INFORMATION TO USERS

This material was produced from a microfilm copy of the original document. While the most advanced technological means to photograph and reproduce this document have been used, the quality is heavily dependent upon the quality of the original submitted.

The following explanation of techniques is provided to help you understand markings or patterns which may appear on this reproduction.

1. The sign or "target" for pages apparently lacking from the document photographed is "Missing Page(s)". If it was possible to obtain the missing page(s) or section, they are spliced into the film along with adjacent pages. This may have necessitated cutting thru an image and duplicating adjacent pages to insure you complete continuity.

2. When an image on the film is obliterated with a large round black mark, it is an indication that the photographer suspected that the copy may have moved during exposure and thus cause a blurred image. You will find a good image of the page in the adjacent frame.

3. When a map, drawing or chart, etc., was part of the material being photographed the photographer followed a definite method in "sectioning" the material. It is customary to begin photoing at the upper left hand corner of a large sheet and to continue photoing from left to right in equal sections with a small overlap. If necessary, sectioning is continued again — beginning below the first row and continuing on until complete.

4. The majority of users indicate that the textual content is of greatest value, however, a somewhat higher quality reproduction could be made from "photographs" if essential to the understanding of the dissertation. Silver prints of "photographs" may be ordered at additional charge by writing the Order Department, giving the catalog number, title, author and specific pages you wish reproduced.

5. PLEASE NOTE: Some pages may have indistinct print. Filmed as received.

University Microfilms International
300 North Zeeb Road
Ann Arbor, Michigan 48106 USA
St. John's Road, Tyler's Green
High Wycombe, Bucks, England HP10 8HR

THE EFFECTS OF FOREBRAIN ABLATIONS ON SOME BEHAVIORS IN *XENOPUS LAEVIS*

by

LAWRENCE P. KUNSTADT

A dissertation submitted to the Graduate Faculty in Biology
in partial fulfillment of the requirements
for the degree of Doctor of Philosophy,
The City University of New York.

1977

Lawrence Kunstadt

This manuscript has been read and accepted for the Executive Committee in Biology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

August 11, 1977
Date

Leslie R. Aronson
Chairman of Examining Committee
Professor L. Aronson

Aug. 25, 1977
Date

Louis G. Moriber
Executive Officer
Professor L. G. Moriber

Max Hamburgh
Professor M. Hamburgh

City College
Institution

Ethel Tobach
Professor E. Tobach

American Museum of Natural History
Institution

Carol Diakow
Professor C. Diakow

Adelphi University
Institution

Walter Riss
Professor W. Riss

Downstate Medical Center
Institution

_____

_____
Institution

_____

_____
Institution

The City University of New York

ABSTRACT

In order to further elucidate the hypothesis that the telencephalon of ectothermic vertebrates functions primarily as a non-specific arousal mechanism, facilitating lower brain areas, (the Arousal Hypothesis) the South African clawed frog, Xenopus laevis, was studied in four behavioral situations before and after telencephalon ablation, olfactory bulb ablation or sham surgery. Particular attention was paid to measurements of the temporal aspects of the behaviors studied.

The behaviors studied were:

1. Habituation to an acoustic-vibratory stimulus
2. The optomotor response
3. Escape from shallow water
4. Feeding behavior

In the habituation experiment no changes were found in the number of responses per session, the strength of the responses or in the strength of the initial responses. Certain telencephalon-ablated individuals, however, showed a marked decrease in the number of responses to habituate which

was not seen in any of the animals in the other groups.

There were no changes in optomotor responses to moving vertical black and white stripes which were attributable to the operations.

In an experiment involving escape from shallow water into deeper water, there was a significant increase in the length of time to escape in the telencephalon ablated-group.

In the feeding experiment, it was found that ablations of the telencephalon eliminated feeding behavior.

It is concluded that 'arousal,' defined as that function regulating frequency and other temporal aspects of behavior (but not response strength) is the major function of the forebrain in Xenopus laevis. The telencephalon, however, does not play a major role in relatively simple behaviors.

ACKNOWLEDGEMENTS

I would like to express deep gratitude to the members of my committee: Drs. Carol Diakow, Max Hamburgh, Walter Riss, Ethel Tobach and my advisor, Dr. Lester Aronson, all of whom contributed to my intellectual development, both through conversations and through their published works.

Several members of the Department of Animal Behavior deserve special appreciation. Patricia Brunauer, especially, and Robert Stolberg aided me far beyond the expected call of duty. Lorraine Tucker and Ellice Kassorla did most of the histology. American Museum of Natural History volunteers Lynn Carey, Hollister Jameson and Jan Miers helped with some of the data.

The staff of the library of the American Museum were also of inestimable help. Mildred Bobrovich, Pam Haas and Janina Gertner deserve special mention.

Some of the animals were donated by Drs. Robert Goode and Darcy Kelley.

The study was supported in part by funds from the City University of New York Graduate Center and from Public Health Service training grant 1 T32 MH14280-01 to the Animal Behavior-Biopsychology Program of the City University of New York and the American Museum of Natural History. The tatoo machine was donated by Tony's Tatoo Parlor of Union Beach, New Jersey.

TABLE OF CONTENTS

LIST OF TABLES

ILLUSTRATIONS

PHOTOGRAPHS

FIGURE

I. INTRODUCTION

A. PURPOSE

A wide body of literature (reviewed by Aronson, 1970, Aronson and Nobie, 1945, and Kaplan and Aronson, 1969) substantiates the view that the telencephalon[1] of fishes, amphibians and reptiles may be removed without the loss of any behavior, given the proper stimulus. This fact led Aronson (1948, 1957, 1967, 1970) to propose that the telencephalon serves as a non-specific arousal mechanism that regulates the organization of behavioral processes, although it does not organize these processes itself. The anatomical substrate for the organization is presumed to lie mainly in the lower brainstem, particularly in the midbrain tegmentum. Removal of the telencephalon leads to a diminution in the frequency of those behaviors predominantly under the influence of internal processes, or, as stated classically, it decreases "spontaneity." It may also lead to changes in temporal aspects of behavior,

---

[1] In the literature on fishes, amphibians and reptiles the term forebrain has come to be equivalent to the term telencephalon. However, based on embryological considerations the forebrain (prosencephalon) should include the diencephalon as well as the telencephalon. In the present paper the term forebrain is meant not to include the diencephalon.

for example, in an increase of latency to respond to a stimulus. The effects of telencephalon ablation seem not to be specifically related to a given sensory modality but to range across behaviors involving any sensory modality.

In order to further study these aspects of the arousal hypothesis (AH) four behavioral situations were studied before and after telencephalic ablations in the South Africa clawed frog, Xenopus laevis. Particular attention was directed to measurements of the temporal changes in these behavioral situations. It was predicted, in general, that major telencephalic ablations would result in increased durations and decreased frequencies across all the behaviors, and that behaviors using different sensory modalities would not be affected differentially. These predictions were only partly validated.

B. EXPERIMENTAL DESIGN

The behaviors studied were (1) habituation to an acoustic-vibratory stimulus, (2) the optomotor response, (3) escape from shallow water and (4) feeding behavior. In the habituation experiment the animal was put in a large metal drum which was struck repeatedly by a metal bar, creating a banging sound. Typically, the subject first

responded with vigorous movements which would subsequently diminish in intensity. The optomotor test was conducted in a standard apparatus consisting of a cylinder with rotating walls covered with vertical black and white stripes. The frog was placed in the cylinder and as the walls slowly rotated the animal's head turned. The escape from shallow water experiment consisted of putting an animal in a shallow trough surrounded by deep water. Almost invariably the animal would, within a few minutes, escape into the deep water. The feeding experiment measured the animal's reaction to small pieces of food dropped into the tank.

There were three groups of experimental animals. In Group I the entire telencephalon was removed. In Group II the olfactory bulbs alone were ablated. Group III animals were sham operates with no incursion into neural tissue. All three groups were tested before and after the operations.

II. MATERIALS AND METHODS - GENERAL

A. THE EXPERIMENTAL ANIMAL

1. TAXONOMY

Superorder	Salientia
Order	Anura
Suborder	Xenoanura
Family	Pipidae
Subfamily	Xenopinae (three genera)
Genus	Xenopus (six species)
Species	laevis (six subspecies)
Subspecies	laevis

After Savage (1973) and Tinsley (1973, 1975).

Xenopus laevis belongs to the family Pipidae, the tongueless aquatic frogs. (See listing above.) Although having many characteristics in common with the other Pipidae, (Lynch, 1973), Xenopus has some unique traits: the atlas is separate from rather than fused to the second vertebra, the atlantal cotyles are juxtaposed, not separate, and the pectoral girdle is arciferal.

Although all animals are mosaics of primitive and specialized characters, Xenopus suffers from being both a primitive anuran and an aquatic one so that it is uncertain which of its "primitive" characters are truly primitive and which are neotenous due to selective pressure from its aquatic habitat (Deuchar, 1972, 1973; Patterson, 1939). For example, Poynton (1964) claimed that the presence of free ribs in larval Xenopus as well as in early Cretaceous pipids, in contrast to ankylosed ribs in extant adult anurans, indicate an early separation of Xenopus. In contrast, Jurgens (1971) reasoned that because "water-smelling" urodeles have simple nasal sacs whereas "air-smelling" urodeles have complex nasal sacs, the presence of complex nasal sacs in Xenopus laevis suggests that it reverted to an aquatic habitat late in its phylogeny, that is, after terrestrially-adapted nasal sacs had been established. Likewise, he showed that the anura, gymnophiona and terrestrial urodeles have an intermaxillary gland, which is absent in the aquatic urodeles, the Cryptobranchidae and in the aquatic Pipinae. Since its function is to make the tongue sticky, it facilitates the capture and swallowing of prey, clearly a superfluous function in an aquatic medium. The presence of the gland in Xenopus laevis

is thus also taken as evidence of a recent return to water. While it is not presently possible to resolve this problem, one is nonetheless safe in viewing Xenopus laevis as a primitive anuran adapted to an aquatic habitat the brain of which must reflect both of these conditions.

It is generally accepted that extant anurans and urodeles derived from crossopterygian fishes called rhipidistians, some 350 million years ago (Schaeffer, 1969; Szarski, 1962). There is some disagreement as to whether anurans derive from one rhipidistian stock (the Osteolepiforms) and urodeles from another (the Porolepiforms), (Jarvik, 1960), or whether they derive from the same stock (Jurgens, 1971; Schaeffer, 1965). The latter seems to be the prevalent contemporary view (Estes and Reig, 1973). Estes (1975b) believes that Xenopus evolved prior to 90 million years ago when Africa and South America were still part of the same continental mass.

2. LIFE HISTORY

Xenopus is totally aquatic, except during occasional overland migrations. It usually lives in still fresh water but can tolerate and even breed in

brackish salinities (Channing, 1976; Channing and van Dijk, 1976; Hewitt and Power, 1913) and can also be found, though rarely, in moving water (Channing, 1976a). Annual floods may carry adults and tadpoles to temporary pools where they remain (Channing, 1976b). They seem to be most active at night, when they tend to move to the edges of the pools. During the day, they are most often located in the deep center of the pools (Channing, 1976). When not feeding or breeding they remain concealed in mud, with just their eyes and forelimbs visible. If disturbed, or during periods when the pools dry up, they bury themselves in bottom mud (Channing, 1976a). They can apparently remain dormant under the mud over the winter (Kalk, 1960).

In its natural habitat Xenopus laevis has a wide-ranging, primarily carnivorous diet, including annelids, molluscs, crustaceans, arachnids, diplopods, many species of insects, small fish, other amphibia and even newly hatched birds, which, presumably, fall into the water (Inger and Marx, 1961). Dreyer (1913) found that Xenopus kept in the laboratory had stomachs full of algae.

Meyer and Wagener (1974) claimed to have individuals which lived 32-33 years in their Institute. A more reasonable estimate for the lifespan of this species in

the wild is 10-15 years (Deuchar, 1975).

3. DISTRIBUTION

Xenopus laevis is found in the temperate regions of southern and western Africa and in Malawi, Rhodesia and Mozambique (Deuchar, 1975; Power, 1929). It has also recently established itself in Southern California (Knefler and Mahrdt, 1972, 1973).

Fossil specimens of Xenopus have been found in early Cretaceous sites in Israel (Nevo, 1968), in Paleocene sites in Brazil (Estes, 1975a,b) and in Miocene and Recent sites in Africa (Vergnaud-Grazzini, in Estes, 1975b).

B. LABORATORY PROCEDURES

1. MAINTENANCE

The animals used were all adult males purchased from domestic suppliers. They were of unknown age and were not from inbred stocks. They were housed in groups of five in ten gallon aquaria kept in a room with south-facing windows. All the animals were kept for several months before being used in the experiments. They were fed a wet mash (see section on Feeding) consisting

of beef liver, pablum, shrimp, lettuce and spinach (Gordon, 1950). Identifying figures were tattooed on the ventra of all subjects.

2. OPERATIVE PROCEDURES

The animals were anaesthetized by immersion in 3% urethane until the righting response was lost, a procedure taking about ten minutes. They were then placed in a freezer for 10-12 minutes at about 0°C, a procedure developed by Riss (personal communication) to minimize bleeding and facilitate anaesthesia. Attempts to control bleeding with epinephrine-soaked cotton or with absorbable gelatin sponge were largely unsuccessful. Continuous aspiration provided some relief. The temporalis muscles overlying the frontoparietal bones were retracted and partially resected. The sagittal crest was shaved down with a dental drill and a hole was made over the olfactory bulb region with a dental trephine. Additional bone was removed manually with forceps. The appropriate neural tissue was aspirated with a glass pipette attached by tubing to a suction pump. In the case of the sham operates, the meninges were cut but the neural tissue was not damaged. The operations were performed under the low power of a binocular microscope. No packing material was used to fill the empty portion of the cranial cavity.

Eventually, when the animals were killed and autopsied, it was noted that connective tissue had resealed the section where bone had been removed. In addition, a closed superficial membrane was usually found around the previously open brain.

3. HISTOLOGICAL PROCEDURE

Transverse sections of the brains were made at 15 micra and were stained with gallocyanin according to the method of Einarson (Luna, 1968). Table 1 illustrates the degree of operative damage. There were four sham animals that sustained some damage. Two of them were not utilized in any of the data analysis and two (S-11 and S-12) were utilized only in the feeding experiment. The brains of the rest of the sham operates were verified as being undamaged. Due to technical reasons it was not possible to do a detailed histological analysis of some of the brains. In these cases data from the animals were assigned to operative groups based on photographs and written descriptions made at the time of autopsy prior to the histological workup.

III. HABITUATION EXPERIMENT

A. MATERIALS AND METHODS

The behavioral test consisted of describing the decrements in a startle response following a repetitive acoustic-vibratory stimulus. The animal was taken from its home tank and put into a 31 cc diameter aluminum drum with 45 cc high walls, containing 6000 cc of home tank water (photos 1 and 2). After a two-minute acclimation period, the trial was initiated by the onset of the stimulus, namely, the striking of the drum by an externally located solenoid bar (photo 3). Preliminary experiments demonstrated that if an animal did not respond (No Response) four consecutive times, it was unlikely that it would again respond to the stimulation. Sample sessions which were lengthened beyond six minutes had not indicated that if an animal had not habituated within 6 minutes, it would not do so even if given more time. Therefore an animal that reached the criterion of four consecutive "No Responses" was considered to be habituated; that is, the animal was considered to be refractory to further presentations of the stimulus. The drum was struck approximately every 4 seconds until the animal reached a criterion of four consecutive "No Responses" or until six minutes had elapsed.

After each session, the animal was returned to its home tank and a new animal was placed in the drum. The

water in the drum was discarded after the five animals from each tank had been tested.

Examination of the sound waves with a sonagraph (Sona-Graph, Kay Electric Company) demonstrated that the repetitive stimulus was producing a constant frequency configuration of sound.

Constant voltage for the solenoid was obtained by using a voltage regulator. While this tended to make the stimulus constant, because of mechanical reasons small variations in the stimulus intensity were not eliminated. (See appendix-1

The data were divided into three time periods: the first nine preoperative days, postoperative days one to nine and fourteen to twenty-two. The third period was chosen to look for recovery of function, and to make the time periods equivalent for all subjects. (See appendix-1).

Because the telencephalon-ablated animals did not eat (see section IV, Feeding Behavior) a group of intact animals which were fed were also run. They appear in table 2 as the "Starvation Controls." Therefore, there were four groups of animals, a telencephalon-ablated group, an olfactory bulb-ablated group, a sham-operated group and a starvation control. The raw data for the starvation controls are in appendix 1, tables and 2. Because there were no differences among the different

periods in this control group these animals are not included in the analyses beyond table 2.

B. RESULTS

1. CATEGORIES OF RESPONSES

In a typical response series the animal initially swims away rapidly for several seconds, followed by decreasing intensity of responses until no movement is seen. The responses were categorized as follows:

Very Strong:	The animals shows a "startle" movement followed by rapid and prolonged forward swimming.
Strong:	Any forward swimming not as prolonged as the Very Strong response.
Medium:	Although the torso may move a bit, there is no actual swimming. All four limbs move.
Weak:	Only two or sometimes three of the limbs move. There is no displacement of the torso.
Very Weak:	Only one limb or a part of one limb moves. This category includes small twitches of a single digit.
No Response:	There is no visible movement.
Air Response:	The frog's external nares are above the surface of the water when the stimulus is presented. The actual response, which might be a dive under the water, an attempt to climb out of the tank or one of the responses of the VS-VW continuum was not recorded.

Although the overall trend of responsivity for a given session was from Very Strong towards No Response, there were occasional reversals from one response level to a higher level, as is typical in habituation experiments (Goodman and Weinberger, 1973; Hinde, 1970). There were days in which the animal did not habituate.

On some days some animals showed an atypical response pattern consisting of two features: the animal spent a large amount of time at the surface (air or A-response) and it swam around seemingly unresponsive ("oblivious") to the stimulus. It did not show a clear response along the Very Strong-Very Weak continuum to each presentation of the stimulus. Although it would have been of value to classify sessions based on both aspects of the aberrant behavior, the qualitative nature of the "unresponsiveness" prevented doing so. Therefore the easily quantified A-responses were used as the measure of this pattern. Those days on which the animal showed five or more consecutive A-responses were classified as A-days. The criterion of five consecutive A-responses was chosen because there were many sessions in which an animal responded normally (within the Very Strong-Very Weak continuum) and also spent two or three consecutive trials at the surface and therefore five consecutive A-responses

(about 20 secs) was considered sufficiently greater than the few seconds at the surface needed to breath to consider it a separate category. A detailed analysis of A-days, habituated days and non-habituated days was done in order to determine whether data taken on A-days should be included in the data analysis (see appendix 1). The analysis justified including the data for all sessions for further statistical treatment.

2. NUMBER OF RESPONSES IN EACH SESSION

a. Analysis of Group Differences

Tables 1 and 2 in appendix 1 show the number of responses given by each animal. A Friedman two-way analysis of variance (Siegel, 1956) of all three periods showed a significant difference ($\chi_r^2 = 39.07$, $P < .05$) for the telencephalon-ablated group but not for the olfaatory bulb-ablated or sham-operated groups. A Friedman two-way ANOVA was not done for the starvation controls. A Wilcoxon's signed ranks test (Siegel, 1956) showed that there was a significant difference ($P < .05$, one-tailed) between preoperative sessions 1-9 and postoperative sessions 1-9 for the telencephalon-ablated group. No other group comparisons between different periods were statistically significant.

These findings made it difficult to make inferences

about the effects of the different procedures as there seemed to be no differences between the preoperative sessions and the second period of postoperative sessions. Accordingly, a one sample runs test over all twenty-seven sessions for each animal in all groups was made. No tendency for an overall decrease in number of responses over time was found ($P < .05$). (See appendix-1, table 3).

b. Responses of Individuals

A One-Sample Runs Test showed a significant difference between the observed and expected distributions of changes for animals T-9, T-10, O-1, O-2, O-8, S-3, S-7 and S-8 (see appendix-1, table 3). In all these cases the increase or decrease of number of responses from session to session changed direction more frequently than expected ($P < .05$, two-tailed). In all the other cases, even though there was no statistically significant change of direction the animals also tended towards high frequencies of change. These findings reinforce the conclusion that there was a high degree of variability in each animal's responsivity from session to session.

c. Summary

We conclude that there was no change in the number of responses per session.

3. QUANTITATIVE ANALYSIS OF RESPONSE CATEGORIES

a. Frequency

The median response frequencies for each category for each animal for each session is shown in appendix-1, table 4). A Wilcoxon signed ranks test was carried out for each animal and within each response category (that is, Very Strong, Strong, Medium, Weak, Very Weak, No Response and Air Response) to determine whether the surgical or other treatments affected the quality of the responses to the stimulus. Although some of the changes in categories of frequency were statistically significant (see table 3) these changes were found in almost all groups during all three periods. Accordingly, it was inferred that the experimental treatments did not affect the quality of responses to the stimulus. To verify these findings the following analyses were also made:

b. Modal Response Frequency in Each Category

Each category was given a numerical value (see appendix-1, table 5 for statistical analyses). A Friedman two-way ANOVA showed no significant differences ($P > .05$) among each group when all three periods were compared (Telencephalon-ablated animals: $\chi^2 = 36.39$, df = 26; Olfactory-bulb ablated animals: $\chi^2 = 38.34$, df = 26; Sham-operated animals: $\chi^2 = 29.62$, df = 26). Wilcoxon

tests showed no differences when medians of sessions involving the preoperative and first postoperative periods in each group were compared but showed statistically significant increases when the two postoperative periods were compared for each of the three groups (see table 4).

4. QUALITY OF INITIAL RESPONSES

A Friedman two-way ANOVA showed no significant differences in the initial responses either within or between time periods (see appendix-1, table 6). (Within preoperative sessions 1-9, χ_r^2 values are: Telencephalon-ablated group - 7.74; Olfactory-bulb ablated group - 4.61; Sham-operated group - 6.69. Within postoperative sessions 1-9, χ_r^2 values are: Telencephalon-ablated group - 5.73; Olfactory-bulb ablated group - 8.03; Sham-operated group - 5.99. Within postoperative sessions 14-22, χ_r^2 values are: Telencephalon-ablated group - 6.25; Olfactory-bulb ablated group - 4.16, Sham-operated group - 13.46. Degrees of freedom = 8 for within group comparisons between preoperative sessions 1-9 and postoperative sessions 1-9, χ_r^2 values are: Telencephalon-ablated group - 17.29; Olfactory-bulb ablated group - 15.41; Sham-operated group - 12.72. Between postoperative sessions 1-9 and 14-22, χ_r^2 values are: Telencephalon-ablated group - 19.77; Olfactory-bulb ablated group - 13.92; Sham-operated

group - 20.86. Between preoperative sessions 1-9 and postoperative sessions 14-22 χ^2_r values are: Telencephalon-ablated group - 18.77; Olfactory-bulb ablated group - 11.17; Sham-operated group - 20.08. Degrees of freedom = 17 for the between group comparisons.)

These findings suggest that there was no retention of the previous session's habituation. Inasmuch as frogs are "notoriously" poor at remembering (Aronson, 1970) this was not an unexpected finding.

C. DISCUSSION

1. INTRODUCTION

Habituation is a decrement in response to a repeated stimulus not attributable to receptor adaptation or muscle fatigue. Thompson and Spencer (1966) suggested nine criteria to serve as an operational definition of habituation: " 1. Given that a particular stimulus elicits a response, repeated application of the stimulus results in decreased response (habituation). The decrement is usually a negative exponential function of the number of stimulus presentations. 2. If the stimulus is withheld, the response tends to recover over time. 3. If repeated series of habituation and spontaneous recovery are given, habituation becomes successively more

rapid. 4. Other things being equal, the more rapid the frequency of stimulation, the more rapid and/or more pronounced is habituation. 5. The weaker the stimulus, the more rapid and/or more pronounced is habituation. 6. The effects of habituation training may proceed beyond the zero or asymptotic response level. 7. Habituation of response to a given stimulus exhibits stimulus generalization to other stimuli. 8. Presentation of another (usually strong) stimulus results in recovery of the habituated response (dishabituation). 9. Upon repeated application of the dishabituatory stimulus, the amount of dishabituation produced habituates." The first criterion was used in this experiment to define habituation.

2. THE PRESENT STUDY

Although the results discussed above lead to the conclusion that removal of the forebrain does not result in any changes in the habituation response, the loss of a specific behavioral component was observed in some cases: As defined previously the Very Strong response typically consisted of an initial "startle" response followed by prolonged forward swimming. In almost all cases the foreward swimming component was abolished by the forebrain ablations. Unfortunately data were not collected to substantiate this observation.

As a general conclusion it may be stated that neither telencephalon ablation nor olfactory bulbectomy resulted in a change in responsiveness to the acoustic-vibratory stimulus used in this experiment. It may be that investigation of the effects of varying the stimulus parameters (e.g., pattern, intensity) would indicate that a change in habituation phenomena had occurred as a result of some surgical procedures.

4. NEUROBIOLOGICAL CONSIDERATIONS

Although the results of our habituation experiment indicate th at loss of the telencephalon did not affect responsivity to an acoustic-vibratory stimulus, Farel's experiments (Farel, 1977, Farel et al., 1973) indicate that descending modulation may play a role in some types of habituation. In addition, Segura et al., (1971) found some evidence that descending reticular information inhibits clasping. Russel (1971) found that the efferent lateral line system inhibits lateral line input and that it fires only in response to active limb movements or to stimulation of descending reticular neurons. These findings suggest that a complex feedback system may be involved in the basic relex mechanism.

3. AMPHIBIAN STUDIES

Habituation studies on anurans have been limited to responses to visual (Birukow, 1939, 1951; Buntz-Kuenzer, 1957; Eikmanns,[1] 1955; Ewert, 1966, 1967a,c; Ewert and Birukow, 1965; Ewert and Harter, 1969; Ewert and Ingle, 1971; Ewert and Rehn, 1969) and tactile (Franzisket, 1963; Kimble and Ray, 1965) stimuli. The present study demonstrates habituation in, presumably, a third (lateral line system) and fourth (inner ear system) modality.

Ewert's laboratory (Ewert, 1966, 1967a,c,; Ewert and Birukow, 1965) has apparently done the only investigations into the effects of the loss of the telencephalon on habituation in amphibia. They studied habituation of a "turning-towards prey" response in Bufo bufo. They found that the greater the forebrain extirpation, the fewer the responses there are in a response series. Complete forebrain extirpation caused a complete loss of the turning-towards reaction. In contrast to this diminution of reactivity (facilitation of habituation), tectally damaged frogs did not habituate. If habituation is seen as a decrease in responsivity organized below the endbrain, then the endbrain could be hypothesized to prolong habituation series by its arousal function,

---

[1]Knowledge of the papers appearing in German is based on discussions in the English literature (Ewert, 1971; Ewert and Ingle, 1971; Goodman and Weinberger).

acting on these lower areas. Ewert (1967c) reported that intermittent tactile stimulation can reduce the rate of habituation of prey-catching activity. Ewert and Borchers (1971) found visual-tactile interactions in subtectal but not in tectal neurons, leading Ewert and Ingle (1971) to suggest that extratectal input modifies the habituation mechanism. They suggested the pretectal region as a possible locus of origin for this modulation.

IV. FEEDING BEHAVIOR

A. MATERIALS AND METHODS

Xenopus laevis normally live in small ponds and feeds on virtually any small animal, including its own tadpoles (Inger and Marx, 1961). In our laboratory the animals were fed on a wet mash which is a standard Museum fish food (Gordon, 1950). Early in the study this diet was occasionally supplemented by fresh beef liver and cod liver oil, although Gurdon (1967) claimed that vitamin supplements do not improve the health of these animals and the supplements were therefore discontinued.

Feeding consisted of dropping small pieces of the customary food (about 1cc) into the tanks by hand. When the animals were first introduced into the laboratory they did not feed by hand. Instead, after the food was dropped into the aquarium they became aroused and swam around eating the food pieces off the substrate. After several weeks they fed readily by hand, to the point that occasionally upon seeing the experimenter, or, especially, hearing the glass cover being removed, they would gather at the surface, making active feeding movements.

Feeding sessions were typically twice a week,

Tuesday, and Friday. The animals were not fed ad libitum because if they ate too much, a day or two after feeding they became bloated and floated at the surface. The feeding "experiment" consisted of normal feeding sessions during which observations on the manner of feeding and which animals fed were made.

The feeding responses were recorded with a pencil and paper.

B. RESULTS

1. DESCRIPTION OF QUALITATIVE RESPONSES

When observed in a tank of water, Xenopus typically hover suspended or lie immobile on the substrate except for occasional quick surfacings to gulp air. If food is introduced into the tank, two general categories of feeding behavior may be seen. One is a specific response to a piece of food falling through the water, which consists of orientation towards the food followed by visual tracking of it as it descends. The other is an "aroused food-searching" which enables the animal to locate food by randomly swimming about rather than moving towards a localized food source.

In the specific response, the animal orients towards

the general direction of the splash created by dropping the piece of food into the water. If the animal then sees the food descending through the water it will usually attempt to track it. If tracking is successful, ingestion usually follows, although the tracking is sometimes inaccurate and the animal misses the food or, in other cases, the frog tracks the food accurately but does not open its mouth. These behaviors have recently been studied quantitatively by Avila and Frye (1977).

The orientation towards the splash is probably mediated by the acoustic and/or lateral line systems (Görner, 1973). Tracking of the falling food piece is most likely mediated by the visual system, although one cannot yet discount the possibility that pressure waves are generated by the moving piece of food.

The other response consists of an active swimming about, with the forelimbs flicking towards the mouth, shoveling in any food encountered on the substrate (Hemmer and Köhler, 1975; Hutchison, 1965). This response is initiated by the detection of diffusing molecules from the food source. It may occur whether or not the animal has ingested food via the tracking behavior, as long as it detects the molecules from the food. It is almost certainly mediated by the olfactory and/or vomeronasal systems. To test whether

the food-searching behavior is mediated by the olfactory and/or vomeronasal systems, the olfactory and vomeronasal nerves of two otherwise intact animals were severed, resulting in the total abolition of the food-searching response, although not of the orientation and tracking responses. A non-operate and a sham operate control showed no deficit in the food-searching response. It should be noted that Onoda and Katsuki (1972) demonstrated that the lateral line organs of Xenopus respond electrophysiologically to chemical stimuli, including liver extract, although in our laboratory this stimulus was unable to evoke the food-searching behavior through the lateral line system alone. Ishi (personal communication to Onoda and Katsuki, 1972) found taste buds in the mouth of Xenopus laevis. Their function has not been investigated.

2. QUANTITATIVE RESPONSES

The results of the feeding experiment (table 5) show that in no case did a forebrainless animal successfully orient, track and ingest food, even after two months of recovery time. In contrast, all but one of the olfactory-bulb-ablated animals and all of the sham-oriented animals fed successfully, (although they did not show the "aroused food-searching" response) generally after only one or two weeks of postoperative recovery time. The olfactory bulb-ablated animals, however, did not show the "aroused

food-searching" behavior. When fed, intact animals almost always ate, both immediately and vigorously. Table 5 refers specifically to the orientation-tracking-ingestion sequence, although in the case of the forebrainless animals, there were no instances of food-searching either as was expected because the removal of the forebrain includes removal of the olfactory and vomeronasal systems also. The forebrainless animals, as a rule, showed no response to the food at all, even if it fell well within the visual field. Schrader (1887), Loeser (1905) and Burnett (1912) (all cited in Aronson and Noble (1945)) reported that decerebrate frogs readily snapped at food. Ingle (1971) reported that the toad is capable of a vigorous feeding response without a telencephalon. In contrast, Aronson and Noble (1945) and Ewert (1967c) reported that forebrainless frogs and toads, respectively, fail to catch prey. Without more detailed descriptions of the deficits found by the different investigators, it is not possible to explain these contradictory findings in the present study.

C. DISCUSSION

1. HYPOTHESES

The forebrainless Xenopus, while not responsive to the visual stimulus, however, are not blind, In a pilot

experiment ablated animals showed no signs of blindness in a light-dark preference maze; neither did they in the optomotor apparatus. These findings raise the following question : Why did the loss of the telencephalon lead to the loss of a specific deficit in visually-mediated behavior (to food only) rather than to a general deficit in vision?

There are several possible explanations: A. Specific Visual Deficit Hypothesis. There may be a specific visual deficit for small vertically moving objects. B. Psychic Blindness Hypothesis. The animals are "psychically blind" or "visually agnostic." The term psychically blind was used by Klüver and Bucy (1937) to describe the behavior of a monkey which had had a bilateral temporal lobectomy in which no impairment in visual acuity or visual localization was found; yet "the monkey seems to be unable to recognize objects by the sense of sight." The use of the term was expanded by Terzian and Ore (1955) to include a clinical case in which an adolescent epileptic boy, after bilateral temporal lobectomy, was able to recognize his parents as individuals, but, apparently attached no psychological meaning to them. In the context of the present experiment, the term may be applicable, but with qualifications. The frog's relatively low level of psychological organization is obviously

different from that of the primates. The frog may see the moving piece of food but the subsequent neural processes which associate the sight of the food with the memory of "food" or with whatever physiological and experiential processes mean "food" to the frog may be absent.
C. Telencephalic Visual Area Hypothesis. The visual input may normally stimulate a primary visual area within the tectum, which, via the thalamus, stimulates telencephalic "visual" areas, which then project to lower motor areas. The absence of the telencephalon would then uncouple this system. D. Arousal Hypothesis. If the visual input reaches areas also receiving input from the telencephalon, such as the midbrain motor areas, there may be an uncoupling between these systems. That is, the telencephalon may regulate the response threshold of competing neural systems within the motor areas. This possibility is related to an aspect of the arousal hypothesis.

Although it is not possible to conclusively assert the validity of one of these hypotheses on the bases of current knowledge, the arousal hypothesis seems to be the most defensible explanation of the data because it is compatible with the data obtained in the present experiment while requiring fewer assumptions than the other hypotheses. A review of neurobiological considerations

may contribute to a resolution of this issue.

2. NEUROBIOLOGICAL CONSIDERATIONS

a. Specific Visual Deficit Hypothesis. Although this hypothesis is neither supported nor contradicted by the available evidence, it is testable. Tectal cells that respond to small vertically moving objects would have to be identified electrophysiologically. It would then be possible to see if loss of the telencephalon changes the firing characteristics of this population.

b. Psychic Blindness Hypothesis. This hypothesis is attractive because of its simplicity and its implication that the amygdaloid region of the frog may play a role similar to that of the amygdaloid region in primates. The absence of knowledge about amydaloid function in frogs makes it difficult to evaluate. To test its validity, the role of the amygdaloid region of the frog should be investigated. A bilateral amygdaloidectomy would have to be performed. The results would be interpreted in light of the Klüver-Bucy syndrome. For example, the Klüver-Bucy syndrome includes the loss of psychologically important learning. The following experiment would be instructive: When frogs were first introduced into the laboratory, they did not readily show the components of the orientation-tracking behavior. Instead of directed

approach responses, they would swim about wildly (a "kinesis" response). After several weeks they readily swam toward the surface when food was introduced. As soon as the glass covering was removed, the frogs showed feeding orientation and sometimes feeding movements of the forelimbs and mouth, suggesting a conditioned response. If amygdaloid lesions abolished this conditioned response response, further research could be carried out to test this hypothesis.

c. Telencephalic Visual Area Hypothesis. The evidence that the telencephalon receives indirect visual projections is relatively recent and still somewhat tentative. To evaluate this evidence it is necessary to discuss the relevant aspects of the frog's visual system in some detail. (See figure 1). The visual system of the frog has the same "bauplan" as does that of other vertebrates studied (Ebbesson, 1970; Riss and Jakway, 1970; and Szekeley, 1971). Retinofugal projections have been found to terminate in a) the nucleus of the accessory optic tract (the basal optic nucleus), (Fite and Scalia, 1976); b) the dorsal thalamus (neuropil of Bellonci, posterior thalamic "nucleus" and geniculate "body" proper); (Scalia and Fite, 1974; Scalia and Gregory, 1970); c) the pretectal region (large-celled pretectal nucleus and uncinate pretectal nucleus) (Scalia and Fite, 1974) and d) the tectum (Maturana,

Lettvin, McCulloch and Pitts, 1960; Scalia, Knapp, Halpern and Riss, 1968). Projections to the hypothalamus, which have been identified in many other vertebrate forms, (Kunstadt, 1969) have not been found in anurans.

d. The Arousal Hypothesis. This hypothesis states that midbrain or lower motor areas which receive input from tectal cells responding to small vertically moving objects, also receive input from the telencephalon. It is known that the tectum sends a large efferent bundle to the interpenduncular nucleus and medial reticular formation of the midbrain tegmentum and to the ventral and intermediate gray areas of the rostral spinal cord (Rubinson, 1968). The telencephalon also sends projections to the midbrain tegmentum (Halpern, 1972) and it has been claimed that fibers even reach the medulla and rostral spinal cord (Kokoros, 1974). If these tectal and telencephalic projections terminate on the same brainstem motor areas, which is likely, the removal of telencephalic modulation would presumably decrease excitability to visual stimuli, in this case, to small vertically moving objects. This idea is consistent with the arousal hypothesis.

Of the four areas of projection, two are not of immediate interest. The nucleus of the accessory tract appears to be involved in the optomotor response and will be discussed in Section V and the pretectal region

has been implicated in the inhibition of tectally-mediated approach responses. While it may thus play an indirect role in allowing the initiation of the feeding response, it is not germane to the question at hand.

It has been known for some time (Maturana, Lettvin, McCulloch and Pitts, 1960) that the retina projects to the tectum. It is now known also (Fite and Scalia, 1976; Scalia and Fite, 1974) that the retina projects both directly and indirectly, via the tectum, to the dorsal thalamus. I would like to hypothesize that the dorsal thalamus actually is a waystation to the telencephalon of the two largely separate anatomical systems, that is: a retino-tecto-posterolatero-striatal system and a retino-geniculo-posterocentro-medial cortical system, each of which is a mediator of distinct types of visual behavior. (See figure 1). The dorsal thalamus contains two posterior nuclei, the posterolateral nucleus and the posterocentral nucleus. Scalia and Colman (1975) demonstrated that the posterolateral nucleus projects to the striatum and that the posterocentral nucleus projects to the medial cortex ("primordial hippocampus"). The discovery of these pathways suggests the existence of retino-tecto-posterolatero-striatal and retino-geniculo-posterocentro-medial cortical circuits, each of which may subserve distinct visual functions,

Based on several lines of evidence, discussed below, I hypothesize that the retino-tecto-posterolatero-striatal pathway mediates responsiveness to small moving objects, and is probably directly involved in the feeding response, and that the retino-geniculo-posterocentro-medial cortical pathway mediates wavelength and/or flux detection and is not directly involved in the feeding response. The evidence in favor of this idea comes from experiments which show that the tectum is involved in small-moving object detection and that the alternative pathway is not.

It is well established that the tectum responds to the movement of small objects across the visual field (Ewert, 1967b, 1970a,b; Ingle, 1973; Lettvin, et al., 1959; Maturana, et al., 1960). There is as yet no electrophysiological evidence that the next link in the hypothesized circuit, the tecto-posterolateral pathway, carries information about small objects to the thalamus. This suggestion is based solely on the demonstration of its anatomical existence (Scalia and Colman, 1975) and the knowledge that the striatum, but not the medial cortex or septum, responds to small moving objects (Gruberg and Ambros, 1974). No retinotopic projection was found by Gruberg and Ambros in the striatal area.

Evidence that the retino-geniculo-posterocentro-dorsomedial cortex pathway is involved in flux and wavelength detection is stronger than that for the function of the alternative pathway and by its strength it argues by exclusion that it is not involved directly in the detection of small moving objects.

Muntz (1962a,b) first demonstrated that cells in the dorsal anterior thalamus respond to specific wavelengths of light, particularly that color humans perceive as blue, but not to moving contours or spots of light. Kicliter (1973a) suggested that the area involved included the lateral geniculate, the nucleus rotundus and the ventrolateral area. Fite and Scalia (1976) found units in the lateral geniculate nucleus which respond to the onset of light; and Kicliter (1973a) demonstrated that lesions of the dorsal anterior thalamus did not diminish movement detection but abolished wavelength discrimination. However, Kicliter (1973b) also found that the dorsal anterior thalamus projects to the striatum, a finding which contradicts the suggested model.

There is additional general evidence of links between the thalamus and the telencephalon (Karamian, et al., 1966; Vesselkin, et al., 1971) but these studies specify neither precise anatomical loci nor do they use stimuli presented to the eye, using rather

direct stimulation of the nervous system. It is thus difficult to integrate them into the sort of exact functional anatomy discussed here.

Assuming that the functional and anatomical pathways of this model (figure 1) exist, the question remains as to whether or not the model can explain the findings of the feeding experiment. Inasmuch as both the lateral and medial telencephalic walls were removed in most cases, the "visual" areas (striatum and dorsomedial area) of both pathways were removed. Yet there was a deficit in only the food-tracking behavior. This would certainly argue against the model suggested in figure 1. It should be stressed, however, that non-olfactory ascending systems to the telencephalon do exist and their functions need to be investigated.

e. In summary, then, loss of the telencephalon leads to a total loss of feeding. The tracking component seems to be almost totally abolished. Although this could be due to any one of several factors, the current experiments suggest that the animals are not blind but that the stimulus is too fast for the animals to utilize the sensory information it provides. Both Noble (1936) and Ribbink (1972) demonstrated th at forebrainless cichlids could not pursue or school with other fish that changed direction rapidly, and Shaw and Sherman (1971) found that

forebrainless cichlids showed a deficit in reversing direction in an optomotor apparatus. To understand the effects of loss of the telencephalon on the visual system many parameters of the visual system would have to be varied.

V. OPTOMOTOR EXPERIMENT

A. MATERIALS AND METHODS

The optomotor apparatus (see Photo 4) consisted of a central, transparent stationary cylinder 15 cm high which was inside a rotating drum having 1.3 cm wide alternating black and white vertical stripes. The central axis of the drum was 9175 cm from the stripes. The outer drum was driven by an electric motor connected to a motor speed control. A wide spectrum (Durolite Plantlite) light bulb was in a position 40 cm above the water level. A clockwise rotation took 45 seconds and a counterclockwise rotation took only 33-37 seconds. Each session consisted of four three-minute trials, always in the order clockwise, counterclockwise, clockwise, counterclockwise. The initial trial was two minutes and 56 seconds; the other trials were three minutes each.

The animal was introduced into the inner drum in about 1185 cc (40 oz) of home-tank water, which provided a depth of 7.75 cm. There was a ten minute acclimation period. Ten degree angles were marked on the white bottom of the drum with differently colored pencils (see Photo 4).

The following measurements were taken:

1. Head Following

a. Latency

This is the number of seconds between initial onset of or change in direction of drum movement and detectable head movement of the frog.

b. Number of Ten Degree Passages

This is the number of ten degree arcs traversed by the tip of the frog's snout during head following.

c. Duration of Ten Degree Passages

This is the number of seconds the animal took to traverse a ten degree arc with its snout.

2. "Stops"

a. Number

This category is self-explanatory

b. Duration

A "Stop" is defined as any period of ninety seconds or more during which the animal traverses less than ten degrees. These almost always occurred after the animal head-followed and its head came to rest at its forelimbs.

3. Surface Behavior

a. Duration

"Surface" behavior is similar to the "Air Response"

of the habituation test. It is defined as the time during which the frog's nares were above the surface of the water. Typically, though not invariably, the animal would continue to follow the moving stripes. Occasionally it would dive and surface repeatedly or attempt to climb out of the inner drum. No attempt was made to record the specific response the animal was making. This category is simply the number of seconds the animal spent in Surface behavior.

4. Whole Body Movements

a. Number

Whole body movements (WBM's) are described above. In general it was easy to distinguish a WBM from head-following. However, it was often difficult to distinguish one WBM from the one that followed, since the animal often swam erratically back andforth. This difficulty was multiplied many-fold in scoring the duration of WBM's.

b. Duration

This is the number of seconds of a WBM.

c. Direction

The WBM's were scored according to their initial direction relative to the turning drum: Same- in the same direction as the drum movement; Opposite- in the opposite direction of the drum movement; Up-Down- in a vertical up and down movement showing no initial direction relative to drum movement.

An Esterline-Angus event recorder was used to record the animal's responses. Keys on the event recorder were pressed:

a) At the onset of the drum-turning, the change-of-direction of the drum turning and the termination of the session.

b) When the frog's head started to move in response to drum-turning. It was kept pressed until the frog's snout passed a ten-degree line in which case another key was pressed to record the subsequent head-following. As the animal passed several ten-degree lines during a trial these two keys were pressed alternately. If the drum changed direction during a ten-dgree passage, the key was not released.

c) For the duration of a whole-body movement.

d) For the duration of surface behavior.

"Latency" was computed taking the difference between the onset of the drum movement or drum change-of-direction and the onset of head following.

The number of ten-degree movements was computed directly by counting the number of ten-degree durations on the recording paper.

The animal's head was initially found positioned between the forelimbs in its typical posture. As it followed the drum, the head reached the forelimb in the direction of turning and then it stopped; it only rarely pushed the limb farther than its initial position unless the trunk of the body flexed with it. The head often appeared to move fastest when it was about midway between the forelimbs and slowest when it was near a forelimb. The animal typically remained in the stopped position until the drum changed directions, at which point the animal would start, after a short latency, to follow in the new direction. There were no cases in which the animal did not follow the moving stripes.

After following the drum for some time, the animal often moved its whole body rather than just its head. These whole body movements (WBM's) were usually not drum-following movements. The frogs limbs flayed about and the animal looked as though it were breaking out from a rigid following pattern into a wild swimming movement. They were very quick jerky movements and may be consider to be reactions to stress, perhaps escape attempts.

The duration of ten-degree passages, duration of "stops", duration of surface behavior, and number and duration of WBM's were counted directly off the recording paper.

The direct of WBM's was counted off pencil and paper data sheets.

There were three groups of animals: a telencephalon-ablated group, an olfactory bulb-ablated group and a sham-operated group.

B. RESULTS

1. CATEGORIES OF RESPONSES

a. General Considerations

When the animal was placed in the drum it usually swam around a bit. It then typically oriented more-or-less along a radius, that is, at right angles to and facing the stripes. Upon the onset of drum rotation or change in direction there was about a 2-7 second latency (see appendix 2 table 1) until head following by the animal. At the slow drum speed used in this study, usually only head-following occurred, although at faster speeds movement of the whole body took place. A satisfactory way of monitoring eye movement was not found so that it is not known if the eyes show following movements.

b. Experimental Results

Of the nine measures taken all but one (direction of WBM's) involued temporal aspects of behavior. The data are presented in appendix 2 tables 1-8.

1) Clockwise vs. Counterclockwise Measures

Data for each of the nine measures had originally been taken separately for the clockwise and counterclockwise trials. A Friedman Two-way ANOVA was done between the clockwise and counterclockwise trials of the measures (except the Up-Down responses, which were too few to allow a meaningful comparison). No significant differences ($p > .05$) were found. (χ_r^2 values were: Latency - T=5.70, O = 0.20, S = 1.20; Number of ten-degree passages - T = 6.38, O = 0.70; S = 4.05; Duration of ten-degree passages - T = 3.08, O = 1.30, S = 1.05; Number of "stops" - T = 0.90, O = 1.70, S = 1.95; Duration of "stops" - T = 3.68, O = 4.90, S = 5.40; Duration of surface behavior - T = 0.38, O = 1.50, S = 5.40; Number of WBM's - T = 1.20, O = 3.60, S = 4.50; Initial directions, "Same" - T = 0.30, O = 1.80, S = 4.80; Initial directions, "Opposites" - T = 0.90, O = 3.40, S = 0.60). The clockwise and and counterclockwise trials were therefore pooled for all further calculations.

2) Summary of Results

A Kruskal-Wallis one-way ANOVA among the three operative groups for both the preoperative and the post-operative periods showed no significant differences ($p > .05$) for any of the measures. (H values for the preoperative and postoperative periods, respectively, were: Latency - 1.18, 1.3; Number of ten-degree movements - 0.28, 0.42; Duration of ten-degree movements - 2.5, 2,5; Duration of "stops" - 1.6, 0.56; Number of "stops" - 0.28, 2,9; Duration of surface behavior - 1.39, 1.99; Number of WBM's - 0.38, 1.08; Duration of WBM's - 4.43, 0.55; Initial directions: Same - 0.38, 0.71; Opposite - 1.44, 0.16; Up-Down - --, 2.0).

Because of the small number of subjects it was not possible to do a paired comparison of the preoperative and postoperative periods. However, it may be seen in appendix 2, tables 2 and 3 that there was a dramatic change in animals T-17 and O-12. Both of these animals showed a large increase in the number of ten-degree movements coupled with a decrease in duration of ten-degree movements. These changes were probably due to the experimenter's having resected too much of the cranial musculature during surgery. These animals had some difficulty in normal head-following and tended to move

their trunks more than did normal animals, causing them to move farther more quickly.

Other than these changes, which were not attributable to the brain lesions, there were no noticeable changes in behavior. As a general conclusion it can be stated that neither telencephalic nor olfactory bulb ablations had a significant effect on the optomotor response.

C. DISCUSSION

There are a few studies on the effects of telencephalic lesions on the optomotor response in amphibians and reptiles. Hertzler and Hayes (1967, 1969) demonstrated the optomotor response in turtles and found no significant deficit in "cortically" lesioned subjects. The earliest report of the effects of telencephalectomy on the optomotor response in frogs was that of Blankenagel (1931, in Birukow, 1937). Working with Rana temporaria he claimed that the cerebrum is responsible for all optomotor responses. In contrast, Diebschlag (1935, in Birukow, 1937), Birukow (1937) and Lazar (1973), using species of Rana, all found that removal of the telencephalon in no way affected the optomotor response.

The results of the present study corroborate the latter view. The optomotor response was studied in detail.

The arousal hypothesis led us to suspect that ablation of the telencephalon would cause a change in some aspect of the timing or frequency of the response. Because Xenopus does not, as a rule, show a true nystagmus as do the Ranid species, there was no a priori reason to suspect that the same motor mechanisms would be involved and hence, that the same results would be obtained. However, detailed investigations on these nine measures of responsivity, involving aspects of timing and frequency, showed no changes that could be attributable to lesions of the telencephalon.

The probable reason for this finding was advanced by Lázár (1973). Utilizing selective lesioning, he found that loss of the tectum or diencephalon or surgical isolation of the telencephalon led to no diminution of the animals' ability to exhibit the optomotor response. In contrast, lesions of the accessory optic tract or rostral midbrain tegmentum abolished the optomotor response. These results strongly

implicate the accessory optic tract-basal optic nucleus system in the mediation of this response in Rana esculenta. They obviously suggest that the same system is involved in Xenopus. It is also of interest to note that a recent study by Brauth and Karten (1977) found that the nucleus ectomammillaris of pigeons, which is considered homologous to the basal optic nucleus of frogs, projects to those parts of the cerebellum involved in the optomotor response.

Burgers (1950) measured the response of intact Xenopus to different shades of gray and various colors in an optomotor apparatus. He found that Xenopus is very sensitive to small differences of light intensity. It may be that the effect of the telencephalon is to modulate a threshold to respond based on light intensity, stripe width, rotational speed or some other parameter and the reason that some experiments find no change in response is that they use supraliminal stimuli. It should also be mentioned that while detailed knowledge of projections to and from the basal optic nucleus are not known, the telencephalon does have a strong projection to the general vicinity of this nucleus.

VI. ESCAPE FROM SHALLOW WATER

During the course of maintaining the animals it was noticed that when the home tank water was siphoned down in order to change it the animals became very agitated when the water level reached 3-5 cm (1-2 inches). This finding led us to postulate that the animals can somehow detect the depth of the water, and that they show a preference for deep rather than shallow water. An experiment was then devised in which the animals were given the opportunity to escape from shallow water into deep water. Indeed, when given the opportunity to do so, the animals almost always escaped into deep water within five minutes.

A. MATERIALS AND METHODS

A 14 by 17.5 cm clear plastic trough with 2.5 cm high walls was built and placed on a brick in a 20 gallon tank such that the water level in the trough was only 2 cm (see photo 5). When an animal was placed in the trough and given the opportunity to escape from the shallow water, it almost always did so within five minutes.

After a fifteen minute acclimation period in the deep water the animal was netted and placed in the trough. Typically, the animal remained still for a few seconds and then moved about intermittently until it

escaped. Timing of the trial started when the animal was out of the net and in the trough and ended when the animal's hindlimbs passed over the side of the trough as it escaped or when five minutes elapsed without a successful escape. There were five such trials in each session, with a two minute intertrial interval. If the animal did not escape after five minutes, the trial was scored as No Escape and the animal was netted and put into the deep water for the two minute intertrial interval. The number of seconds the animal swam around "attempting" to escape was also scored.

All but two of the animals were run for 9 sessions preoperatively. Animals T-15 and T-16 were run for 27 and 26 sessions preoperatively, respectively, to see if the extended number of sessions would facilitate the escape response (see section 2b). A stopwatch and an electric digital timer which were mechanically connected so that they would start at the same time were used to time the trials and the behaviors. The stopwatch was left on until the animal escaped or 6 minutes elapsed. The electric timer was on only while the animal was swimming. Data were recorded with pencil and paper after each trial.

There were three groups of animals: a telencephalon-ablated group, an olfactory bulb-ablated group and a sham-operated group.

B. RESULTS

1. DESCRIPTION OF QUALITATIVE RESPONSES

Typically, after the animal was placed in the shallow trough, there was a period of a few seconds during which the animal did not move, which was followed by a period of active escape activity consisting of short swimming bouts interspersed with short periods of relative inactivity. Escape consisted of swimming to the side of the trough and raising the head above the side but these behaviors alone were not sufficient for the frog to escape. At times the animal swam to the side and put its head over the side but did not jump over.

2. QUANTITATIVE RESULTS

a. Latency to Escape

There was a significant increase in the latency to escape between the first six preoperative (median = 37 secs, range = 18-193.5) and first six postoperative (median = 282 secs, range = 138.5-300) sessions for the forebrain ablated group ($P < .05$, Wilcoxon, two-tailed). (See appendix-3, table 1). There was no change in the olfactory or sham groups (see appendix-3, table 1 B, C). Although on many trials the forebrain-ablated postoperative animals did not escape, in no case was the ability to escape lost,

as is evidenced by the fact that all the postoperative animals escaped on at least one occasion.

Although the animals had different numbers of preoperative sessions (see section 2c) there was no apparent relation between the number of preoperative sessions and postoperative behavior.

b. Efficiency of Escape

The percentage of time spent swimming prior to escape was calculated for each trial as a measure of efficiency of escape behavior. (See appendix-3, table 2). That is, an animal with a four minute escape time could have obtained that duration by remaining motionless for three minutes and fifty-five seconds and then swimming directly to the edge and jumping out or it might have swum around continuously for four minutes without escaping until the end. Because of the small sample size, the telencephalon-ablated and sham preoperative groups could not be compared directly with their postoperative groups. However, a Kruskal-Wallis one-way ANOVA among both the preoperative and post-operative groups showed no significant differences ($P > .05$). Preop $H = 3.02$, Postop $H = 1.46$. A paired comparison of the olfactory bulb-ablated preoperative and postoperative scores also showed no significant difference ($P > .05$, Wilcoxon, two-tailed).

These analyses suggest that there was no change in the relative amount of swimming after the operations. However, two of the three telencephalon-ablated animals (T-13 and T-14) show an obvious large decrease. (Table appendix-3, table 2). This was attributable to an increase in the latency before swimming started, rather than a decrease in the amount of swimming time. The median amount of time spent swimming was 14 seconds preoperatively and 5 seconds postoperatively for animal T-13, and 12 seconds preoperatively and 26 seconds postoperatively for animal T-14. Although the latencies to escape of these two animals were increased dramatically by the forebrain ablations (see appendix-3, table 1A) the time spent swimming remained relatively short, indicating that while it took a long time for the animal to start moving, once it did so it escaped relatively rapidly. That is, the efficiency of escape was not changed by loss of the forebrain but the latency to initiate a successful escape was increased in these two animals. This may reflect a change in the threshold of response to the situation or in the stimulus related to the escape response.

c. Improvement of Performance

Two of the animals (T-15 and T-16) were run for seven months preoperatively (appendix-3, table 3). Animal

T-15 was given 27 sessions, animal T-16 was given 26 sessions. A Wilcoxon test ($P < .05$ one-tailed) demonstrated that the time to escape for both animals decreased significantly between the first six and last six pre-operative sessions. Although this precluded the use of the later sessions in pooling the intragroup data, it is instructive to note that the animals did show an improvement in performance due, apparently, to experience in the apparatus alone. Frogs are very difficult to train (Aronson, 1970). Indeed, the literature on learning in amphibians is comprised mainly of negative findings. It may be simply that the animals need very long periods of time in which to show changes. There is also some evidence that some of the forebrain-ablated and olfactory-bulb ablated animals showed a trend towards postoperative recovery. This needs to be investigated as a phenomenon in its own right.

C. DISCUSSION

The fact that the telencephalon-ablated group showed an increased time to escape while in no case losing the ability to do so (see appendix-3, table 1) after the operation supports the arousal hypothesis, as discussed by Aronson (1970): " . . . Damage to, or complete removal of the forebrain results in a quantitative reduction in the frequency of specific behavioral patterns and <u>sometimes</u>

changes the timing of these events. . . . No behavioral sequences are completely eliminated by the operation. . ." (Italics mine.) The apparent decrease in percentage of time spent before escape in two of the telencephalon-ablated animals partially defines the commonly found loss first labelled "spontaneity" by Flourens in 1824 (Aronson, 1970). "Loss of spontaneity was characterized by a failure to move or respond unless strong stimulation was applied. When a forebrainless frog was aroused, the resulting behavior was, in most cases, similar to that of an intact animal." However, this finding, that two of the three telencephalon-ablated animals were very efficient in escaping once they started to swim, raises an interesting theoretical point. Up until now, the arousal hypothesis had, in general, dealt with responsiveness to a changing stimulus. The present finding suggests that removal of the telencephalon also effects responsiveness to a tonic stimulus. However, it is not certain what the stimulus in this experiment is. The animals may be detecting the depth of the water or they may be making a postural adjustment to a changing stimulus such as the drying of that part of the skin which is above water. Although the latter may be a contributory stimulus the former is more likely because completely submerged animals respond by escaping from shallow water also.

Another confounding factor is the procedure used in this experiment. The animal is placed in the shallow trough by netting it from the deep water. One could argue that this procedure arouses the animal and acts as the strong stimulus. However, the fact that a netted animal in deep water does notuusually swim around after release from the net, and that it usually does not swim around after escape from the shallow water into the deep water, suggests that the netting process, while possibly arousing the animal, is not the immediate stimulus for escape.

VII. GENERAL DISCUSSION

A. INTRODUCTION - FOREBRAIN FUNCTION AND THE AROUSAL HYPOTHESIS

In C. Judson Herrick's 1921 article, "A Sketch of the Origin of the Cerebral Hemispheres," the phylogenetic advancement of the forebrain was envisaged as a progressive emancipation from olfactory domination. This process involved primarily the elaboration and specialization of ascending non-olfactory pathways, which were seen as projecting into wide areas of the hemispheres in all vertebrantes. In "The Functions of the Olfactory Parts of the Cerebral Cortex," Herrick's 1933 paper, he stressed the inability of the olfactory system to localize the source of scent, in contrast to the ability of most other sensory systems to localize their sources of stimulation. The olfactory system, he theorized, does not function in simple reflexes but works conjointly with other sensory systems. Reiterating his earlier anatomical viewpoint, Herrick saw the central connections of the olfactory nerves in all vertebrates as being "diffuse, widely dispersed and interconnected with one another and with other sensory systems in an intricate web of correlation fibers." To

him, these arrangements suggested that "exteroceptive olfactory excitations serve chiefly as activators of complex sensorimotor systems whose pattern of performance is determined primarily by other senses with sharper localization both physiologically and anatomically. The histological structure of the olfactory centers favors both irradiation and summation or intensification and it provides no recognizable apparatus adapted for the preservation of sharply defined local patterns of excitation such as is so characteristic of optic pathways and centers." In concord with his earlier idea that the pallium evolved by an increase in non-olfactory ascending systems, not by an elaboration of any olfactory field, he saw this ever present olfactory activation as acting on the expanding pallial systems. He saw the three pallial fields as receiving thalamic projections, as projecting to the midbrain and as interacting with each other via assocation fibers. "The net result of the total cortical activity would probably be in the main a differential inhibition or reinforcement of subcortical adjustments already in process with a minimum of either analytic or synthetic specificity within the cortex itself. In other words, this primitive cortex would tend to act as a whole to influence responses to external stimulations whose specific pattern is determined subcortically."

Herrick's ideas were based almost exclusively on neuronanatomical considerations. He acknowledged the absence of physiological and behavioral studies on these questions. The first major study on fish which saw the forebrain in the context suggested by Herrick was that of Janzen in 1933, which conceptualized the forebrain's role as mediating "initiative." The first major study on amphibians deriving from this line of thought was Aronson and Noble's paper (1945),"The Sexual Behavior of Anura: Neural mechanisms controlling mating in the male leopard frog Rana pipiens."

Two points emerge from this paper which are particularly relevant to the present discussion. One is that an extensive review of the literature on forebrain extirpation in Anura from Robert Boyle's work in 1663 to the date of the paper shows that forebrain ablations caused a decrease in "spontaneity," defined as a "failure to move or respond unless strong stimulation is applied." The other point is that Aronson and Noble's study found that removal of the forebrain except for the preoptic area or of the preoptic area alone did not interfere with male reproductive behavior although removal of both loci markedly reduced the tendency of males to pursue and attempt to clasp estrous females. The clasp response, warning croak,

sex call, spawning movements, release and swimming movements were all seen to be organized below the forebrain but facilitated by it.

In The Brain of the Tiger Salamander (1948) Herrick continued his earlier theme and cited the Aronson-Noble study as confirming his hypothesis and suggested that more studies of this sort be done.

Many have been done. Since that time many attempts have been made to elucidate forebrain function in ectothermic vertebrates. These studies have generally centered around two themes. One is that "the amphibian brain closely resembles the generalized prototype from which the higher vertebrates" evolved. This view has been expressed by such neuroanatomists as Herrick, Kuhlenbeck, Ariens-Kappers, Huber and Crosby and Papez (Aronson, 1970). The other is the concept that the forebrain functions as a non-specific activator of lower brain areas, a concept now labelled the arousal hypothesis. This view, with its implied understanding that "arousal" mediates various behaviors, is based on the anatomical view that first saw a hypothalamico-preoptico-septo-hippocampal circuit as a unitary system whose loci are homologous to the same-named structures in mammals. In a 1968 paper on the teleostean forebrain Aronson wrote, "It is evident that [surgical] invasions of the

forebrain often result in the decline or change, but not the elimination of certain behavioral patterns. . . . One should recall that the major fiber tracts of the forebrain form a descending system running caudally to the diencephalon and midbrain, which suggest a dynamic influence of the forebrain on lower centers. Only a relatively few small tracts run in the opposite direction." Referring to fishes, amphibians and reptiles Aronson (1970) wrote, "It is evident that damage to, or complete removal of, the forebrain usually results in a quantitative reduction in the frequency of specific behavioral patterns and sometimes changes in these events. Since it is evident that no behavioral sequences (including those generated by learning experiments) are completely eliminated by the operation, one must conclude that the behavior in question is organized (or the conditioned connections established) in neural centers below the forebrain." In the 1968 paper Aronson also wrote, "The concept of arousal is very general, and has been used to explain a multitude of situations where specific either-or, cause and effect relationships do not seem to apply. Herrick (1948) viewed arousal as a diffuse, nonspecific function, but to maintain its usefulness we must now begin to think in more definite terms."

The present experiment was designed to elucidate

the role of the telencephalon in Xenopus laevis, the South African clawed frog, as a means of expanding and clarifying the arousal hypothesis. As will be seen, the arousal hypothesis while in general supported by the results of the present experiment, is not adequate to explain their complexity. While the general form of the hypothesis accurately describes the function of the forebrain in ectothermic vertebrates, some modifications of its details must be made. An attempt will be made to refine the hypothesis so that it can adequately account for the results of the present experiment as well as the previous relevant literature.

B. COMPARATIVE STUDIES --REVIEW OF THE LITERATURE SINCE 1970

A major study of the role of the forebrain in relation to behavior in ectothermic vertebrates was published by Aronson in 1970. There have been several relevant papers published since then, most of which report experiments on learning in fishes. The present section will review all those papers which appeared subsequent to Aronson's review.

The word 'telenx' will mean 'telencephalon ablated'

and the word 'olfacx' will mean 'olfactory bulb ablated.' The categories used in regard to Fishes are based, with one exception, on Flood, Overmier and Savage (1976).

1. FISHES

a. Behaviors Other Than Learning

Savage (1971) found that electrophysiological stimulation of different telencephalic areas of the goldfish resulted in arousal reactions but not in specific behavior patterns. The lowest thresholds were in the posterior and central dorsal areas. Demski and Knigge (1971) stimulated various telencephalic areas of the bluegill and were able to obtain specific behavioral components, such as the "sweeping" of gravel which is part of nestbuilding. This finding is potentially crucial to the arousal hypothesis. If it were shown that the area stimulated, the central dorsal area, actually organized the behavior, this would disprove the arousal hypothesis. Lesioning of this area or stimulation of pathways leading from it would illuminate this issue. Davis, Kassel and Schwagmeyer (1976) and Kassel, Davis and Schwagmeyer (1976) found that telencephalic ablations in the teleost Macropodus opercularis blocked reproductive behavior as evidenced by the elimination of nest building and mating. Because the telenx males

interacted continuously with intact females in the normal fashion, they suggest that the telencephalon plays a specific role in this behavior. Although a possible mechanism is not proposed they imply one by raising a very important issue, which is that hormonal changes may occur as a result of telencephalic ablations. Noble (1939) and Aronson and Noble (1945) considered, respectively, the possible affects of telencephalic ablations on hormonal levels and the effects of hormones on telencephalic functioning. Segura, et al., (1971) and Colombo and Segura (1972) actually studied the effects of gonadal hormones on the EEG of toads and found that castration of females caused cessation of alpha activity while estradiol treatment was correlated with a return of the alpha rhythm although the estradiol-injected animals were not castrated. Recordings were made over the forebrain. While consideration of the whole question of hormonal-nervous system interaction is probably not crucial for learning studies, it certainly deserves more consideration, especially in studies of species-typical behaviors, such as reproductive behaviors.

Shapiro, Schuckman, Sussman and Tucker (1974) found that telencephalic ablations result in a decreased response frequency but not in decreased response strength of the gill cover extension response in Siamese fighting fish. Furthermore, over a period of three weeks the intact animals decreased their responsivity (which they incorrectly call habituation) whereas telencephalic animals start with a low response level and continue at this level. They interpret their results in terms of arousal. It should be cautioned that this experiment may contain several methodological errors.

Peeke, Peeke and Williston (1972) demonstrated that habituated levels of a biting response in the goldfish are not retained from day to day after forebrain ablation while they are retained in sham and non-operates. Animals were tested on five consecutive days starting after the operations. On the first day the forebrain ablated animals responded at the same level and habituated at the same rate as did the control groups. On subsequent days the forebrain ablated group showed initial levels of responding commensurate with the initial level of the first day whereas the control groups initial levels for days two to five was equivalent to the habituated level for the preceding day. They interpret those results

to mean that there is a specific deficit in memory of the previous day's habituation in the forebrainless fish although there is no deficit due to loss of inhibitory processes (presumably underlying the within session habituation) or to activating processes (presumably underlying initial responsivity).

b. Learning Studies

(1) Experiments Labelled Classical Conditioning

Overmier and Curnow (1969) studied classical conditioning to a shock UCS and red light CS and found, as had others, that there is no deficit attributable to forebrain ablations. Curnow and Overmier (1968) demonstrated that telenx goldfish acquired a classical conditioning task (USC = shock, SC = red light) as well as controls.

(2) Instrumental Training with Positive Reinforcement

Savage and Swingland (1969) reported two experiments. In the first, goldfish could obtain food by swimming through a photocell beam. Training started postoperatively. The intact animals were highly successful whereas the telenx animals were not. However, the telenx animals recovered to the normal level after about a month. In the second experiment the fish had to make a discrimination of a horizontal vs. a vertical rectangle in order to receive a food reward. The telenx animals did almost as well as the

controls if reinforcement was immediate. If the delay were five seconds the telenx animals did poorly. Savage and Swingland take these results to mean that the telencephalon does not affect memory traces but affects associability between the memory (horizontal vs. vertical) leading to the operant and the reward.

Flood (1975) corroborated the first part of this conclusion by reporting that both normal and telenx goldfish retained a learned food-reinforcement visual discrimination equally well over an eight day practice free period. She concludes that long-term memory is not affected by forebrain ablations.

Flood and Overmier (1971) found that telenx animals acquired a food-reinforced response as well as did intact animals as measured by number of responses to acquisition and latency from onset cue. The onset cue they used was the raising of a vertical door. It is possible, based on the results of Lazar (1973) and our results in the optomotor experiment that the detection of the movement of the horizontal line which was the bottom of the door is mediated by the same anatomical pathway, the basal optic tract, that mediates the optokinetic response in frogs and hence, is not related to telencephalic function. If this is the case, one would not expect a change in latency. On the other hand Shaw and Sherman (1971) found deficits in the optomotor response

in forebrainless Tilapia. It would clarify the meaning of the Flood and Overmier (1971) experiment if the response of forebrainless goldfish to moving horizontal lines were studied. Frank, Flood and Overmier (1972) investigated reversal learning, a process previously unexamined in this field. They taught goldfish to go down the lit arm of a Y-maze to receive food. The correct arm was then reversed. Both telenx and olfactory tract sectioned controls learned the tasks; however, the telenx group reached a somewhat lower criterion and was markedly inferior in learning reversals. They interpret their results as suggesting that the forebrain is not necessary for learning an appetitive task but that it facilitates this learning by inhibiting previously acquired responses. They interpret the finding that there is no difference in "between group latency to respond" as evidence against the arousal hypothesis.

(3) Conditioned Avoidance Experiments

Dewsbury and Bernstein (1969) stated that in previous conditioned avoidance studies telenx fish had been handicapped by the fact that they do not readily swim through narrow channels and that they take a long time to execute avoidance behaviors. Using an improved experimental design, they demonstrated that telenx goldfish

show decreased activity levels, as had been shown before, and performed as well as normal fish which contradicted the findings of the experiments using narrow channels and short CS-USC intervals. In addition, removal of the telencephalon after training resulted in only partial savings. They concluded that the telencephalon is not only not necessary for classical conditioning but it is not necessary for avoidance conditioning if the experimental parameters are appropriate. This qualification led them to suggest that, in contrast to the arousal hypothesis, the telencephalon transmits information from the external world to the lower nervous system, selecting stimuli from the environment and integrating this information so that appropriate behavioral patterns, organized elsewhere in the brain, are emitted under appropriate sets of stimulus conditions.

It is difficult to see how their results support it since the forebrainless fish were able to utilize information gained during the experiment

Overmier and Flood (1969) found that telenx goldfish were inferior compared to normals in a passive avoidance task. They first trained the fish to swim down a rectangular alley to receive food. Both normal and operated fish learned this task equally well. The fish were then given trials on which they were shocked if they swam into the

chamber that had previously been the reward chamber. Telenx fish did not learn to avoid the shock. They continued to swim down the alley as they had done when it led to being fed. The authors interpreted this to mean that the telencephalon functions to inhibit previously learned responses.

2. AMPHIBIANS

There have been several studies on the amphibian forebrain during the past decade, most of which have emanated from Ewert's laboratory. Of particular relevance is one study in which telencephalon ablation in the toad Bufo bufo decreases the frequency of "turning towards prey" responses in a general positive correlation with the quantity of forebrain removed. Complete forebrain extirpation led to complete loss of this response. Inasmuch as the prey were moving insect larvae Ewert's results raise the same issues as did our feeding experiment. It should also be noted that the animals were tested the day after the operation.

Hoffman and Lico (1972a,b) found that stimulation of the toad's septum resulted in variation in arterial blood pressure, pulmonary respiration, vocalization and movements. The motor responses consisted of asymmetrical

movements of all four limbs, limited to the stimulus duration. Stimulation of the dorsomedial pallium resulted in some increase of arterial blood pressure, shortlasting midriasis, an increase in amplitude and frequency of throat oscillations and phasic bilateral asymmetric limb movements. Stimulation of the amygdala resulted in approximately the same effects. No changes were observed after stimulating the olfactory bulbs, striatum or dorsal pallium. Their experiments demonstrate that certain telencephalic areas are capable of stimulating autonomic responses. The absence of an effect, particularly of a motor effect, after stimulation of the striatum is puzzling. A particularly interesting finding was that stimulation of the same area could result in either sympathetic or parasympathetic-type responses, depending on the nature of the stimulus parameters, although the sympathetic type predominated. This finding suggested to them that another area, perhaps the hypothalamus, is involved. They conclude that the telencephalon exerts a "rather stereotyped modulation over autonomic and motor behavior." They do not believe that it plays an integrative role. This belief may stem from the fact that they investigated simple physiological responses, not complex behaviors, as will be discussed later. Schmajuk and Segura (1971) studied the role of various brain areas in reproductive behavior of the toad and concluded that the

pallium is the locus of "decisions" regarding whether or not to perform a behavior, the septum "programs" the pallium and the limbic system underlies "basic motivations" involving the autonomic nervous system.

Segura and de Castello (1969) studied classical conditioning in the toad and found, as had previous workers, no loss attributable to removal of the forebrain. On the other hand, they found that avoidance conditioning could not be re-established after the operation, a finding also corroborating previous work.

3. REPTILES

Schapiro and Goodman (1969) demonstrated that a .20-.60 ma stimulus in the dorsolateral corpus striatum of alligators caused ipsilateral locomotor movements of the head, body, limbs and tail resulting in a continuous circling in the direction of the side of the electrode. Weaker stimulation caused head movements alone whereas stimulation slightly stronger than that producing head movements evoked autonomic responses. They summarize their results as meaning that the dorsal pallium may have an influence on motor function restricted to the head region and that stimulation of the corpus striatum evokes a "preformed organization of behavior" which is

"flexible, goal-directed and modifiable by the external environment."

Stimulation of the amygdaloid complex in the caiman by Keating, Kormann and Horel (1970) resulted in withdrawal responses consisting of turning 90-180 degrees followed by pedalling away from the side of the electrode. If a wooden barrier were placed in the animal's path it would circumvent the barrier. Stimulation of the striatum resulted in head movements and autonomic changes, as Schapiro and Goodman (1969) had reported for the alligator. Keating et al., suggested that "the function of the amygdala is to discriminate on the basis of sensory information the appropriate orientation the animal should make toward a stimulus, either approach or avoidance, and to regulate the appropriate intensity of this orientation. The striatum then guides the animal through the sensorimetor adjustments required by the environment to carry out the response."

Sikharulidze (1971) compared the effects of removing the forebrain and cerebellum with the effects of removing the cerebellum alone in Caspian and pond turtles. Whereas no loss was found after cerebellectomy in a visual appetitive task or in a classical conditioning experiment, removal of the forebrain in addition to the cerebellum caused a deficit in the visual appetitive task and did not prevent

a partial recovery of the conditioned responses.

Greenberg (1977) found that paleostriatal lesions decreased the frequency of arousal and challenge displays in Anolis carolenensis. His detailed findings have not yet been published.

Bass, Pritz and Northcutt (1973) demonstrated that forebrainless turtles could relearn a horizontal-vertical discrimination in the same amount of time it took them to learn it preoperatively.

In a major conference on neural mechanisms in turtle behavior there were only two papers relating to forbrain function. One, that of Hall (1972) discussed visual input to the telencephalon. In a more directly relevant paper, Morlock (1972) reported that ablation of the dorsal cortex resulted in no change of activity or in latency to eat in an aquarium with no obstacles (open field situation) or in a simple appetitive learning situation.

4. ANALYSIS OF A REVIEW OF THE ROLE OF THE TELEOST TELENCEPHALON IN LEARNING

A major review of the role of the teleost telencephalon in learning by Flood, Overmier and Savage has recently appeared (1976). These authors take the arousal hypothesis (AH) to task for its apparent inability to explain certain

experimental findings. It is thus valuable to examine their criticisms in detail.

Learning experiments are classified by them according to three general experimental paradigms: a) classical conditioning, in which the presentation of a signal and reinforcement are independent of the animal's behavior, b) instrumental conditioning in which the presentation of reinforcement or reward is contingent upon the animal's behavior, and c) conditioned avoidance training, in which the omission of an aversive event is contingent upon the animal's behavior. These three paradigms may be considered hierarchically organized with respect to one another, classical conditioning being the simplest and avoidance training being the most complex. In general, the more complex the training the greater is the effect of forebrain ablations. Several experiments have shown that classical conditioning is not diminished by loss of the forebrain, although not all parameters of conditioning have been measured. In instrumental training using food deprivation and reinforcement simple paradigms such as go/no go discriminations do not seem to be impaired by telencephalic ablations, while more complex tasks that offer more opportunity for alternative responses show deficits in acquisition, although not in final performance level.

Forebrainless animals seem to be particularly unable to utilize reinforcement if there is a time delay between performance and reward. Flood, Overmier, and Savage also point out that very few studies using food as reinforcement have included olfactory bulb or tract controls. In instrumental training using shock, that is to say, escape training, forebrainless fish "show the normal activity response to shock,the normal increased reactivity to shock over repeated exposures and escape latencies during avoidance training that are similar to [those of] normal fish." Detailed studies of escape alone are not available.

The third paradigm, avoidance training, shows the largest effects of telencephalic ablations. "Ablation reliably interferes with all types of avoidance learning regardless of the task demands. . . The impairing effect of telencephalon ablation on acquisition of avoidance behavior is of greater magnitude, persistence and reliability than any other behavior studied."

Flood, Overmier and Savage consider five major hypotheses to explain the available data. As they point out, the telencephalon must have more than one function and the hypotheses discussed often address themselves to less

than the total outlook. The arousal hypothesis, in our opinion, although criticized for its generality, finds its strength in its attempt to include the results of all the available experiments, not just those involving learning paradigms. More specific hypotheses attempting to explain the results of specific learning paradigms must be evaluated on their own merits. When enough hypotheses about specific functions turn into accepted explanations, the arousal hypothesis may fall by the wayside. Until then it seems to be the most parsimonious attempt to come to grips with most of the phenomena found, although it certainly has its shortcomings, as will be discussed.

Flood, Overmier and Savage's first criticism of the arousal hypothesis is that it cannot explain the lack of loss of "behavioral output" found in classical conditioning, pseudo-conditioning, sensitization and simple appetitive instrumental tasks. They challenge Aronson to spell out the rationale for asserting that simple learning tasks should not be as affected by telencephalic ablations as are more complex tasks. In their 1969 paper Kaplan and Aronson suggested that acquisition of more complex tasks are interfered with because they involve higher levels of integrative processes for which the forebrain is necessary. "During the learning of a conditioned avoidance

response, many levels of conditioning also take place, e.g., autonomic conditioning, as well as conditioning to incidental stimuli that occurs prior to onset of the US. In addition, second or third order conditioning may take place . . . Control subjects appeared to have formed a 'gestalt' of the situation. . . . It seems probable that the decline in arousal is mainly in the more complex associative (integrative) functions of the brain." As can be seen, Aronson's concept of the forebrain function includes integration, in the sense of utilizing or putting together diverse aspects of sensory information or motor responses. As Peeke, Peeke and Williston (1972) suggested, the forebrain is not "necessarily either the storage location of the engram or, if that were the case, the only storage location." They hypothesize that "forebrain structures, either separately or in concert, exert tonic or modulating influences on the consolidation of memory" and the engram is stored in some lower structure(s). Farel (1971a,b; 1974a,b), indeed, has demonstrated that one locus of habituation in the frog is the spinal cord. It is consistent with Aronson's ideas that the processes underlying simple learning tasks may be located predominantly in lower brainstem or spinal areas, perhaps with minimal influence from the forebrain, while more complex learning tasks,

involving higher levels of psychological integration are more involved with forebrain function.

Flood, Overmier and Savage's second criticism of the arousal hypothesis is that it cannot explain instances of increased "behavioral output" after telencephalon ablation. This criticism seems to be based on a misunderstanding of what the classical arousal hypothesis stated. Herrick (1933) wrote, "The net result of the total cortical activity would probably lie in the main in a differential inhibition or reinforcement of subcortical adjustments already in process with either a minimum of either analytic or synthetic specificity within the cortex itself." (Underlining ours). Kaplan and Aronson (1969) wrote, "We view arousal as a non-specific, non-directive effect on a variety of neural processes, sensory, integrative and motor. These effects may be excitatory or inhibitory. Although in most experiments, forebrain deprivation appears to result in a decrease in arousal, yielding sluggish, less-reactive subjects, forebrainless fish may also become overreactive and even violent." Arousal includes both excitatory and inhibitory processes. Certain behaviors may increase in frequency after forebrain ablation due to either a direct loss of inhibition or a loss of

facilitation of competing behaviors.

The third criticism by Flood, Overmier and Savage of the arousal hypothesis is based on Segaar (1960, 1961), and Segaar and Nieuwenhuy's (1963) findings (also discussed in Segaar, 1965) that telencephalic ablation in sticklebacks disrupts the organization and sequencing of reproduction. While these findings could be, in part, accounted for by an assumption of differential facilitation of competing responses, we believe that evidence is accumulating that the forebrain is involved in integrative processes and we take this as a valid addition to the known functions of the forebrain. Segaar's studies, although not reported in detail, found that forebrainless sticklebacks showed components of nest-building behavior in different parts of the tank at different times but did not exhibit the entire sequence in an integrated pattern. While the details of this integrative process probably relate to complex aspects of sensorimotor feedback systems, at this stage of investigation integration may be a sufficient explanatory construct.

Their fourth criticism of the arousal hypothesis also comes from Segaar's studies. He had found that localized lesions in different telencephalic areas caused differential effects of various behaviors. Contemporary neuroanatomical evidence indeed (Halpern, 1972; Heimer, 1969; Riss, Haltern and Scalia, 1969) suggests thatolfactory projections to

learning task. This is because either excitatory or inhibitory lower centers may be facilitated and these may influence behaviors which are either compatible or incompatible with that required in a task. Thus, the arousal hypothesis can explain anything, but predict nothing a priori.

This has always been the major criticism of the arousal hypothesis and in the sense that it says the arousal hypothesis cannot predict specific changes in behavior it is entirely valid. To make specific predictions we would have to know how the brainstem and spinal cord organize various behaviors, which clearly we do not. Another way of wording this criticism is to say that the arousal hypothesis is not, in theory, falsifiable with respect to changes in specific behaviors, and thus fails to meet the criterion of a scientific statement (Popper, 1965). However, until a particular behavior is shown to have been eliminated because it is organized by the telencephalon or until a particular sensory modality is shown to have been lost because of telencephalon removal or until the engram, whatever complex processes it comprises, is shown to be located in the telencephalon, the arousal hypothesis remain defensible.

and efferent projections from the telencephalon comprise considerably more discrete tracts and terminal areas than previously thought. These findings suggest that the telencephalon does not function "as a whole." Demski and Knigge's (see page 64) study suggests the same thing. An interpretation of the results of our habituation and feeding experiments based on discrete functional forebrain areas is not satisfactory. Ewert's finding (see page 22) that loss of a turning-towards prey behavior is proportional to the extent of telencephalic damage does not support the discrete area hypothesis either. Clearly behavioral experiments have not, in general, been sensitive enough to detect changes due to discrete lesions. It is suggested that a good starting point for such studies would be to contrast the relations to behavior of those structures contributing to the medial forebrain bundle with the relations to behavior of those structures contributing to the lateral forebrain bundle.

A final criticism of the arousal hypothesis by Flood, Overmier and Savage is based on logical grounds. "It cannot predict the effect of telencephalic ablation on any species-specific behavior or on the acquisition, asymptotic performance or extinction of any particular

C. DISCUSSION OF THE PRESENT EXPERIMENTS

1. DEFINITIONS

The term "arousal" has recently been discussed by Andrew (1974) who found five distinct meanings for it in the literature. In the present context, two are applicable. One is that "arousal" is defined operationally by the temporal and frequency characteristics of behavior. We agree with Andrew that the intensity of a response should not fall within the confines of the narrow definition we seek. The second relevant meaning is arousal as a brain mechanism. In most of the studies reported, the brain mechanism is inferred from changes in behavior after telencephalon ablations.

The term "arousal" is usually taken to mean activation as "reflected in an increasing behavioral activity of an organism that previously had been relatively quiescent or even lethargic" (Pfaffman, et al., 1977). This meaning is usually associated with the phrase "nonspecific arousal function" as used in reference to the mammalian reticular activating system (RAS). In many ways the lower vertebrates forebrain is seen to function analogously to the mammalian RAS. However, an often neglected but salient feature of the forebrain arousal hypothesis, as well as the mammalian system, is that "arousal" includes

both facilitation and inhibition, although a facilitatory function is found more frequently. The inclusion of an inhibitory function as part of the arousal hypothesis is often forgotten by its critics. The essence of the AH is that the telencephalon modulates or regulates brainstem and perhaps spinal motor mechanisms which are intrinsically organized. Whether it does this by facilitation, as is usually the case, or inhibition or some combination of the two is not the major issue. It would undoubtedly have led to less confusion had the AH been named the Regulator or Modulator Hypothesis.

The terms "organize" and "integrate" must also be defined, inasmuch as the former is specifically excluded by the AH as a forebrain function and the latter is viewed equivocably. An organizing function, in this context, would determine the spatial aspects of a behavior. The underlying mechanisms are undoubtedly open to variations caused by experience, maturation, hormonal effects and the like and are themselves probably complex interactions of the peripheral and central nervous system or of the stimulus and the nervous system, but they are seen, as a whole, to direct a pattern of spatial movement. By virtue of the fact that most studies find that removal of the telencephalon does not

abolish any behavior, it is said not to have an organizing function. Most likely, however, when the appropriate physiological studies are done it will be shown that complex interactions occur between the forebrain and lower systems. The question of the role of the thalamus has been largely neglected, although Ewert and Ingle (in Fite, 1976) have made inroads into its role in the amphibian visual system.

"Integration" in the context of the AH refers to the putting together of behavioral components, organized below the forebrain, into a meaningful sequence. The arousal hypothesis has been equivocal about whether or not that is a function of the forebrain.

2. THE PRESENT EXPERIMENTS

a. Forebrain Function and Motor Processes

The results of the Escape and Optomotor experiments most clearly demonstrate that there was no motor impairment attributable to loss of the telencephalon. In the Escape experiment, once the animal started to swim it escaped rapidly, without apparent difficulty. In the optomotor experiment, there was no change in behavior after the operation. The results of the feeding experiment are difficult to interpret with reference to motor function. Inasmuch as the forebrain-ablated animals generally did

not respond to moving food, and as it is not known what systems relate to this loss it is not possible to ascribe the change to either sensory or motor deficits, although either is possible. On rare occasions when an animal responded to the food, it was with an abbreviated movement, for example by moving forward somewhat without opening the mouth. While this might be a motor deficit, normal animals would, on occasion, move this way also. It is thus not possible to assert one way or the other that motor deficits were present.

The habituation experiment clearly demonstrated a motor loss in some animals. It was observed that the forward-swimming component of the Very Strong response was abolished (see definition of Very Strong, p 13). This finding apparently contradicted the prevalent view that "the forebrain plays no direct role in the maintenance of posture, equilibrium or locomotion" (Kaplan and Aronson, 1969), although two other points must be considered. One is that it is not known whether this motor loss is attributable to its being organized in the forebrain or whether it is because the behavior is a "highly aroused" one. The fact that it occurs at the highest level of a response strength continuum and the fact that some recovery is seen after two months argues in favor of viewing the loss as a function of arousal and not as a loss of the underlying organization of the behavior.

b. Forebrain Function and Sensory Processes

Other than the loss of the "aroused" food-searching behavior in the feeding response, which is also lost by severing the olfactory and vomeronasal nerves alone, there is no direct evidence that ablation of the telencephalon resulted in sensory loss. The optomotor experiment demonstrated no loss of the ability to respond to moving vertical stripes. The habituation experiment demonstrated no loss of hearing or lateral line sense. On the contrary, if we interpret the meaning of the air-responses correctly, the animal continued to raise its head above the water postoperatively as a means of minimizing the acoustic-vibratory input. The escape experiment demonstrated that the animals were able to escape; inasmuch as the stimulus is unknown it is not possible to correlate it with the findings. The feeding experiment was somewhat ambiguous on this point. If an animal does not respond, to what is the absence of behavior attributed? Although it is possible that there was a visual deficit, on rare occasions there was a response, however abbreviated, suggesting that the animals did see the stimulus. Results from a light-dark preference experiment, not given in detail here, show no loss of a preference for the dark after forebrain ablations.

c. Forebrain Function and Integrative Processes

The Arousal Hypothesis deals uncertainly with integration as a forebrain function. Neither Herrick nor Aronson states specifically that integration is a telencephalic function. Kaplan and Aronson (1969) viewed the arousal function of the forebrain as capable of acting on integrative processes. Although it is not clear how a distinction between integration itself and a function acting as an integrative process might be distinguished, especially in the psychologically primitive ectothermic vertebrates in which integrative processes are modest compared to those of mammals, the role of the forebrain in mediating various levels of behavioral integration must be considered. In the optomotor experiment, head-following may, tentatively, be seen as a lower level of behavioral integration than the whole-body-movements. In the habituation experiment, the decremental responses from Very Strong to Very Weak may be seen as a lower level of behavioral integration than the Air-responses in which the animal swam around unresponsive to the stimulus. And yet there was no significant decrease of either whole-body movements or Air responses after loss of the forebrain, suggesting that even if they are relatively high levels of behavioral processes the telencephalon is not the major locus for their underlying physiological processes.

The general finding, however, that the more complex the learning task the greater is the role of the telencephalon and Segaar's (1965) finding that loss of the forebrain disrupted behavioral sequences in sticklebacks lend support to the view that higher levels of behavioral integration are integrated at higher anatomical levels, in this case the telencephalon. This idea is not unlike the concept of hierarchical organization proposed in the last century by Hughlings Jackson (1958) and more recently by Riss (1968) and Riss, Pedersen, Jakway and Ware (1972).

The arousal hypothesis may, indeed, be in error in not having explicitly attributed an integrative function to the forebrain with respect to complex behaviors. This had not been tested for directly.

3. HYPOTHESES OF FOREBRAIN FUNCTION

There have been many recent suggestions as to what the forebrain of lower vertebrates does. They seem to fall into five general categories: arousal, learning, integration, coordination of internally-detected states with ambient conditions and mediation of specific behaviors. Each will now be considered.

a. Arousal. The arousal hypothesis has a long history. It is the predominant thesis against which newer ideas are devised. Originating with Herrick (1922,23) who formulated it based solely on anatomical evidence, it was expanded and developed by Aronson (1963, 1970, Aronson and Herbeman, 1960; Aronson and Kaplan, 1963, 1965, 1968; Aronson and Noble, 1945; Kamrin and Aronson, 1955; Kaplan and Aronson, 1967, 1969) who emphasized the findings of behavioral studies and later by Herrick (1948) who also expanded his earlier views. As the experimental literature grew it was refined and amended. In our opinion, it is presently best able to account for the widest range of experimental findings. Arousal should be seen as the major role of the forebrain, with the many qualifications discussed in this section.

b. Learning. The learning studies have been outlined in a previous section. They comprise the bulk of the recent literature. We view the theories of forebrain function in learning, particularly the dual-process mediational theory proposed by Flood, Overmier and Savage (1976) as applicable to a restricted field of behavior, as even they note, and not as models for forebrain function in general. The dual-process mediational theory is, in fact, strongly supported by the available

evidence and may likely be confirmed. We suggest that support for this theory, which posits that the telencephalon of teleosts is involved in the utilization of conditioned motivational states as secondary reinforcers, may come from a detailed analysis of the role of the telencephalon in autonomic function, rather than analysis of constructs such as "conditioned fear" or "incentive motivation." The function of the telencephalon in the utilization of secondary or tertiary cues was predicted by Kaplan and Aronson (1969, page 196, section 4).

c. Integration. Integration has two meanings in the context of putative forebrain function. One is the putting together of separate behaviors into a sequence. This is viewed as a complex, "higher" function than, for example, a mere reflex response. Support for the view that the forebrain of certain species has this function comes mainly from Segaar's (1960, 1961, 1965) findings that forebrainless male sticklebacks show the components of nestbuilding behavior but that these behaviors are displaced spatially and are not sequentially meaningful. We take this function as a major possible amendation of the arousal hypothesis, although it should be emphasized that Segaar's results are not reported in great detail and stand alone in the literature.

d. Coordination of Internal State and External Conditions. The second meaning of the concept "integration" in the present context is the detection and coordination of the internal state of the animal with the animal's ambient conditions. This role has been suggested for the mammalian (and other) limbic systems by several authors in very different contexts (McGowan, Hankins and Garcia, 1972; Riss, Halpern and Scalia, 1969).

Ribbink (1972) suggested that one function of the teleost forebrain is the "conversion of the endogenous and exogenous sensory input into a behavioral output." We consider this function a serious possibility, although it must be seen in a larger anatomical context than just the telencephalon. The two telencephalic afferent systems and the telencephalon's connections with the hypothalamus must also be considered. The largest input to the telencephalon is from the olfactory bulb. As recent reviews have emphasized (Alberts, 1974; Cain, 1974; Wenzel, 1974) the olfactory bulb of mammals and birds is not merely a sensory structure -- it also plays an activating role in the mediation of many behaviors not directly utilizing olfaction. This is an extremely important consideration which has not been studied in the lower vertebrate forebrain literature. As Flood, Overmier and Savage (1976) point out, very few studies have even

included olfactory controls. The ascending systems have also been neglected. Recent studies on sharks have found input to the thalamus from the retina (Ebbesson and Ramsey, 1968), spinal cord, tectum (Ebbesson et al., 1972) and cerebellum (Ebbesson and Campbell, 1973) and have demonstrated thalamo-cortical projections (Schroeder and Ebbesson, 1974). The evidence for non-olfactory telencephalic input in frogs was described in section 4 (Feeding Behavior)., Although Herrick (1921) and Aronson and Noble (1945) mentioned thalamo-telencephalic pathways their role in behavior was not discussed. Now that anatomical and neurophysiological techniques are well-developed, these pathways and reciprocal telencephalic-hypothalamic pathways must be given some consideration in behavioral studies also.

e. Organization Of Specific Behaviors

No one has strongly contended that specific behaviors are organized in the telencephalon. Davis, Kassel and Schwagmeyer (1976) considered this possibility when they found that telencephalic ablations eliminated nest-building in male paradise fish. As supporting evidence they mentioned Demski and Knigge's (1971) finding that stimulation of the central dorsal telencephalon elicits nest-building in Lepomis macrochiris. Of course, other possible explanations exist, the simplest of which is that the threshold to respond was elevated. We were

faced with analogous findings. The loss of feeding behavior could suggest that it is organized by the forebrain. At present the explanation must be left unanswered.

VIII. SUMMARY

Xenopus laevis were tested in four behavioral experiments before and after ablation of the telencephalon, ablation of the olfactory bulbs or sham surgery. The behavioral situations and the consequences of the operations were as follows:

1. In a test of habituation to a complex acoustic-vibratory stimulus there were no changes attributable to the operations in the number of responses per session, the strength of all the responses or in the strength of the initial responses. Certain forebrain-ablated individuals, however, showed a marked decrease in the number of responses to habituate which was not seen in any of the animals in other groups.

2. There were no changes in optomotor responses to moving vertical black and white stripes attributable to the operations.

3. In an experiment involving escape from shallow water, there was a significant increase in the length of time to escape after telencephalon ablations.

4. In the feeding experiment it was demonstrated that the forebrainless animals did not approach or ingest food.

The results of the habituation experiment are consistent with that aspect of the arousal hypothesis which states that the telencephalon is involved only minimally with simple behaviors. The results of the optomotor experiment suggest that there are behaviors that are not influenced by telencephalic processes. The results of the escape experiment are consistent with the classical arousal hypothesis which stated that loss of the telencephalon results in a change in some temporal aspects of behavior but not in their abolition.

The results of the feeding experiment are not easily interpretable in terms of any known hypothesis. The total loss of feeding is attributed to the animals' inability to utilize information from a rapidly moving stimulus - the falling piece of food. It is hypothesized that this deficit is caused by the removal of telencephalic influence on tegmental areas which also receive tectal input, rather than being a specific visual deficit.

It is concluded that 'arousal,' defined as that function regulating the frequency and temporal aspects (but not response strength) of behavior, is the major function of the forebrain in ectothermic vertebrates. It is suggested that

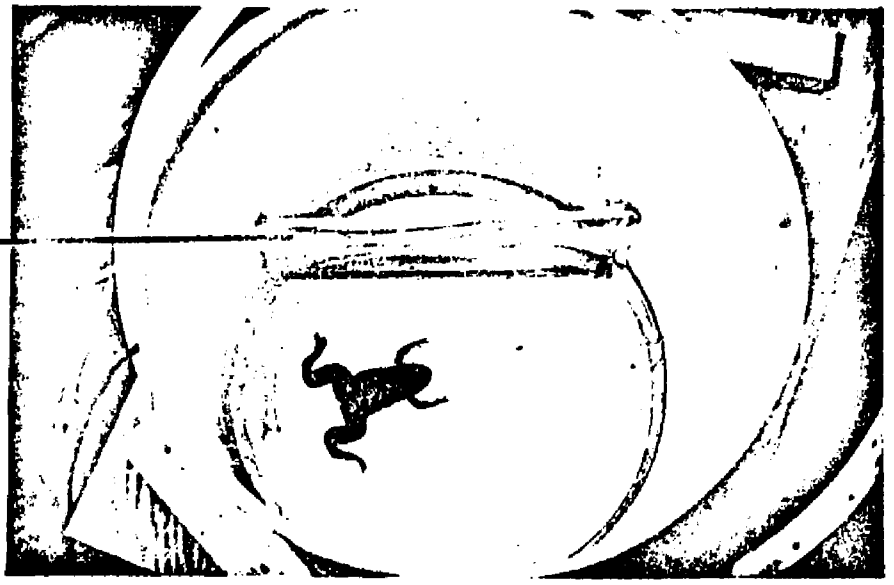
the forebrain may, in some cases, be involved in the integration of behavioral sequences (Segaar, 1965) and most probably is involved in specific functions relating to learning (Flood, Overmier and Savage, 1976). It is also stressed that additional attention be paid to the telencephalic afferent systems, namely the olfactory bulb and its possible role as a non-sensory activating system and the thalamic system which, as yet, have unknown functions but may, along with the limbic system, be involved in mediating the animal's internal state with its external environmental conditions. Additionally, it is emphasized that the forebrain may not function "as a whole." The sophistication of contemporary neuroanatomical and neurophysiological techniques should be complemented by the application of more sophisticated behavioral studies.

PHOTO 1

THE HABITUATION APPARATUS VIEWED FROM ABOVE

PHOTO 2

THE HABITUATION APPARATUS AND SEAT FROM WHICH THE ANIMALS WERE OBSERVED


Plastic Screen

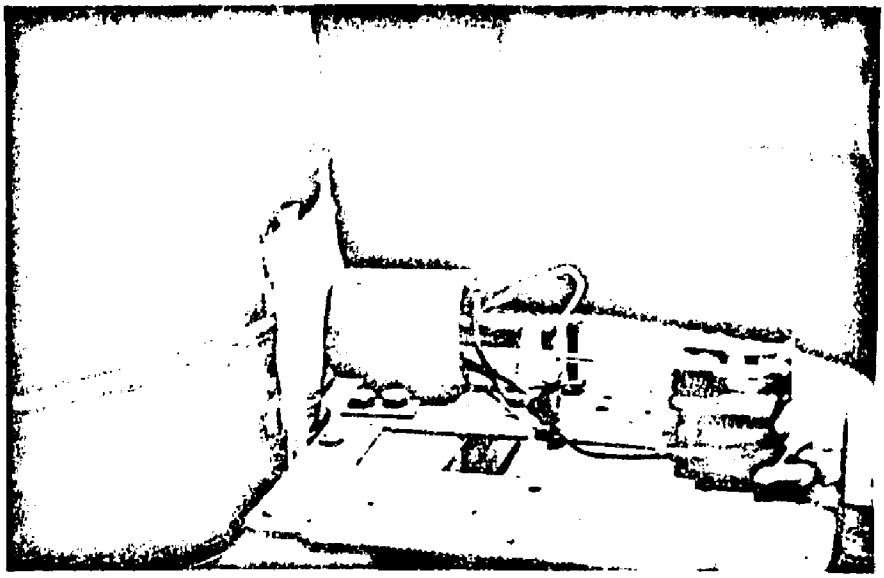

Solenoid

PHOTO 3

THE SOLENOID

PHOTO 4

THE OPTOMOTOR APPARATUS



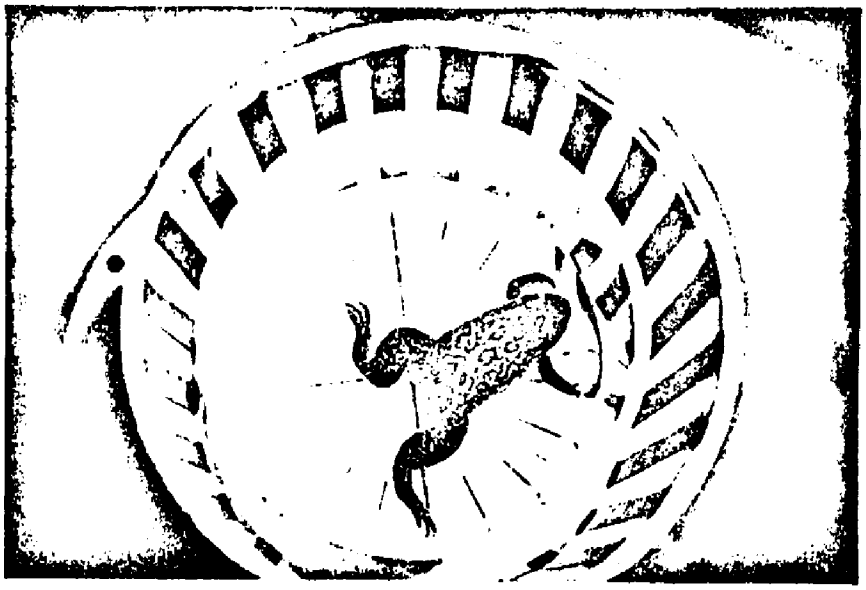


PHOTO 5

THE APPARATUS FOR THE ESCAPE EXPERIMENT

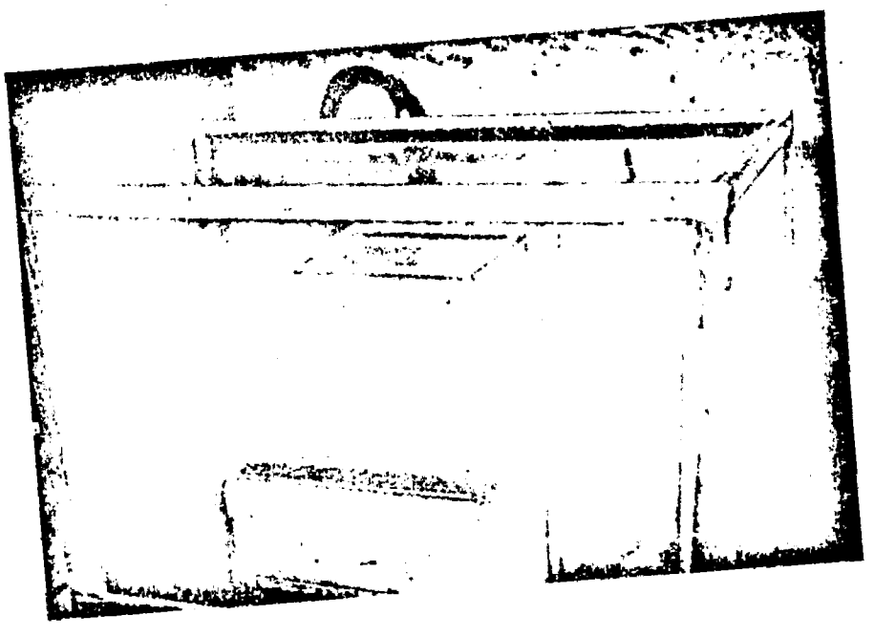
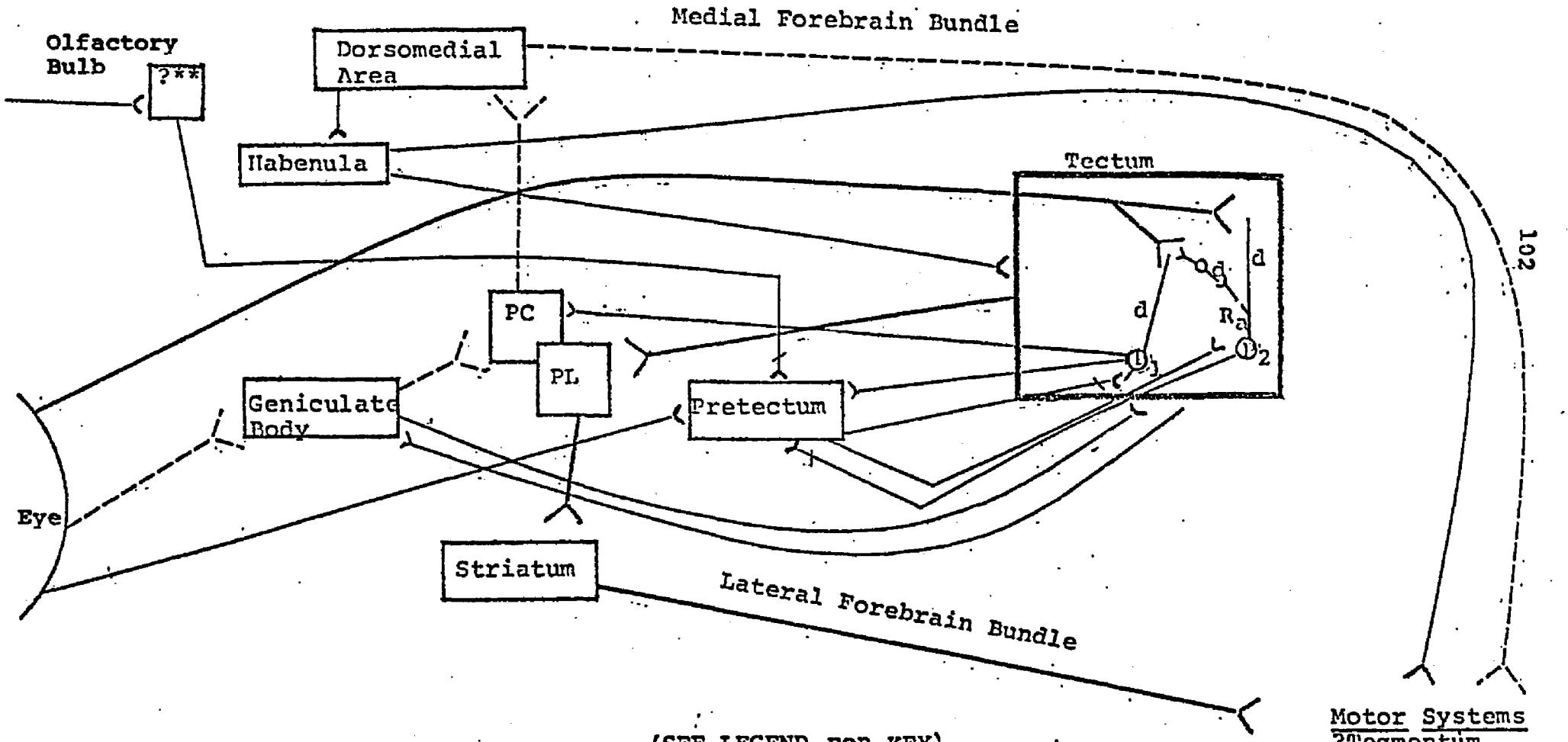


FIGURE 1

THE VISUAL SYSTEMS OF THE FROG RELATING TO BEHAVIOR POSSIBLY MEDIATED BY THE TELENCEPHALON



(SEE LEGEND FOR KEY)

FIGURE 1 LEGEND

THE VISUAL SYSTEMS

KEY

·——— =Flux and wavelength detection
——— =Food or prey-catching: Detection of small moving objects
P_1, P_2 =Pyramidal cells of the tectum
R_a =Recurrent axon of P_2
d =Dendrite
s =Stellate cell
PC =Posterocentral nucleus
PL==Posterolateral nucleus
?* =Habenulo-tectal tract. Frontera (1952) claimed it exists, Ingle (1973) claimed it does not.
?** =Unspecified telencephalic nucleus
—— =Excitatory synapse or synapse of unknown function
—⊣ =Inhibitory synapse

After Frontera (1952), Ingle (1973), Lázár (1969), Rubinson (1968), Rubinson and Colman (1972) and Trachtenberg and Ingle (1974).

TABLE 1

HISTOLOGICAL ANALYSIS OF SECTIONED BRAINS AND LIST OF EXPERIMENTS FOR EACH ANIMAL

FROG	OLFACTORY BULB	ANTERIOR OLFACTORY NUCLEUS (O.B.) *	(TEL.)*	DORSAL PALLIUM	PIRIFORM PALLIUM	STRIATUM	SEPTUM	DORSOMEDIAL PALLIUM	AMYGDALA	PREOPTIC NUCLEUS
					TELENCEPHALON ABLATED					
T-1 S:F,F	4*	4	4	4	4	4	4	4	4	1
T-2 P:H,F	May be thalamic damage.									
T-3 P:H,F	Extensive telencephalic damage. May be thalamic damage.									
T-4 W:F	Total telencephalic removal up to optic chiasm.									
T-5 P:H,F	Total telencephalic removal									
T-6 P:H	Right telencephalon complete removed. Left telencephalon almost completely removed.									
T-7 S:H	4	4	3	4	4	3	3	4	1	1
T-8 S:H	4	4	4	4	4	4	4	4	1	1
T-9 S:H	4	4	4	4	4	1	4	4	2	1

(SEE LEGEND)*

TABLE 1 CONTINUED

HISTOLOGICAL ANALYSIS OF SECTIONED BRAINS

FROG	OLFACTORY BULB	ANTERIOR OLFACTORY NUCLEUS (O.B.)	ANTERIOR OLFACTORY NUCLEUS (TEL.)	DORSAL PALLIUM	PIRIFORM PALLIUM	STRIATUM	SEPTUM	DORSOMEDIAL PALLIUM	AMYGDALA	PREOPTIC NUCLEUS
					TELENCEPHALON ABLATED					
T-10 S:H	4	4	4	4	4	2	3	4	1	1
T-11 S:H	4	4	4	4	4-Rt 3-Lft	4-Rt 3-Lft	4-Rt 3-Lft	4	2	1
T-12 P:O,E	Telencephalon almost completely removed.									
T-13 S:F,O,E	4	4	4	4	4	4	4	4	4	4
T-14 S:F,O,E	4	4	4	4	4	4	4	4	4	4
T-15 S:E	4	4	4	4	4	4	4	4	4	4
	Some thalamic damage									
T-16 S:E	4	4	4	4	3	2	2	3	2	1
T-17 S:F,O	4	4	4	4	4	4	4	4	4	1

(SEE LEGEND)

TABLE 1 CONTINUED

HISTOLOGICAL ANALYSIS OF SECTIONED BRAINS

FROG	OLFACTORY BULB	ANTERIOR OLFACTORY NUCLEUS (O.B.)	ANTERIOR OLFACTORY NUCLEUS (TEL.)	DORSAL PALLIUM	PIRIFORM PALLIUM OLFACTORY BULB ABLATED	STRIATUM	SEPTUM	DORSOMEDIAL PALLIUM	AMYGDALA	PREOPTIC NUCLEUS
O-1 P:H,F	4		1	1	1	1	1	1	1	1
O-2 P:H,F	4		1	1	1	1	1	1	1	1
O-3 P:H,F	4		1	1	1	1	1	1	1	1
O-4 P:H,F	4		1	1	1	1	1	1	1	1
O-5 P:H,F	4		1	1	1	1	1	1	1	1
O-6 S.H	4		1	1	1	1	1	1	1	1
O-7 S:H	4	4	1	1	1	1	1	1	1	1
O-8 S:H	4	4	1	1	1	1	1	1	1	1

(SEE LEGEND)

TABLE 1 CONTINUED

HISTOLOGICAL ANALYSIS OF SECTIONED BRAINS

FROG	OLFACTORY BULB	ANTERIOR OLFACTORY NUCLEUS (O.B.)	(TEL.)	DORSAL PALLIUM	PIRIFORM PALLIUM	STRIATUM	SEPTUM	DORSOMEDIAL PALLIUM	AMYGDALA	PREOPTIC NUCLEUS
					OLFACTORY BULB ABLATED					
O-9										
S:H	4	4	2	1-Rt 2-Lft	1-Rt 2-Lft	1	1	1	1	1
O-10										
P:O,E	4	1	1	1	1	1	1	1	1	1
O-11										
S:E	4-Rt 3-Lft	4	1	2-Rt 1-Lft	1	1	1	1	1	1
O-12										
S:F,O,E	4	2-Rt 1-Lft	2 1	1	1	1	1	1	1	1
O-13										
O-14										
S:F,O	4	4		3-Rt 1-Lft	3-Rt 1-Lft	1	3-Rt 1-Lft			

Slight damage to right dorsal telencephalon. Damage to left rostral and caudal pole of telencephalon.

O-15										
P:F	4		1	1	1	1	1	1	1	1

(SEE LEGEND)

TABLE 1

HISTOLOGICAL ANALYSIS OF SECTIONED BRAINS

LEGEND

The designations under the name of the animal refer to the method of evaluating the brain damage and the experiment or experiments the animal was used in. For example, S:H means the evaluation of brain damage was by the analysis of histological slides and the animal was used in the habituation experiment.

S = slides

P = photo and written description taken during autopsy but before histological workup.

W = written description taken during autopsy but before histological workup.

H = habituation experiment

F = feeding experiment

O = optomotor experiment

E = escape experiment

KEY

1 = No damage
2 = Slight damage
3 = Major damage
4 = Total removal
O.B. = Olfactory bulb
Tel. = Telencephalon

TABLE 2

HABITUATION EXPERIMENT
NUMBER OF RESPONSES FOR EACH TREATMENT GROUP
FOR ALL SESSIONS

PREOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 14-22	
Median	Range	Median	Range	Median	Range
		TELENCEPHALON ABLATED			
32	12-93	10	5-93	28	6-93
		OLFACTORY BULB ABLATED			
23	16-93	15	8-88	27	8-93
		SHAM OPERATED			
32.5	14-93	19	9-93	45	6-93
		STARVATION CONTROLS			
PERIOD I		PERIOD II		PERIOD III	
44	15-58	38	7-65	36.5	8-51

For explanation of "Periods I, II and III see footnote of Table 1, Appendix 1.

TABLE 3

HABITUATION EXPERIMENT

SIGNIFICANT DIFFERENCES* BETWEEN MEDIANS OF EACH CATEGORY OF RESPONSE

PREOPERATIVE SESSIONS 1-9 VS. POSTOPERATIVE SESSIONS 1-9	POSTOPERATIVE SESSIONS 1-9 VS. POSTOPERATIVE SESSIONS 14-22	PREOPERATIVE SESSIONS 1-9 VS. POSTOPERATIVE SESSIONS 14-22
TELENCEPHALON ABLATED		
Very Strong W = 2.5 N = 7	----	Very Strong W = 0 N = 6 Medium W = 10 N = 11 Weak W = 6 N = 8
OLFACTORY BULB ABLATED		
Weak W = 0 N = 8	Very Weak W = 2.5 N = 6	Very Strong W = 0 N = 5 Medium W = 0 N = 8 Weak W = 0 N = 7
SHAM OPERATED		
Weak W = 0 N = 5	Very Strong W = 0 N = 5	Very Strong W = 0 N = 6 Very Weak W = 2 N = 7

*Significant changes were decreases with time at $p < .05$, Wilcoxon signed ranks test. Based on data in Appendix 1 Table 4

TABLE 4

HABITUATION EXPERIMENT

SIGNIFICANT DIFFERENCES* AMONG THE MODAL RESPONSE

PREOPERATIVE SESSIONS 1-9 VS. POSTOPERATIVE SESSIONS 1-9	POSTOPERATIVE SESSIONS 1-9 VS. POSTOPERATIVE SESSIONS 14-22	PREOPERATIVE SESSIONS 1-9 VS. POSTOPERATIVE SESSIONS 14-22
	TELENCEPHALON ABLATED	
Not Significant	Significant*	Not Significant
	W = 3.5 N = 9	
	OLFACTORY BULB ABLATED	
Not Significant	Significant	Significant
	W = 0 N = 5	W = 0 N = 8
	SHAM OPERATED	
Not Significant	Significant	Not Significant
	W = 0 N = 5	

Based on data in Appendix 1 Table 5.
*Wilcoxon signed ranks test ($p < .05$, two-tailed)

TABLE 5

POSTOPERATIVE FEEDING BEHAVIOR

Week	1	2	3	4	5	6	7	8
TELENCEPHALON ABLATED								
T-1	-	-	-	-	-	-	-	
T-2	-	-	-	-	-	-	-	
T-3	-	-	-	-	-	-	-	
T-5	-	-	-	-	-	-	-	
T-13	?	-	-	-	-	-	-	-[1]
T-14	-	-	-	-	-	-	-	-
T-17	-		-	-	-	-	-	
OLFACTORY BULB ABLATED								
O-1	-	+	+	+	+	+	+	+
O-2	-	-	-	-	-	-	-	-
O-3	+	+	+	+	+	+	+	+
O-4	+	+	+	+	+	+	+	+
O-5	+	+	+	+	+	+	+	+
O-12	+		+	+	+	+	+	+
O-14	-	+	+	+	+	+	+	+
O-15	+		?	+	+	+	-	+
SHAM OPERATED								
S-1	+	+	+	+	+	+	+	+
S-3	+	+	+	+	+	+	+	
S-9	?	+	+	+	+	+	+	+
S-10	?	+	+	-	-	+	-	+
S-11	-	+	+	+	+	+	+	
S-12	-	-[2]	-	+	+	+	+	

(SEE LEGEND)

TABLE 5

FEEDING BEHAVIOR

KEY

+ = Normal feeding response

- = No feeding response

? = It was not possible to ascertain whether or not the animal fed.

1 = The animal tracked thefood, but did not open its mouth.

2 = The animal showed the 'aroused'searching but not the visual tracking.

Appendix 1

HABITUATION EXPERIMENT - SPECIAL ANALYSES

Before the analysis of the data by operative groups could be done, two questions had to be answered. First, two groups of animals were run, one starting in March, 1975, the other in August 1975. It had to be demonstrated thatthe data from these groups could justifiably be pooled. Second, the question arose as to how to organize the data pertaining to habituated days, non-habituated days and Air-days (A-days): should the Air-days be included in the sets of habituated and non-habituated days in which they occurred or should they be treated separately?

A. Number of Responses: March and August Groups

Because of a mechanical error there was some variation in the intensity and frequency of the acoustic-vibratory stimulus and there was evidence that the stimulus was not the same for the March and August groups. In order to ascertain whether or not this caused a significant difference between the groups a Mann-Whitney U test was done comparing the March and August groups (see table 7). It showed no significant difference ($p > .05$)

between any of the operative groups or any of the time periods. Therefore the two groups were pooled for all further calculations.

B. Air-Response Days

1. Justification for Including Air-Day Data

The question of how to properly treat the A-days and the non-habituated days was more complex. Table 8 shows the distribution of A-days over the three time periods. A consistent overall pattern does not emerge. It seems that certain animals never had A-days (T-1, O-3 and S-5); some animals had a consistently high number of A-days (e.g., T-4, O-4 and S-7); some animals had occasional A-days during all phases of the experiment (T-8, O-5 and S-4); and certain animals had large variations in their number of A-days in all experimental periods (T-2, O-1 and S-2). In addition, the same inconsistency was found for the starvation controls (see table 9). There does not appear to be a pattern of changes attributable to any of the operative treatments. These findings led us to consider that pooling of the data of the A-days with the non-A-days was justified.

2. Relationship of A-Days to Habituation

Table 10 shows the number of sessions in which the frogs did or did not habituate and the number of sessions of A-days during each of the periods. The sessions are arranged as for a Fisher Exact Poobability Test, which was used to determine

whether there was a relation between the factors of habituation and A-days. This arrangement of the data shows that the typical pattern is for the animal to show more sessions of habituation than sessions of non-habituation.

C. Summary

Consideration of the March and August groups and of the A-days, non-habituated days and habituated days resulted in the decision to pool the two groups and to include all the sessions in the analysis of the data.

D. Air Responses

Although it was decided to include the A-days in the analysis of the data, the significance of A-responses remained unsettled.

Because A-responses were at first thought to be due to overfeeding, which caused the animals to become bloated and float at the surface, the distribution of A-days and of A-responses of A-days was tabulated for the starvation control to see if lack of feeding would abolish the A-responses (table 9). This table demonstrates that the A-responses show patterns for individuals, not for the group as a whole, just as table 8 showed for the operative groups. In addition, the same individual types of patterns are seen, even in this small group: J-1 is a strong A-responder, J-2, J-3 and J-5 are not A-responders and J-4 falls in between. Because A-

responses occurred even in the starvation animals and because the A-responses showed the same inconsistent pattern as in the operated animals it was concluded that feeding is not a necessary cause for A-responses.

To further the analysis, the number of the trials within each session on which an A-response occurred was tabulated (table 9). This was done to examine the hypothesis that the A-responses were reactions to stress which implies that A-responses should occur during the latter part of a session. Table 9 shows that some pattern is apparent: except for occasional A-responses which occurred as the first response in a session the A-responses tend to cluster in the middle or end of a session. There are no clusters of A-responses before the twentieth response. This pattern is generally true for the experimental animals also, although some clusters of A-responses near the beginning of sessions and some sessions of only A-responses also occurred. This suggests that A-responses were reactions to stress, perhaps escape "attempts." On occassion animals did actually attempt to climb out of the apparatus. There were also occassions on which animals habituated with their head above the surface. In these cases the perceptual strength of the stimulus may have been decreased by the elevation of the lateral line stitches out of the

water. This may be considered a postural adaptation to stress.

APPENDIX 1

TABLE 1

HABITUATION EXPERIMENT

NUMBER OF RESPONSES

FROG	PREOPERATIVE SESSION 1	2	3	4	5	6	7	8	9	POSTOPERATIVE SESSION 1	2	3	4	5	6	7	8	9	POSTOPERATIVE SESSION 14	15	16	17	18	19	20	21
TELENCEPHALON ABLATED																										
T-1	7	34	60	37	10	28	35	93	9	4	5	11	5	4	6	4	7	5	5	6	6	8	7	5	9	6	4
T-2	8	20	20	10	10	11	12	12	15	5	10	8	9	10	12	13	92	11	57	83	93	5	93	93	93	77	93
T-3	93	93	93	69	93	9	70	93	93	50	73	53	41	16	9	14	17	14	63	19	13	43	46	26	28	51	22
T-4	17	18	89	7	8	32	21	93	93	93	93	93	93	93	93	93	19	93	93	93	93	93	93	93	93	93	93
T-5	28	43	67	24	18	15	10	31	17	7	7	10	5	19	11	6	55	5	67	93	93	93	56	5	6	93	33
T-6	93	32	14	93	19	24	13	10	70	34	7	5	11	6	5	5	5	5	5	5	93	5	6	7	6	5	11
T-7	93	78	93	93	93	80	90	74	93	10	55	89	93	39	28	73	68	60	86	62	61	37	48	88	35	51	27
T-8	70	59	16	93	93	9	63	93	84	93	93	9	12	7	9	6	11	7	6	7	9	5	8	9	18	5	6
T-9	30	10	33	9	31	17	11	6	22	14	43	40	39	69	6	29	69	16	93	39	9	93	15	30	13	14	12
T-10	47	93	93	16	54	21	15	64	7	25	75	19	63	22	31	45	32	93	74	76	12	36	22	52	40	93	8
T-11	69	27	75	32	10	55	48	9	6	5	6	4	6	4	5	6	5	5	16	12	18	12	18	24	23	9	8
OLFACTORY BULB ABLATED																										
O-1	23	25	19	23	8	22	17	18	39	35	93	39	93	27	93	48	19	35	12	21	93	93	8	64	93	93	93
O-2	17	12	16	13	17	6	22	8	23	11	5	11	17	5	20	7	20	8	14	5	18	16	5	12	5	8	6
O-3	51	76	61	93	11	14	17	6	7	12	14	9	74	18	8	10	15	20	23	9	21	4	8	7	6	9	21
O-4	93	93	93	73	88	93	93	93	93	86	51	37	68	93	93	88	93	93	93	57	78	13	93	93	93	93	93
O-5	8	21	51	16	14	42	17	5	34	15	93	8	7	63	18	6	78	93	93	93	92	50	93	93	4	4	5
O-6	82	93	93	22	93	66	12	10	27	67	64	5	36	12	8	5	6	6	9	5	19	57	8	24	13	1	8
O-7	93	93	37	27	20	30	11	15	19	9	93	10	15	14	16	8	78	9	93	27	93	93	93	11	14	5	7
O-8	26	77	20	73	93	67	20	31	10	17	51	6	26	8	7	11	16	10	25	8	17	15	32	93	5	19	5
O-9	15	55	32	25	21	74	14	19	23	9	14	16	20	13	12	20	38	7	14	93	93	17	93	93	93	93	43

APPENDIX 1

TABLE 1 CONTINUED

HABITUATION EXPERIMENT

NUMBER OF RESPONSES

	PREOPERATIVE SESSION									POSTOPERATIVE SESSION									POSTOPERATIVE SESSION								
	1	2	3	4	5	6	7	8	9	1	2	3	4	5	6	7	8	9	14	15	16	17	18	19	20	21	22
SHAM OPERATED																											
S-1	9	93	93	9	10	14	10	16	93	11	5	6	11	93	6	12	8	9	5	5	7	6	53	6	25	8	5
S-2	93	79	9	47	31	32	25	23	27	54	93	93	26	93	93	93	93	25	17	93	55	45	12	93	16	16	46
S-3	10	39	12	18	5	8	49	30	53	36	93	93	31	85	46	76	93	8	44	40	80	60	93	93	18	12	93
S-4	59	11	93	20	35	12	16	93	52	43	13	12	5	21	41	25	5	8	19	32	17	6	93	70	19	9	8
S-5	54	12	19	17	83	6	9	14	11	9	15	41	13	12	8	6	12	8	10	7	6	6	10	6	19	12	9
S-6	56	59	93	93	34	25	23	10	23	21	23	15	10	22	17	26	11	6	26	24	13	12	39	8	17	8	9
S-7	73	61	93	93	93	93	93	93	16	38	93	93	93	50	93	79	93	93	93	93	93	84	93	93	93	42	93
S-8	50	60	93	93	93	32	93	19	77	19	74	15	69	8	10	16	71	30	66	28	57	6	46	19	93	6	44
STARVATION CONTROLS*																											
J-1	29	18	93	65	68	87	93	48	93	32	87	84	23	26	50	51	25	30	38	29	13	66	93	34	93	29	20
J-2	18	15	10	14	15	21	12	11	19	6	6	10	8	10	10	6	7	8	12	8	8	8	10	8	11	7	9
J-3	44	32	35	78	93	91	39	42	89	34	22	65	93	16	93	68	23	93	56	93	33	44	46	56	16	36	30
J-4	23	28	14	54	23	38	37	93	60	33	93	50	69	41	50	44	55	59	59	54	34	62	48	38	40	93	25
J-5	93	16	49	55	27	29	57	46	93	30	23	21	38	19	47	78	38	40	46	44	44	93	70	25	29	12	7

*Periods I, II and III for the starvation control group are comparable to Preoperative Sessions 1-9, Postoperative Sessions 1-9 and 14-22 for the experimental animals in three ways: a) they each consist of nine sessions over comparable number of days b) animals were fed during Period I and Preoperative Sessions 1-9 c) animals were not fed during Periods II and III and the telencephalon-ablated animals did not eat during the two postoperative periods.

APPENDIX 1
TABLE 2

HABITUATION EXPERIMENT
NUMBER OF RESPONSES PER SESSION

	PREOPERATIVE SESSIONS 1-9	POSTOPERATIVE SESSIONS 1-9	POSTOPERATIVE SESSIONS 14-22
		TELENCEPHALON ABLATED	
T-1	35	5	6
T-2	12	10	93
T-3	93	17	28
T-4	21	93	93
T-5	24	7	67
T-6	24	5	6
T-7	93	68	51
T-8	70	9	7
T-9	22	39	15
T-10	47	32	40
T-11	32	5	16
		OLFACTORY BULB ABLATED	
O-1	22	39	81
O-2	16	8	8
O-3	17	14	9
O-4	93	88	93
O-5	17	18	93
O-6	66	18	13
O-7	27	15	27
O-8	31	11	19
O-9	23	14	93
		SHAM OPERATED	
S-1	14	9	6
S-2	31	93	45
S-3	18	76	59
S-4	35	13	19
S-5	17	12	10
S-6	34	17	13
S-7	93	93	93
S-8	77	19	46
		STARVATION CONTROLS	
	PER I	PER II	PER III
J-1	58	32	36
J-2	15	7	8
J-3	44	65	45
J-4	40	50	51
J-5	52	58	36.5

For explanation of "Periods I, II and III" see footnote of Table 1, Appendix 1.

APPENDIX-1

TABLE 3

HABITUATION EXPERIMENT

ONE-SAMPLE RUNS OF TESTS OF NUMBER OF RESPONSES IN 27 SESSIONS

FROG	TELENCEPHALON ABLATED
T-1	Not significant
T-2	Not significant
T-3	Not significant
T-4	Not significant
T-5	Not significant
T-6	Not significant
T-7	Not significant
T-8	Not significant
T-9	Significant
T-10	Significant
T-11	Significant
	OLFACTORY BULB ABLATED
O-1	Significant
O-2	Significant
O-3	Not significant
O-4	Not significant
O-5	Not significant
O-6	Not significant
O-7	Significant
O-8	Significant
O-9	Not significant
	SHAM OPERATED
S-1	Not significant
S-2	Not significant
S-3	Significant
S-4	Not significant
S-5	Not significant
S-6	Not significant
S-7	Significant
S-8	Significant

"Significant" means that the number of changes (in number of responses) from session to session was greater than expected at $p < .05$

APPENDIX 1

TABLE 4

NUMBER OF RESPONSES IN EACH CATEGORY OF RESPONSE.

FROG	PREOPERATIVE SESSIONS 1-7							POSTOPERATIVE SESSIONS 1-9							POSTOPERATIVE SESSIONS 14-22						
	VS*	S	M	W	VW	N	A	VS	S	M	W	VW	N	A	VS	S	M	W	VW	N	A
TELENCEPHALON ABLATED																					
T-1	0	0	2	1	25	5	0	0	0	0	0	0	4	0	0	1	0	0	0	4	1
T-2	2	2	2	0	0	5	0	0	3	1	0	0	5	1	0	33	5	0	0	9	33
T-3	1	3	4	11	41	4	0	0	2	1	2	1	7	2	0	5	0	0	0	9	17
T-4	4	2	2	0	2	6	0	0	60	1	0	0	8	21	0	65	0	0	0	7	18
T-5	0	3	5	1	6	5	0	0	3	1	0	0	4	0	0	0	0	0	0	4	31
T-6	0	2	2	3	8	4	0	0	0	0	0	0	4	0	0	0	1	0	0	4	0
T-7	9	25	10	19	7	7	0	0	0	26	10	5	6	0	0	21	8	3	3	9	0
T-8	0	24	4	1	1	10	19	0	0	1	0	0	4	0	0	1	1	0	0	4	0
T-9	0	3	2	1	3	6	0	2	2	5	1	6	5	0	0	5	3	1	2	5	0
T-10	3	1	5	3	3	5	0	0	0	2	11	13	7	0	0	1	2	22	9	5	0
T-11	3	4	4	1	1	7	0	0	0	1	0	0	4	0	2	4	3	0	1	4	0
OLFACTORY BULB ABLATED																					
O-1	2	7	2	1	1	5	0	0	8	5	0	0	4	26	0	5	2	0	1	4	77
O-2	0	0	1	1	3	4	0	0	0	1	0	2	4	0	0	1	1	1	1	4	0
O-3	1	1	4	4	2	4	0	0	1	2	0	4	5	0	0	1	1	1	1	5	0
O-4	3	50	10	5	2	4	12	0	21	12	1	2	5	21	0	50	7	1	0	3	13
O-5	1	4	2	2	0	4	0	0	7	6	0	0	4	2	0	16	9	0	0	7	17
O-6	0	1	4	3	38	7	0	0	1	1	1	1	4	0	0	1	3	1	0	6	0
O-7	1	3	4	3	1	5	0	0	1	1	0	1	4	0	0	0	0	0	0	4	8
O-8	0	3	4	2	13	8	0	0	1	1	0	0	4	0	0	6	1	0	0	4	0
O-9	0	2	3	3	7	7	0	1	1	1	1	2	5	0	0	25	12	0	2	11	35

APPENDIX 1

TABLE 4 CONTINUED

NUMBER OF RESPONSES IN EACH CATEGORY OF RESPONSE

	PREOPERATIVE SESSIONS 1-9							POSTOPERATIVE SESSIONS 1-9							POSTOPERATIVE SESSIONS 14-22						
PROG	VS	S	M	W	VW	N	A	VS	S	M	W	VW	N	A	VS	S	M	W	VW	N	A
SHAM OPERATED																					
S-1	0	1	2	1	2	4	0	0	0	1	1	0	4	0	0	0	1	1	1	4	0
S-2	2	16	4	0	1	5	0	1	25	7	0	0	2	36	0	5	4	1	2	5	0
S-3	2	1	2	1	6	5	0	2	12	3	0	0	9	31	0	15	2	0	0	8	22
S-4	4	8	3	1	0	4	0	1	4	0	0	0	4	0	1	10	1	0	0	4	0
S-5	0	2	3	2	2	4	0	0	2	1	0	1	4	0	0	3	2	0	0	4	0
S-6	3	7	7	3	6	6	0	3	2	1	1	3	5	0	1	3	1	1	1	5	0
S-7	13	28	4	4	3	8	26	20	25	5	4	2	4	54	7	17	5	3	2	5	46
S-8	3	16	7	7	7	4	1	1	2	1	2	9	6	0	0	11	6	4	3	7	1

KEY

VS = Very Strong
S = Strong
M = Medium
W = Weak
VW = Very Weak
N = No Response
A = Air Response

APPENDIX 1

TABLE 5

HABITUATION EXPERIMENT

MODAL QUALITATIVE RESPONSES

FROG	PREOPERATIVE SESSIONS 1-9	POSTOPERATIVE SESSIONS 1-9	POSTOPERATIVE SESSIONS 14-22
		TELENCEPHALON ABLATED	
T-1	6*	6	3
T-2	4	3	3
T-3	6	3	1
T-4	2	3	3
T-5	6	4	1
T-6	6	3	4
T-7	3	4	3
T-8	1	4	3
T-9	6	4	3
T-10	6	6	5
T-11	3	4	3
		OLFACTORY BULB ABLATED	
O-1	3	1	1
O-2	6	6	4
O-3	6	6	3
O-4	3	3	3
O-5	3	3	1
O-6	6	6	4
O-7	6	4	1
O-8	6	3	3
O-9	6	3	3
		SHAM OPERATED	
S-1	6	5	4
S-2	3	3	3
S-3	6	3	1
S-4	3	3	3
S-5	4	4	3
S-6	3	6	3
S-7	3	1	1
S-8	3	6	3

A Wilcoxon test comparing changes over time for each group showed an increase in the mode of response strength for all three groups between postoperative sessions 1-9 and 14-22 ($p < .05$, two-tailed) and between preoperative sessions 1-9 and postoperative sessions 14-22.

APPENDIX 1

TABLE 5 CONTINUED

HABITUATION EXPERIMENT

MODAL QUALITATIVE RESPONSES

KEY

*1 = Air response

2 = Very Strong

3 = Strong

4 = Medium

5 = Weak

6 = Very Weak

7 = No response

APPENDIX 1

TABLE 6

HABITUATION EXPERIMENT
INITIAL RESPONSE FOR EACH SESSION

FROG	PREOPERATIVE SESSIONS 1-9									POSTOPERATIVE SESSIONS 1-9									POSTOPERATIVE SESSIONS 14-22								
TELENCEPHALON ABLATED																											
T-1	4	4	4	4	4	6	4	1	4	7	7	1	5	7	1	7	3	4	1	1	1	3	3	1	1	7	1
T-2	2	2	4	2	2	2	1	2	2	4	1	6	1	3	1	1	3	7	1	4	4	1	1	1	1	1	1
T-3	2	4	3	4	3	4	4	2	4	4	1	5	1	1	1	1	1	1	1	1	1	1	1	1	1	5	1
T-4	2	2	2	2	2	1	4	2	1	3	3	3	7	3	1	1	1	2	1	1	2	2	2	3	2	3	3
T-5	3	4	3	4	4	4	3	4	1	3	3	3	4	1	1	1	4	4	4	1	4	1	1	1	1	1	1
T-6	3	4	3	4	3	3	4	2	3	1	3	3	7	1	5	6	4	3	5	4	4	4	4	4	4	5	1
T-7	2	2	2	2	4	2	3	2	3	4	3	3	4	4	4	4	4	4	4	3	4	2	2	3	3	3	4
T-8	3	2	3	3	7	3	3	3	3	7	1	1	2	3	3	4	4	4	4	4	2	3	3	3	5	3	3
T-9	2	2	4	4	4	4	3	4	4	5	3	3	2	2	4	3	3	2	3	2	3	2	2	4	4	4	6
T-10	2	2	3	3	2	2	2	3	2	3	3	4	2	4	2	3	4	4	3	3	4	3	3	3	3	4	4
T-11	2	2	3	3	4	2	3	3	2	4	4	7	4	7	4	4	5	4	3	2	2	2	2	2	2	2	2
OLFACTORY BULB ABLATED																											
O-1	2	2	4	2	2	2	2	2	3	4	1	1	1	1	4	1	3	4	1	4	1	7	7	4	1	1	1
O-2	2	4	3	2	4	1	1	1	3	1	6	4	1	4	4	4	4	1	4	4	3	4	4	3	4	3	3
O-3	4	4	2	2	3	2	1	1	4	4	3	4	1	1	3	4	1	3	3	1	3	7	7	4	1	4	1
O-4	2	2	2	2	2	2	2	4	3	3	4	3	1	3	3	3	3	3	3	4	3	3	3	3	3	3	3
O-5	2	2	1	5	2	2	4	5	4	4	3	4	4	1	3	1	1	3	4	1	3	1	1	1	7	7	4
O-6	3	4	3	4	3	4	4	4	3	3	4	5	2	2	1	3	5	4	3	3	4	3	3	4	3	2	4
O-7	3	3	3	2	4	1	3	3	3	3	1	3	3	3	4	4	1	3	1	3	1	1	1	3	1	5	3
O-8	2	4	6	3	4	4	4	4	3	7	4	4	4	3	4	3	3	3	3	4	3	3	3	1	4	3	5
O-9	4	2	4	3	3	1	3	3	3	2	3	3	2	3	2	2	3	3	3	3	1	3	3	3	3	3	4

APPENDIX 1

TABLE 6 CONTINUED

HABITUATION EXPERIMENT

INITIAL RESPONSES FOR EACH SESSION

	PREOPERATIVE SESSIONS 1-9									POSTOPERATIVE SESSIONS 1-9										POSTOPERATIVE SESSIONS 14-22									
FROG	SHAM OPERATED																												
S-1	2	4	3	4	2	4	4	4	3	4	4	5	4	1	3	4	5	7	4	5	4	2	2	4	4	5	4
S-2	3	2	3	3	4	2	2	3	3	2	3	2	2	3	3	3	3	2	4	3	3	3	3	2	2	4	3
S-3	2	2	2	4	5	2	2	2	2	2	1	2	1	2	3	3	4	2	1	1	2	2	2	3	1	3	3
S-4	2	4	3	3	2	2	2	4	2	3	3	2	6	3	2	1	5	3	4	3	2	4	4	2	2	3	4
S-5	2	5	3	3	4	5	3	3	4	3	2	4	2	3	4	4	3	3	3	4	4	4	4	1	3	3	1
S-6	2	2	2	2	2	2	2	3	2	3	2	2	2	2	2	5	2	4	3	2	3	3	3	2	2	4	3
S-7	2	2	2	2	2	2	3	3	3	2	2	3	3	2	3	8	2	2	3	2	2	2	2	3	2	3	2
S-8	2	2	4	3	2	3	2	3	3	3	3	2	2	3	3	4	2	2	3	3	3	4	4	3	2	4	4

A Friedman two-way ANOVA showed no significant differences ($p > .05$) in the initial responses either with or between any time periods for any of the operative groups

KEY

1=Air response
2=Very strong
3=Strong
4=Medium
5=Weak
6=Very weak
7=No response

APPENDIX 1

TABLE 7

HABITUATION EXPERIMENT

RANGE AND MEDIANS OF NUMBER OF RESPONSES
MARCH GROUP VS. AUGUST GROUP

PREOPERATIVE SESSIONS 1-9

	MARCH		AUGUST	
FROGS	Median	Range	Median	Range
		TELENCEPHALON ABLATED		
T-6	24	10-93	T-1	35	7-93
T-7	93	74-93	T-2	12	8-20
T-8	70	9-93	T-3	93	9-93
T-10	47	7-93	T-4	32	7-93
T-11	32	6-69	T-5	24	10-64
		OLFACTORY BULB ABLATED		
O-6	30	11-93	O-1	22	8-39
O-7	31	10-93	O-2	16	6-22
O-8	66	10-93	O-3	17	6-93
O-9	25	14-55	O-4	93	73-93
			O-5	17	8-51
		SHAM OPERATED		
S-4	35	12-93	S-1	14	9-93
S-5	17	6-83	S-2	31	9-93
S-6	34	10-93	S-3	18	5-53
S-7	93	16-93			
S-8	77	32-93			

APPENDIX 1

TABLE 7 CONTINUED

HABITUATION EXPERIMENT

RANGE AND MEDIANS OF NUMBER OF RESPONSES
MARCH GROUP VS. AUGUST GROUP

PREOPERATIVE SESSIONS 1-9

	MARCH		AUGUST	
FROGS	Median	Range	Median	Range
		TELENCEPHALON ABLATED		
T-6	5	5-34	5	4-11
T-7	60	10-93	10	5-92
T-8	9	7-93	17	9-73
T-10	32	19-93	93	19-93
T-11	5	4-06	7	5-58
T-9	39	6-69		
		OLFACTORY BULB ABLATED		
O-6	8	5-67	39	19-93
O-7	14	8-93	11	7-20
O-8	11	6-51	14	8-74
O-9	14	7-38	39	19-93
		SHAM OPERATED		
S-4	13	5-93	9	5-93
S-5	12	6-41	93	25-93
S-6	17	6-23	76	8-93
S-7	93	38-93		
S-8	19	8-74		

APPENDIX 1

TABLE 7 CONTINUED

HABITUATION EXPERIMENT

RANGE AND MEDIANS OF NUMBER OF RESPONSES
MARCH GROUP VS. AUGUST GROUP

POSTOPERATIVE SESSIONS 14-22

	MARCH		AUGUST	
FROGS	Median	Range	Median	Range
		TELENCEPHALON ABLATED		
T-6	6	5-93	T-1	6	5-19
T-7	51	27-88	T-2	93	5-93
T-8	8	5-18	T-3	28	13-51
T-10	15	9-93	T-4	93	77-93
T-11	16	8-24	T-5	93	5-93
T-9	15	9-93			
		OLFACTORY BULB ABLATED		
O-6	19	5-57	O-1	21	5-93
O-7	27	5-93	O-2	12	5-23
O-8	17	5-93	O-3	9	4-23
O-9	93	14-93	O-4	93	13-93
			O-5	93	4-93
		SHAM OPERATED		
S-4	19	6-93	S-1	6	5-53
S-5	10	6-19	S-2	45	12-93
S-6	13	8-39	S-3	60	12-93
S-7	93	42-93			
S-8	46	6-93			

APPENDIX 1

TABLE 8

DISTRIBUTION OF AIR-DAYS FOR THE EXPERIMENTAL ANIMALS

PREOPERATIVE SESSIONS 1-9

FROG	NUMBER AIR-DAYS	AIR-HABITUATED DAYS	AIR-NONHABITUATED DAYS	NUMBER OF TRIALS TO HABITUATE DURING HABITUATED DAYS MEDIAN	HABITUATED DAYS RANGE	NONHABITUATED DAYS MEDIAN	NONHABITUATED DAYS RANGE
			TELENCEPHALON ABLATED				
T-1	0	--	--	--	--	--	--
T-2	0	--	--	--	--	--	--
T-3	1	--	1	--	--	87	--
T-4	4	2	2	25	10-40	34.5	31-38
T-5	1	1	0	47	47	--	--
T-6	0	--	--	--	--	--	--
T-7	2	1	1	18	--	36	--
T-8	5	2	3	20	53-81	56	53-81
T-9	0	--	--	--	--	--	--
T-10	1	0	1	--	--	30	--
T-11	3	3	0	27	--	--	--
			OLFACTORY BULB ABLATED				
O-1	0	--	--	--	--	--	--
O-2	1	1	--	16	--	--	--
O-3	0	--	--	--	--	--	--
O-4	5	2	3	20.5	12-29	14.5	10-26
O-5	2	2	0	32	7-43	--	--
O-6	0	--	--	--	--	--	--
O-7	2	1	1	11	--	76	--
O-8	0	--	--	--	--	--	--
O-9	1	1	0	53	--	--	--

APPENDIX 1

TABLE 8 CONTINUED

DISTRIBUTION OF AIR-DAYS FOR THE EXPERIMENTAL ANIMALS

PREOPERATIVE SESSIONS 1-9

FROG	NUMBER AIR-DAYS	AIR-HABITUATED DAYS	AIR-NONHABITUATED DAYS	NUMBER OF TRIALS TO HABITUATE DURING HABITUATED DAYS MEDIAN	HABITUATED DAYS RANGE	NONHABITUATED DAYS MEDIAN	NONHABITUATED DAYS RANGE
			SHAM OPERATED				
S-1	2	0	2	--	--	31	23-29
S-2	1	0	1	--	--	30	--
S-3	0	--	--	--	--	--	--
S-4	4	3	1	30	7-71	75	--
S-5	0	--	--	--	--	--	--
S-6	0	--	--	--	--	--	--
S-7	5	1	4	26	34-51	36.5	34-51
S-8	4	--	4	--	16-96	78	16-96

APPENDIX 1

TABLE 8 CONTINUED

DISTRIBUTION OF AIR-DAYS FOR THE EXPERIMENTAL ANIMALS

POSTOPERATIVE SESSIONS 1-9

FROG	NUMBER AIR-DAYS	AIR-HABITUATED DAYS	AIR-NONHABITUATED DAYS	NUMBER OF TRIALS TO HABITUATE DURING HABITUATED DAYS MEDIAN	HABITUATED DAYS RANGE	NONHABITUATED DAYS MEDIAN	NONHABITUATED DAYS RANGE
			TELENCEPHALON ABLATED				
T-1	0	--	--	--	--	--	--
T-2	1	--	1	--	--	7	--
T-3	3	3	0	9	4-12	--	--
T-4	6	0	6	--	--	22	19-26
T-5	1	1	0	50	--	--	--
T-6	1	1	0	17	--	--	--
T-7	0	--	--	--	--	--	--
T-8	2	0	2	0	--	93	--
T-9	0	--	--	--	--	--	--
T-10	0	--	--	--	--	--	--
T-11	0	--	--	--	--	--	--
			OLFACTORY BULB ABLATED				
O-1	6	3	3	26	16-34	58	47-89
O-2	0	--	--	--	--	--	--
O-3	0	--	--	--	--	--	--
O-4	5	1	4	21	--	26.5	21-43
O-5	4	2	2	29	17-41	68	58-78
O-6	0	--	--	--	--	--	--
O-7	2	1	1	72	--	93	--
O-8	1	1	0	18	--	--	--
O-9	0	--	--	--	--	--	--

APPENDIX 1

TABLE 8 CONTINUED

DISTRIBUTION OF AIR-DAYS FOR THE EXPERIMENTAL ANIMALS

POSTOPERATIVE SESSIONS 1-9

FROG	NUMBER AIR-DAYS	AIR-HABITUATED DAYS	AIR-NONHABITUATED DAYS	NUMBER OF TRIALS TO HABITUATE DURING HABITUATED DAYS MEDIAN	HABITUATED DAYS RANGE	NONHABITUATED DAYS MEDIAN	NONHABITUATED DAYS RANGE
			SHAM OPERATED				
S-1	1	0	1	0	--	87	--
S-2	6	0	6	--	--	39	27-69
S-3	6	3	3	31	11-54	70	55-74
S-4	2	1	1	20	--	93	--
S-5	0	--	--	--	--	--	--
S-6	0	--	--	--	--	--	--
S-7	7	1	1	8	--	61	34-75
S-8	0	--	--	--	--	--	--

APPENDIX 1

TABLE 8 CONTINUED

DISTRIBUTION OF AIR-DAYS FOR THE EXPERIMENTAL ANIMALS

POSTOPERATIVE SESSIONS 14-22

FROG	NUMBER AIR-DAYS	AIR-HABITUATED DAYS	AIR-NONHABITUATED DAYS	NUMBER OF TRIALS TO HABITUATE DURING HABITUATED DAYS MEDIAN	HABITUATED DAYS RANGE	NONHABITUATED DAYS MEDIAN	NONHABITUATED DAYS RANGE
			TELENCEPHALON ABLATED				
T-1	0	--	--	--	--	--	--
T-2	8	2	6	31	--	38.5	30-52
T-3	7	7	0	17	4-43	--	--
T-4	7	0	7	--	--	21	13-25
T-5	6	2	4	31	29-52	93	27-93
T-6	0	--	--	--	--	--	--
T-7	0	--	--	--	--	--	--
T-8	0	--	--	--	--	--	--
T-9	0	--	--	--	--	--	--
T-10	0	--	--	--	--	--	--
T-11	0	--	--	--	--	--	--
			OLFACTORY BULB ABLATED				
O-1	6	1	5	44	--	83	76-93
O-2	0	--	--	--	--	--	--
O-3	0	--	--	--	--	--	--
O-4	4	1	3	16	--	23	15-25
O-5	3	1	2	10	--	44	38-50
O-6	0	--	--	--	--	--	--
O-7	5	1	4	8	--	93	--
O-8	2	1	1	10	--	93	--
O-9	5	0	5	--	--	46	35-58

APPENDIX 1

TABLE 8 CONTINUED

DISTRIBUTION OF AIR-DAYS FOR THE EXPERIMENTAL ANIMALS

POSTOPERATIVE SESSIONS 14-22

FROG	NUMBER AIR-DAYS	AIR-HABITUATED DAYS	AIR-NONHABITUATED DAYS	NUMBER OF TRIALS TO HABITUATE DURING HABITUATED DAYS MEDIAN	HABITUATED DAYS RANGE	NONHABITUATED DAYS MEDIAN	NONHABITUATED DAYS RANGE
			SHAM OPERATED				
S-1	0	--	--	--	--	--	--
S-2	1	0	1	--	--	32	--
S-3	6	3	3	22	11-48	69	32-83
S-4	3	2	1	24	17-31	78	--
S-5	0	--	--	--	--	--	--
S-6	0	--	--	--	--	--	--
S-7	7	2	5	41	40-42	49	46-61
S-8	3	2	1	11.5	5-18	35	--

APPENDIX 1
TABLE 9

HABITUATION EXPERIMENT

AIR RESPONSES ON AIR-DAYS
STARVATION CONTROLS

	PERIOD I				PERIOD II			
FROG	Day	# A Responses	Trial # of A's	# to Habituate	Day	# A Responses	Trial # of A's	# to Habituate
J-1	a	83	1,12-93	93	a	12	43,54-58, 70-75	84
	b	14	1,32-44	65				
	c	40	26-43,54-67, 86-93					
	d	13	20-32	51				
J-2	--	--	--	--	--	--	--	--
J-3	--	--	--	--	--	--	--	--
J-4	a	11	35,48-57	78	a	14	20,21,37, 56-60,67, 80-82,92, 93	93
J-5	--	--	--	--	--	--	--	--

APPENDIX 1

TABLE 9 CONTINUED

HABITUATION EXPERIMENT

AIR RESPONSES ON AIR DAYS

STARVATION CONTROLS

PERIOD III

FROG	DAY	# A RESPONSES	Trial of A's	# to Habituate
J-1	a	6	32,46-50	60
J-2	--	--	--	--
J-3	--	--	--	--
J-4	a	11	53-58,88, 89,91-93	93
J-5	--	--	--	--

For explanation of "Periods I, II and III" see footnote of Table 1, Appendix 1.

APPENDIX 1

TABLE 10

RELATIONSHIP BETWEEN AIR-DAYS AND HABITUATION (NUMBER OF DAYS ARRANGED FOR FISHER EXACT PROBABILITY TEST ANALYSIS)

		PREOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 14-22	
		HABITUATED		HABITUATED		HABITUATED	
		NO	YES	NO	YES	NO	YES
		TELENCEPHALON ABLATED					
T-1							
AIR DAYS:	YES	0	0	0	0	0	0
	NO	1	8	0	9	0	9
T-2	Y	0	0	1	0	6	2
	N	0	9	0	8	0	1
T-3	Y	1	0	0	3	0	6
	N	5	3	0	6	0	3
T-4	Y	2	2	6	0	4	3
	N	0	5	2	1	2	0
T-5	Y	0	1	0	1	4	2
	N	0	8	0	8	0	3
T-6	Y	0	0	0	1	0	0
	N	2	7	0	8	1	8
T-7	Y	1	1	0	0	0	0
	N	4	3	1	8	0	9
T-8	Y	3	2	2	0	0	0
	N	0	4	0	7	0	9
T-9	Y	0	0	0	0	1	1
	N	0	9	0	9	0	0
T-10	Y	1	0	0	0	0	0
	N	1	7	1	8	1	8
T-11	Y	0	3	1	0	0	0
	N	0	6	0	8	0	9

APPENDIX 1

TABLE 10 CONTINUED

RELATIONSHIP BETWEEN AIR-DAYS AND HABITUATION
(NUMBER OF DAYS ARRANGED FOR FISHER EXACT PROBABILITY TEST ANALYSIS)

		PREOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 14-22	
		HABITUATED		HABITUATED		HABITUATED	
		NO	YES	NO	YES	NO	YES
		OLFACTORY BULB ABLATED					
O-1	AIR DAYS: YES	0	0	3	3	5	1
	NO	0	9	0	3	0	3
O-2	Y	0	1	0	0	0	0
	N	0	8	0	9	0	9
O-3	Y	0	0	0	0	0	0
	N	1	8	0	9	0	9
O-4	Y	3	2	4	1	3	1
	N	3	1	0	4	2	3
O-5	Y	0	2	2	2	2	1
	N	0	7	0	5	2	4
O-6	Y	0	0	0	0	0	0
	N	3	6	0	9	0	9
O-7	Y	1	1	1	1	4	1
	N	1	6	0	7	0	4
O-8	Y	0	0	0	1	1	1
	N	1	8	0	8	0	7
O-9	Y	0	1	0	0	5	0
	N	0	8	0	9	1	3
		SHAM OPERATED					
S-1	Y	2	0	1	0	0	0
	N	0	7	0	8	0	9
S-2	Y	1	0	6	0	2	0
	N	0	8	0	3	0	7
S-3	Y	0	0	3	3	3	3
	N	0	9	0	3	0	3
S-4	Y	1	3	1	1	1	2
	N	0	5	0	7	0	6

APPENDIX 1

TABLE 10 CONTINUED

RELATIONSHIP BETWEEN AIR-DAYS AND HABITUATION (NUMBER OF DAYS ARRANGED FOR FISHER EXACT PROBABILITY TEST ANALYSIS)

		PREOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 1-9		POSTOPERATIVE SESSIONS 14-22	
		HABITUATED		HABITUATED		HABITUATED	
		NO	YES	NO	YES	NO	YES
		SHAM OPERATED					
S-5							
AIR DAYS:	YES	0	0	0	0	0	0
	NO	0	9	0	9	0	9
S-6	Y	0	0	0	0	0	0
	N	1	8	0	9	0	9
S-7	Y	4	1	6	1	5	2
	N	0	4	0	2	1	1
S-8	Y	4	0	0	0	1	2
	N	0	5	0	9	0	6

APPENDIX 2

TABLES FOR THE OPTOMOTOR EXPERIMENT

APPENDIX 2

TABLE 1

OPTOMOTOR EXPERIMENT

LATENCY OF HEAD FOLLOWING (SECS)

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	7.25	3.5-16	5.00	3.5-9
T-13	3.00	1-4	1.50	1-6
T-14	2.50	1.5-3.5	2.00	1-15
T-17	3.00	1-11.5	1.75	1-3
		OLFACTORY BULB ABLATED		
O-10	4.25	3-7	3.25	2-6.5
O-12	2.50	1.5-14	2.00	1-8
O-14	3.00	1.5-6	4.25	2.5-16.5
		SHAM OPERATED		
S-9	3.00	2-9	3.00	1.5-9.5
S-10	2.00	1.5-4.5	2.75	1.5-7.5

APPENDIX 2

TABLE 2

OPTOMOTOR EXPERIMENT

NUMBER OF TEN DEGREE MOVEMENTS

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	2.0	0-5	2.5	0-46
T-13	8.5	1-7	6.0	1-44
T-14	3.0	0-11	3.5	0-17
T-17	5.0	0-13	66.0	19-99
		OLFACTORY BULB ABLATED		
O-10	1.0	0-7	3.5	0-22
O-12	9.0	5-28	26.5	7-77
O-14	6.0	0-12	2.0	0-13
		SHAM OPERATED		
S-9	3.5	0-6	5.0	1-10
S-10	4.0	0-15	5.5	3-10

APPENDIX 2

TABLE 3

OPTOMOTOR EXPERIMENT

DURATION OF TEN DEGREE MOVEMENTS (SECS)

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	29.0	7-54	5.7	2-52
T-13	4.3	2-12	6.6	3-18.5
T-14	14.7	2-30.5	8.5	3-30
T-17	13.0	3-19	2.0	2-3
		OLFACTORY BULB ABLATED		
O-10	9.5	4-55	7.3	2-36.5
O-12	9.4	2-16	3.9	2-5
O-14	6.9	2-15	12.1	3.30
		SHAM OPERATED		
S-9	22.4	7-61	17.3	5-46.5
S-10	10.2	3-20	7.8	2-17

APPENDIX 2

TABLE 4

OPTOMOTOR EXPERIMENT

NUMBER OF "STOPS"

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	2	0-2	1	0-2
T-13	0	0-1	0	0-1
T-14	0	0-2	0	0-2
T-17	1	0-2	0	---
		OLFACTORY BULB ABLATED		
O-10	2	1-2	1	0-2
O-12	1	0-2	0	0-1
O-14	0	0-2	1	0-2
		SHAM OPERATED		
S-9	1	0-2	1	0-2
S-10	1	0-2	1	0-2

APPENDIX 2

TABLE 5

OPTOMOTOR EXPERIMENT

DURATION OF "STOPS" (SECS)

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	146	98-169.5	135.5	90-171.5
T-13	119	---	105	91-110
T-14	114.5	96-167	112	107-162
T-17	125	104-177.5	---	
		OLFACTORY BULB ABLATED		
O-10	155.5	117.5-170.5	151	96-169
O-12	115.8	95-140.5	101	91-153
O-14	121	107-144	143.5	103-161
		SHAM OPERATED		
S-9	151.5	110-169	110	101.5-161
S-10	128	115-149	125	102-157.5

APPENDIX 2

TABLE 6

OPTOMOTOR EXPERIMENT

DURATION OF TIME SPENT AT SURFACE (SECS)

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	0	---	0	0-34
T-13	45.5	4-123.5	64.25	0-105
T-14	84.8	0-180	24.25	0-63
T-17	3.0	0-85	8.5	2-15
		OLFACTORY BULB ABLATED		
O-10	0	0-0.5	0	0-2.5
O-12	0.25	0-73	7.5	0-76.5
O-14	16.5	0-128	0.25	0-1
		SHAM OPERATED		
S-9	0.5	0-92	0.5	0-26
S-10	34	0-175	2.25	0-51

APPENDIX 2

TABLE 7

OPTOMOTOR EXPERIMENT

NUMBER OF WHOLE BODY MOVEMENTS

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	0	0-2.5	1.38	0-6.5
T-13	10.75	5-17	0.75	1-4.5
T-14	2.25	0-10	3.5	1-5.5
T-17	3.50	0-11.5	2.75	12-18.5
		OLFACTORY BULB ABLATED		
O-10	0	0-1.5	0.50	0-5
O-12	1.25	0-9	10.25	3-16.5
O-14	4.0	0-10	1.75	0-5.5
		SHAM OPERATED		
S-9	1.0	0-3.5	2.75	1-5
S-10	2.75	0-6.5	4.75	0-12.5

APPENDIX 2

TABLE 8

OPTOMOTOR EXPERIMENT

DURATION OF WHOLE BODY MOVEMENTS (SECS)

	PREOPERATIVE SESSIONS 1-7		POSTOPERATIVE SESSIONS 1-7	
FROG	MEDIAN	RANGE	MEDIAN	RANGE
		TELENCEPHALON ABLATED		
T-12	2.0	1-2.5	2.4	1-5
T-13	2.25	1.6-3.1	3.4	1.5-4.6
T-14	2.4	1.7-3.8	3.15	1.1-7
T-17	2.2	1.7-4	1.9	1.3-3.1
		OLFACTORY BULB ABLATED		
O-10	3.0	3-4	2.4	1.5-4
O-12	2.0	1.1-6.5	1.9	1.1-3.1
O-14	2.0	1.3-8	3.0	1-5
		SHAM OPERATED		
S-9	3.9	1-5.8	2.2	1.5-6
S-10	3.2	1.9-8	2.7	1.9-4.2

APPENDIX 2

TABLE 9

OPTOMOTOR EXPERIMENT

NUMBER OF INITIAL DIRECTIONS OF WHOLE BODY MOVEMENTS IN THOSE SESSIONS SHOWING WHOLE BODY MOVEMENTS

	PREOPERATIVE SESSIONS 1-7						POSTOPERATIVE SESSIONS 1-7					
	S		Op		UD		S		Op		UD	
FROG	MEDIAN	RANGE	MEDIAN	RANGE	MEDIAN	RANGE	MEDIAN	RANGE	MEDIAN	RANGE	MEDIAN	RANGE
						TELENCEPHALON ABLATED						
T-12	0	0-1	0	0-4	0	0-1	1	0-8	0	0-9	0	---
T-13	11	6-21	8.5	4-16	0	0-3	4	1-8	2	0-5	0	0-2
T-14	3	0-12	2	0-8	0	---	3	2-11	2	1-6	0	0-2
T-17	4.5	0-16	1	0-6	0	0-1	21	14-30	9	3-14	0	0-6
						OLFACTORY BULB ABLATED						
O-10	0	0-3	0	0-1	0	---	1	0-7	1	0-3	0	0-1
O-12	2	0-13	1	0-5	0	0-3	13	6-23	3	2-9	3	0-11
O-14	5.5	0-12	1	0-7	0	0-3	1.5	1-8	1	0-3	0	---
						SHAM OPERATED						
S-9	1	0-5	0	0-3	0	---	3.5	1-6	1	0-6	0	0-1
S-10	4	1-12	1	0-5	0	0-2	10.5	0-18	3	0-9	0	0-3

KEY

S = The animal turned in the same direction as the drum was turning.

Op = The animal turned in the direction opposite to drum movement.

UD = The animal made an "up-down" movement not related to the direction of drum movement.

APPENDIX 3

TABLES FOR THE ESCAPE FROM SHALLOW WATER EXPERIMENT

APPENDIX 3

TABLE 1A

ESCAPE FROM SHALLOW WATER EXPERIMENT

LATENCY TO ESCAPE IN SECONDS

		PREOPERATIVE SESSIONS 1-6	POSTOPERATIVE SESSIONS 1-6
			TELENCEPHALON ABLATED*
FROG	SESSION		
T-12	1	191	300
	2	196	300
	3	157	300
	4	190	273
	5	246	300
	6	231	300
	Median	193.5	300
T-13	1	30	300
	2	28	300
	3	23	150
	4	23	300
	5	16	153
	6	21	143
	Median	23	216.5
T-14	1	23	273
	2	9	300
	3	20	300
	4	24	300
	5	14	27.5
	6	16	112
	Median	18	286.5
T-15	1	35	251
	2	52	152
	3	67	247
	4	37	106
	5	37	130
	6	29	
	Median	37	138.5
T-16	1	155	300
	2	115	259
	3	175	282
	4	57	195
	5	159	300
	6	141	282
	Median	148	282

APPENDIX 3

TABLE 1B CONTINUED

ESCAPE FROM SHALLOW WATER EXPERIMENT

LATENCY TO ESCAPE IN SECONDS

		PREOPERATIVE SESSIONS 1-6	POSTOPERATIVE SESSIONS 1-6
		OLFACTORY BULB ABLATED	
FROG	SESSION		
O-10	1	208	300
	2	241	287
	3	114	300
	4	247	300
	5	219	300
	6	185	300
	Median	213.5	300
O-11	1	86	95
	2	67	261
	3	67	243
	4	52	163
	5	47	126
	6	44	128
	Median	59.5	145.5
O-12	1	5	8
	2	55	18
	3	14	21
	4	21	15
	5	16	10
	6	5	16
	Median	17.5	15.5
O-13	1	62	60
	2	46	76
	3	57	72
	4	59	93
	5	52	55
	6	37	134
	Median	55.5	74
O-14	1	26	30
	2	59	32
	3	41	10
	4	38	28
	5	19	39
	6	12	43
	Median	32	31

APPENDIX 3

TABLE 1C CONTINUED

ESCAPE FROM SHALLOW WATER EXPERIMENT

LATENCY TO ESCAPE IN SECONDS

		PREOPERATIVE SESSIONS 1-6	POSTOPERATIVE SESSIONS 1-6
		SHAM OPERATED	
FROG	SESSION		
S-9	1	35	88
	2	50	197
	3	100	22
	4	88	26
	5	34	49
	6	60	13
	Median	55	39.5
S-10	1	9	74
	2	22	70
	3	34	24
	4	12	40
	5	20	26
	6	5	26
	Median	16	31

*There was a significant increase between the preoperative sessions and the postoperative sessions for the telencephalon-ablated animals (Wilcoxon signed ranks test, $p < .05$, one-tailed). Differences for the other groups were not statistically significant ($p > .05$).

	Preoperative Sessions		Postoperative Sessions	
	Median	Range	Median	Range
Olfactory bulb ablated group	55.5	17.5-213.5	74	15.5-300
Sham-operated group	35.5	16-55	35.3	31-39.5

APPENDIX 3

TABLE 2

ESCAPE FROM SHALLOW WATER EXPERIMENT

PROPORTION OF TIME SPENT SWIMMING

		PREOPERATIVE SESSIONS 1-6	POSTOPERATIVE SESSIONS 1-6
		TELENCEPHALON ABLATED	
FROG	SESSION		
T-12	1	.16	.36
	2	.31	.32
	3	.25	.41
	4	.32	.49
	5	.23	.33
	6	.31	.46
	Median	.28	.385
T-13	1	.57	---
	2	.61	---
	3	.63	.13
	4	.65	---
	5	.60	.05
	6	.60	.12
	Median	.62	.025
T-14	1	.74	.04
	2	.84	.06
	3	.59	.04
	4	.69	.12
	5	.64	.12
	6	.63	.18
	Median	.665	.09

APPENDIX 3

TABLE 2 CONTINUED

ESCAPE FROM SHALLOW WATER EXPERIMENT

PROPORTION OF TIME SPENT SWIMMING

PREOPERATIVE SESSIONS 1-6 POSTOPERATIVE SESSIONS 1-6

OLFACTORY BULB ABLATED

FROG	SESSION			
O-10	1		.15	.26
	2		.13	.16
	3		.14	.17
	4		.18	.19
	5		.27	.21
	6		.16	.24
		Median	.155	.20
O-11	1		.22	.45
	2		.33	.34
	3		.45	.38
	4		.56	.40
	5		.49	.48
	6		.52	.38
		Median	.47	.39
O-12	1		.62	.39
	2		.09	.24
	3		.37	.33
	4		.41	.32
	5		.26	.35
	6		.45	.31
		Median	.39	.53
O-13	1		.34	.39
	2		.39	.24
	3		.42	.33
	4		.33	.32
	5		.22	.35
	6		.35	.31
		Median	.345	.325
O-14	1		.24	.30
	2		.25	.32
	3		.27	.36
	4		.19	.29
	5		.39	.29
	6		.59	.17
		Median	.26	.295

APPENDIX 3

TABLE 2 CONTINUED

ESCAPE FROM SHALLOW WATER EXPERIMENT

PROPORTION OF TIME SPENT SWIMMING

		PREOPERATIVE SESSIONS 1-6	POSTOPERATIVE SESSIONS 1-6
		SHAM OPERATED	
FROG	SESSION		
S-9	1	.21	.22
	2	.16	.08
	3	.14	.21
	4	.15	.32
	5	.26	.24
	6	.14	.15
	Median	.185	.215
S-10	1	.47	.28
	2	.25	.27
	3	.13	.41
	4	.25	.34
	5	.30	.32
	6	.60	.38
	Median	.275	.335

A Kruskal-Wallis one-way ANOVA showed no significant differences ($p > .05$) among the operative groups for either the preoperative or thepostoperative periods.

APPENDIX 3

TABLE 3

ESCAPE FROM SHALLOW WATER EXPERIMENT
RESULTS OF EXTENSIVE PREOPERATIVE
REPETITION OF SESSIONS

	FIRST 6 SESSIONS			INTERVENING SESSIONS			LAST 6 SESSIONS		
FROG	NUMBER	MEDIAN	RANGE	NUMBER	MEDIAN	RANGE	NUMBER	MEDIAN	RANGE
T-15	35	37	29-67	28	20	12-28	32	22	14-48
	52			20			14		
	67			18			29		
	37			22			48		
	37			12			14		
	29			23			15		
				20					
				25					
				20					
				20					
				16					
				19					
				24					
				26					
				28					
T-16	155	149	57-175	61	51.5	33-112	49	44	25-67
	117			59			25		
	175			80			42		
	57			58			38		
	159			56			46		
	141			50			67		
				51					
				75					
				112					
				40					
				41					
				44					
				36					
				52					

A Wilcoxon test demonstrated that both animals showed a significant ($p<.05$, one-tailed) decrease in time to escape between the first and last six sessions.

REFERENCES

Alberts, J. 1974. Producing and interpreting experimental olfactory deficits, Physiol. and Behav. 12:657-670.

Andrew, R. 1974. Arousal and the causation of behavior, Behav. 51(3-4):135-163.

Aronson, L. 1948. Problems in the behavior and physiology of a species of African mouthbreeding fish, Trans. N.Y. Acad. Sci. 2, II:33-42.

Aronson, L. 1957. Reproductive and parental behavior. In Brown, M. (ed.), The Physiology of Fishes, Academic Press, N.Y.

Aronson, L. 1963. The central nervous system of sharks and bony fishes with special reference to sensory and integrative mechanism. In Gilbert, P. (ed.), Sharks and Survival, D.C. Heath, Boston, pp. 165-241.

Aronson, L 1967. Forebrain function in teleost fishes, Trans. N.Y. Acad. Sci ser. 2, 29(4): 390-396.

Aronson, L. 1970. Functional evolution of the forebrain in lower vertebrates. In Aronson, L., Tobach, E., Lehrman, D. and Rosenblatt, J. (eds.), Development and Evolution of Behavior, W. H. Freeman, San Francisco, pp. 75-107.

Aronson, L. and Herberman, R. 1960. Persistence of a conditioned response in the cichlid fish, Tilapia macrocephala, after forebrain and cerebellar ablation, Anat. Rec. 138:332.

Aronson, L. and Kaplan, H. 1963. Forebrain function in avoidance conditioning, Am. Zool. 3(4): 483-4.

Aronson, L. and Kaplan, H. 1965. Effect of forebrain ablation on the acquisition of a conditioned avoidance response in the teleost fish, Tilapia h. macrocephala, Am. Zool. 5(4):654.

Aronson, L. and Kaplan, H. 1968. Function of the teleostean forebrain. In Ingle, D. (ed.), The Central Nervous System and Fish Behavior, U. of Chicago Press, Chicago.

Aronson, L. and Noble, G. 1945. The sexual behavior of anura 2. Neural mechanisms controlling mating in the male leopard frog, Rana pipiens, Bull. Amer. Mus. Nat. Hist. 86(3):87-139.

Avila, V. and Frye, P. 1977. Feeding behavior in the African clawed frog, (Xenopus laevis Daudin), Herpetol. 33:152-161.

Bass, A., Pritz, M. and Northcutt, R. 1973. Effects of telencephalon and tectal ablations on visual behavior in the side-necked turtle, Podocnemis unifilis, Brain Res. 55:455-460.

Birukow, G. 1939. Untersuchungen über den optischen Drehnystagmus und über die Sehscharfe des Grasfrosches (Rana temporaria), Z. Vergl. Physiol. 25:92-142.

Brauth, S. and Karten, H. 1977. Direct accessory optic projections to the vestibulo-cerebellum: A possible channel for oculomotor control systems, Exp. Brain Res. 28:73-84.

Burgers, A. 1950. Optomotor reaction of Xenopus laevis, Physiol. Comparata et Oecologia 2:272-281.

Butz-Kuenzer 1957. Optische und labyrinthäre Auslösung der Lagerreaktionen beim Amphibien, Z. Tierpsych. 14:429-447.

Cain, D. 1974. The role of the olfactory bulb in limbic mechanisms, Psych. Bull. 81(10):654-671.

Channing, A. 1976a. Personal communication.

Channing, A. 1976. Life histories of frogs in the Namib Desert, Zool. Africana 11(2): 299-312.

Colombo, J. and Segura, E. 1972. Effects of estradiol on brain electrogram of the female toad Bufo grenum Hensel during the breeding season, Gen. Comp. Endocrin. 18:268-275.

Curnow, P. and Overmier, J. 1968. Failure to show associative loss as the source of avoidance deficits following forebrain ablation in goldfish, Proc. 76th Ann. Conv. APA.

Davis,R. 1975. Readiness to display in the paradise fish Macropodus opercularis (L.), Belontiidae: The problem of general and specific effects of social isolation, Behav. Biol. 15:419-433.

Davis, R., Kassel, J. and Schwagmeyer, P. 1976. Telencephalic lesions and behavior in the teleost, Macropodus opercularis: Reproduction, startle reaction and operant behavior in the male, Behav. Biol. 18:165-177.

Demski, L. and Knigge, K. 1971. The telencephalon and hypothalamus of the Bluegill (Lepomis macrochirus): Evoked feeding, aggressive and reproductive behavior with representative frontal sections, J. Comp. Neurol. 143:1-16.

Deuchar, E. 1972. Xenopus laevis and developmental biology, Biol. Rev. 47:37-112.

Deuchar, E.,1975. Xenopus: The South African Clawed Frog, John Wiley, London.

Dewsbury, D. and Bernstein, J. 1969. Role of the telencephalon in performance of conditioned avoidance responses by goldfish, Exp. Neurol. 23:445-456.

Dreyer, R. 1913. The "Plathander" (Xenopus laevis), Trans. Roy. Soc. Afr. 3:341-355.

Ebbesson, S. 1970. On the organization of central visual pathways in vertebrates, Brain Behav. Evol. 3:178-198.

Ebbesson, S. and Campbell, C. 1973. The organization of cerebellar efferents in the nurse shark (Ginglymostoma cirratum), J. Comp. Neurol. 152:233-254.

Ebbesson, S., Jane, J. and Schroeder, D. 1972. A general overview of major interspecific variations in thalamic organization, Brain, Behav. Evol. 6:92-130.

Ebbesson, S. and Ramsey, J. 1968. The optic tracts in two species of sharks (Galeocerdo cuvieri and Ginglymostoma cirratum), Brain Res. 8:36-53.

Eikmanns, K. 1955. Verhaltensphysiologische Untersuchungen uber den Beutefang und das Bewegunssehen der Erdkrote (Bufo bufo L.), Z. Tierpsych. 12:229-253.

Estes, R. 1975a. Fossil Xenopus from the Paleocene of South America and the zoogeography of pipid frogs, Herpetrol. 31:263-278.

Estes, R. 1975b. Xenopus from the Palaeocene of Brazil and its zoogeographic importance, Nature 254:48-51.

Estes, R. and Reig, O. 1973. The early fossil record of frogs: A review of the evidence, in Vial, J.,(ed.), Evolutionary Biology of the Anurans, U. Missouri Press, Columbia, Missouri, pp. 11-63.

Ewert, J. 1966. Auslosung des Beute-und Flacht-verhaltens durch elektrische Mittelhirn-Reizung bei der Erdkrote (Bufo bufo L.), Naturwiss. 53:589.

Ewert, J. 1967a. Aktivierung der Verhaltensfolge beim Beutefang der Erdkrote (Bufo bufo L.) durch electrische Mittelhirnreizung, Z. Vergl. Physiol. 54:455-481.

Ewert, J. 1967b. Elektrische Reizung des retinaten projecktionsfeldes im Mittelhirn der Erdkrote, (Bufo bufo L.), Pflugers Arch. ges. Physiol. 259-90-98.

Ewert, J. 1967c. Untersuchungen uber die Anteile zentralnervoser Aktionen an der taxisspezi-fischen Ermudung beim Beutefang der Erdkrote (Bufo bufo L.), Z. Vergl. Physiol. 57:263-298.

Ewert, J. 1970a. Aufnahme und Verarbeitung visueller Informationen im Beutefang-und Fluchtverharlten der Erdkrote (Bufo bufo L.) Verh. d. Dtsch. Zool. Ges. 64:218-226.

Ewert, J. 1970b. Neural mechanisms of prey-catching and avoidance behavior in the toad (Bufo bufo L.) Brain Behav. Evol. 3:35-56.

Ewert, J. 1971. Single-unit response of the toad (Bufo americanus) caudal thalamus to visual objects, Z. Vergl. Physiol. 74:81-102.

Ewert, J. and Birukow, G. 1965. Über den Einfluss des Centralnervensystems auf die Ermüdbarkeit der Richtbewegung im Beuteschema der Erdkröte (Bufo bufo L.), Naturwiss. 52: 68-69.

Ewert, J. and Borchers, H. 1971. Reaktionscharakteristik von Neuronen aus dem Tectum opticum und Subtectum der Erdkröte (Bufo bufo L.), Z. Vergl. Physiol. 71:165-189.

Ewert, J. and Harter, H. 1969. Der hemmende Einfluss gleichzeitig bewegter Beuteattrappen auf das Beutefangverhalten der Erdkröte (Bufo bufo L.), Z. Vergl. Physiol. 64:135-153.

Ewert, J. and Ingle, D. 1971. Excitatory effects following habituation of pre-catching activity in frogs and toads, J. Comp. Physiol. Psych. 77:369-374.

Ewert, J. and Rehn, B. 1969. Quantitative Analyze der Reiz-Reaktion Abeziehungen bei visuellem Auslösen des Fluchtvehaltens der Wechselkröte (Bufo viridis Laur), Behav. 35:212-234.

Farel, P. 1977. Modulation of response threshold in frog spinal cord: Dependence upon descending influences, Behav. Biol., in press.

Farel, P., Glanzman, D. and Thompson, R. 1973. Habituation of a monosynaptic response in vertebrate central nervous system: Lateral column-motoneuron pathway in isolated frog spinal cord, J. Neurophys. 36(6):1117-1130.

Fite, K. and Scalia, F. 1976. Central visual pathways in the frog, in Fite, K. (ed.), The Amphibian Visual System, Academic Press, N.Y., pp. 87-118.

Flood, N. 1975. Effect of forebrain ablation on long-term retention of an appetitive didscrimination by goldfish, Psych. Rept. 36:783-786.

Flood, N. and Overmier, J. 1971. Effects of telencephalic and olfactory lesions on appetitive learning in goldfish, Physiol. Behav. 6:35-40.

Flood, N., Overmier, J. and Savage, G. 1976. Teleost telencephalon and learning: An interpretive review of data and hypotheses, Physiol. Behav. 16:783-796.

Frank, A., Flood, N. and Overmier, J. 1972. Reversal learning inforebrain ablated and olfactory tract sectioned teleosts, Carassius auratus, Psychon. Sci. 26(3):149-151.

Frankzisket, L. 1963. Characteristics of instinctive behavior and learning in reflex activity of the frog, Anim. Behav. 11:318-324.

Frontera, J. 1952. A study of the anuran diencephalon, J. Comp. Neurol. 96:1-47.

Goodman, D. and Weinberger, N. 1973. Habituation in "lower" tetrapod vertebrates: Amphibia as vertebrate model systems, in Peeke, H. and Herz, M., Habituation vol. 1, Academic Press, N.Y., pp. 85-140.

Gordon, M. 1950. Fishes as laboratory animals, in Farris, E., (ed.), The Care and Breeding of Laboratory Animals, John Wiley and Sons, N.Y., pp. 345-449.

Gorner, P. 1973. The importance of the lateral line system for the perception of surface waves in the claw toad, Xenopus laevis Daudin, Experientia 29(3):295-296.

Greenberg, N. 1977. Forebrain function in Anolis carolenensis, lecture given at Amer, Mus. Nat. Hist.

Gruberg, E. and Ambros, V., 1974. A forebrain visual projection in the frog (Rana pipiens) Exper. Neurol. 44:187-197.

Gurdon, J. 1967. African clawed frogs, in Wilt., F., Wessels, H. and Normans, K.(eds.), Methods in developmental Biology, Th. Crowell, pp. 75-84.

Hall, W. 1972. Visual pathways to the telencephalon in reptiles and mammans, Brain Behav. Evol. 5:95-113.

Halpern, M., 1972. Some connections of the telencephalon of the frog, Rana pipiens, Brain Behan. Evol. 6:42-68.

Heimer, L. 1969. The secondary olfactory connections in mammals, reptiles and sharks, Ann, N.Y. Acad. Sci. 167:129-146.

Hemmer, H. and Köhler, S. Rhythmische Anderungen der Reaktion auf verschiedene olfaktorische Reise beim Krallenfrosch Xenopus laevis Daudin, Experientia 29(3):295-296.

Herrick, C.,1921. A sketch of the origin of the cerebral hemispheres, J. Comp. Neurol. 32:429-452.

Herrick, C. 1922. Functional factors in the morphology of the forebrain of fishes, in Libro en Honor de D.S. Ramon y Cajal, Book 1, La junta para el homenaje a Cajal, Madrid, 1922, pp. 143-203.

Herrick, C. 1933. The functions of the olfactory parts of the cerebral cortex, Proc. Nat. Acad. Sci. 19:7-14.

Herrick, C. 1948. The Brain of the Tiger Salamander, U. Chicago press, Chicago.

Hertzler, D. and Hayes, W. 1967. Cortical and tectal function in visually guided behavior of turtles, J. Comp. Physiol. Pshych. 63:444-447.

Hertzler, D. and Hayes, W. 1969. Effects of monocular vision and midbrain transection on movement detection in the turtle, J. Comp. Physiol. Psych. 67:473-478.

Hoffman, A. and Lico, M. 1972a. Autonomic and motor responses to electrical stimulation of the toad's septum, Physiol. Behav. 8:1035-1037.

Hoffmann, A. and Lico, M. 1972b. Autonomic and motor responses to electrical stimulation of the toad's brain, Physiol. Behav. 8:1039-1043.

Hughlings Jackson, J., In Taylor 1958. Selected Writings of John Hughlings Jackson, Basic Books, N.Y.

Hutchison, J. 1965. Investibations on the neural control of clasping and feeding in Xenopus laevis (Daudin), Behav. 24:47-66.

Inger, R. and Marx, H. 1961. The food of amphibians, Expl. du Parc National de L'Umpemba, Mission G.F. de Witte 64:1-81.

Ingle, D. 1971. Prey-catching behavior of anurans toward moving and stationary objects, Vision Res. Supp. 3:447-456.

Ingle, D.,1973. Evolutionary perspectives on the function of the optic tectum, Brain.Behav. Evol. 8:211-237.

Janzen, W. 1933. Untersuchungen über Grosshirn-funktionen des Goldfisches, Jahrb. Abt. Allg. Zool. Physiol. 52(4):591-628.

Jarvik, E. 1960. Theories de L'Evolution des Vertebres, Masson, Paris.

Jurgens, J. 1971. The morphology of the nasal region of amphibia and its bearing on the phylogeny of the group, Annale Universiteit von Stellenbosch 46(2):1-146.

Kalk, M. 1960. Climate and breeding in Xenopus laevis So. Afr. J. Sci. 56:271-276.

Kamrin, R. and Aronson, L. 1954. The effects of forebrain lesions on mating behavior in the male platyfish, Xiphophorus maculatus, Zoolog. 39(4): 133-140.

Kaplan, H. and Aronson, L. 1967. Effect of forebrain ablation on the performance of a conditioned response in the teleost fish, Tilapia h. macro-cephala, Anim. Behav. 15(4):436-446.

Kaplan, H. and Aronson, L. 1969. Function of the forebrain and the cerebellum in the acquisition and performance of a conditioned avoidance response in the teleost fish, Tilapia h. macrocephala, Bull. Amer Mus. Nat. Hist. 142(2):141-208.

Karamian, A., Vesselkin, N., Belekhova, M. and Zagorulko, R. 1966. Electrophysiological characteristics of tectal and thalamo-cortical divisions of the visual system in lower vertebrates, J. Comp. Neurol. 127(4):559-576.

Kassel, J., Davis, R. and Schwagmeyer, P. 1976. Telencephalic lesions and behavior in the teleost, Macropodus opercularis: Further analysis of reproductive and operant behavior in the male, Behav. Biol. 18:179-188.

Keating, E, Kormann, L. and Horel, J. 1970. The behavioral effects of stimulating and ablating the reptilian amygdala (Caiman sclerops), Physiol. Behav. 5:55-59.

Kicliter, E.,1973a. Flux, wavelength and movement discrimination in frogs: Forebrain and midbrain contributions, Brain, Behav. Evol. 8:340-365.

Kicliter, E. 1973b. The forebrain visual system of the frog (Rana pipiens), Diss. Abstr. Intl. B, 34(6):2420B.

Kimble, D. and Ray, R. 1965. Reflex habituation and potentiation in Rana pipiens, Anim. Behav. 13:530-533.

Klüver, H. and Bucy, P., 1937. "Psychic blindness" and other symptoms following bilateral temporal lobectomy in rhesus monkeys, Am. J. Physiol. 119:352-353.

Kokoros, J. 1974. Efferent connections of the telencephalon in the toad, Bufo marinus, and the tiger salamander, Ambystoma tigrinum, Diss. Abstr. B35(2):644B.

Kunstadt, L. 1969. The vertebrate infranuclear optic pathway, unpublished Master's Thesis, New York University.

Lázár, G. 1969. Efferent pathways of the optic tectum in the frog, Acta. Biol. Acad. Sci. Hung. 20:171-183.

Lázár, G. 1973. Role of the accessory optic system in the optokinetic nystagmus of the frog, Brain Behav. Evol. 5:443-460.

Letvin, J., Maturana, H., McCulloch, W. and Pitts, W. 1959. What the frog's eye tells the frog's brain, Proc. Inst. Rad. Eng. 47:1940-1951.

Luna, L. (ed.), 1968. Manual of Histologic Staining of the Armed Forces Institute of Pathology, McGraw-Hill, N.Y.

Lynch, J. 1973. The transition from archaic to advanced frogs, in Vial, J., Evolutionary Biology of the Anurans, U. Missouri Press, Columbia, Missouri, 1973.

McGowan, B. Hankins, W. and Garcia, J. 1972. Limbic lesions and control of the internal and external environment, Behav. Biol. 7(6):841-852.

Mahrdt, C. and Knefler, F. 1972. Pet or pest? Envir. Southwest #446:2-5.

Mahrdt, C. and Knefler, F. 1973. The clawed frog-again, Envir. Southwest #450:1-3.

Maturana, H., Lettvin, J., McCulloch, W. and Pitts, W. 1960. Anatomy and physiology of vision in the frog (Rana pipiens), J. Gen. Physiol. 43(suppl.): 129-175.

Meyer, W. and Wagener, K. 1974. Ein Hinweis zum Maximallebensalter von Xenopus laevis (Daudin), Sitzungsberichte der Gesellschaft Naturforschender Freunde zu Berlin 12:71-72.

Morlock, H. 1972. Behavior following ablation of the dorsal cortex of turtles, Brain Behav. Evol. 5:256-263.

Muntz, W. 1962a. Microelectrode recordings from the diencephalon of the frog (Rana pipiens) and a blue-sensitive system, J. Neurophysiol. 25:699-711.

Muntz, W. 1962b. Effectiveness of different colors of light in releasing the positive phototactic behavior of frogs, and a possible function of the retinal projections to the diencephalon, J. Neurophysiol. 25:712-720.

Nevo, E. 1968. Pipid frogs from the early cretaceous of Israel and pipid evolution, Bull. Mus. Comp. Zool., Harvard Univ. 136(8):255-318.

Noble, G. 1936. Function of the corpus striatum in the social behaviour of fishes, Anat. Rec. 64(3)suppl:34.

Schaeffer, B. 1969. Adaptive radiation of the fishes and the fish-amphibian transition, Ann. N.Y. Acad. Sci. 167:5-17.

Schmajuk, N. and Segura, E. 1971. Modelo del comportaniento sexual de los anuroa, Revta. Soc. Argent. Biol. 47:40-56.

Schroeder, D. and Ebbesson, S., 1974. Nonolfactory telencephalic afferents in the nurse shark (Ginglymostoma cirratum), Brain, Behav. Evol. 9:121-155.

Segaar, J. 1960. Ethophysiological experiments with male Gasterosteus aculeatus, in Tower, D. and Schade. J., Structure and Function of the Cerebral Cortex, Elsevier, Amsterdam, pp. 301-305.

Segaar, J. 1961. Telencephalon and behavior in Gasterosteus aculeatus, Behav. 18:256-287.

Segaar, J. 1965. Behavioural aspects of degeneration and regeneration in fish brain: A comparison with higher vertebrates, Prog. Brain Res. 14:143-231.

Segaar, J. and Nieuwenhuys, R. 1963. New ethophysiological experiments with male Gasterosteus aculeatus, with anatomical comment, Anim. Behav. 11:331-344.

Segura, E. and de Castello, G. 1969. Condicionamento respondente y operante en el Bufo arenarum: Papel de los hemisferios, IX Congreso de la Asociacion Latinoamericana de Cs. Fisiologicas, Belo Horizonte, Brazil, Res. Comunic.:14-15.

Segura, E., Colombo, J. and de Hardy, D., Effects of castration on brain electrogram of the female toad Bufo arenarum Hensen, Gen. Comp. Endocrin. 16:298-303 (1971).

Segura, E., de Juan, A., Colombo, J. and Kacelnik, A. 1971. The sexual clasp as a reticularly controlled behavior in the toad, Bufo arenarum Hensel, Physiol. Behav. 7: 157-160.

Shapiro, H. and Goodman, D., 1969. Motor functions and their anatomical basis in the forebrain and tectum of the alligator, Exp. Neurol. 24:187-195.

Riss, W. and Jakway, J. 1970. A perspective on the fundamental retinal projections of vertebrates, Brain Behav. Evol. 3:30-35.

Riss, W., Pedersen, R., Jakway, J. and Ware, C., 1972. Levels of function and their representation in the vertebrate thalamus, Brain Behav. Evol. 6:26-41.

Rubinson, K. 1968. Projection of the optic tectum of the frog, Brain Behav. Evol. 1:529-561.

Rubinson, K. and Colman, d. 1972. Designated discussion: A preliminary report on ascending thalamic afferents in Rana pipiens, Brain Behav. Evol. 6:69-74.

Russell, I.,1971. The role of the lateral-line efferent system in Xenopus laevis, J. Exp. Biol. 54:621-641.

Savage, G. 1971. Behavioral effects of electrical stimulation of the telencephalon of the goldfish, Carassius auratus, Anim. Behav. 19:661-668.

Savage, J. 1973. The geographical distribution of frogs: patterns and predictions, in Vial, J., ed., Evolutionary Biology of the Anurans, Univ. Missouri Press, Columbia, Missouri, pp. 351-445.

Savage, G. and Swingland, I. 1969. Positively reinforced behaviour and the forebrain in goldfish, Nature 221:878-879.

Scalia, F. and Colman, D. 1975. Identification of telencephalic afferent thalamic nuclei associated with the visual system of the frog, Soc. for Neurosci. Abstr. #65, 1975.

Scalia, F. and Fite, K. 1974. A retinotopic analysis of the central connection of the optic nerve in the frog, J. Comp. Neurol. 158:455-478.

Scalia, F. and Gregory, K. 1970. Retinofugal projections in the frog: Location of the post-synaptic neurons, Brain Behav. Evol. 3:17-30.

Scalia, F., Knapp, H., Halpern, M. and Riss, W. 1968. New Observations on the retinal projection in the frog, Brain Behav. Evol. 1:324-353.

Schaeffer, B.,1965. The rhipidistian-amphibian transition, Amer. Zool. 5:267-276.

Shapiro, S., Shuckman, H., Sussman, D. and Tucker, A. 1974. Effects of telencephalic lesions on the gill cover response of Siamese fighting fish, Physiol. Behav. 13:749-755.

Shaw, E. and Sherman, R. 1971. The optomotor response of a forebrainless fish, Tilapia mossambica, American Mus. Novitates, #2446:1-6.

Siegel, S., Nonparametric Statistics for the Behavioral Sciences, McGraw-Hill, N.Y., 1956.

Sikharulidze, N. 1972. Functions of the cerebellum and forebrain in the behavior of turtles (Emys orbicularis and Clemmys caspica), translated from Doklady Nauk SSSR 205(5):1258-1260 by Consultants Bureau of Plenum press.

Szarski, H., 1962. The origin of amphibia, Quart. Rev. Biol. 37:189-211.

Szekeley, G. 1971. The mesencephalic and diencephalic optic centers in the frog, Vision Res. 3(suppl.): 269-279.

Terzian, H. and Ore, G. 1955. Syndrome of Kluver and Bucy reproduced in man by bilateral removal of the temporal lobes, Neurol. 5:373-380.

Thompson, R. and Spencer, W. 1966. Habituation: a model phenomenon for the study of neuronal substrates of behavior, Psych. Rev. 173:16-43.

Tinsley, R. 1973. Studies on the ecology and systematics of a new species of clawed toad, the genus Xenopus from western Uganda, J. Zool., Lond., 169:1-27.

Tinsley, R. 1975. The morphology and distribution of Xenopus vestitus (Anura: Pipidae) in Central Africa, J. Zool., Lond., 175:473-492.

Trachtenberg, M. and Ingle, D. 1974. Thalamotectal projections in the frog, Brain Res. 79: 419-430.

Vesselkin, N. Agayan, A. and Nomokonova, L. 1971. A study of thalamo-telencephalic afferent systems in frogs, Brain Behav. Evol. 4:295-306.

Wenzel, B., 1974. The olfactory system and behavior, in DiCara, L. (ed.), Limbic and Autonomic Nervous System Research, Plenum, N.Y., pp. 1-40.