the case of the birds; in the rat, during the activated phase of sleep, muscle tone did disappear.

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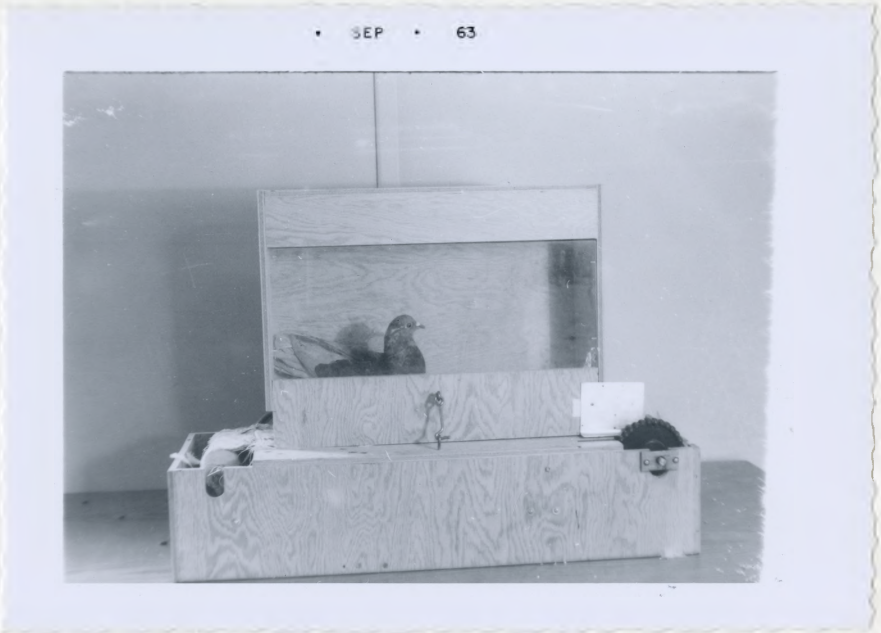
APPENDIX



Photograph of Treadmill Apparatus Used

In Experiment 2