Retching Responses in *Caiman sklerops* Elicited by Central Nervous Stimulation* (33459)

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The control of emesis has been demonstrated to reside in a vomiting center (1) and a chemoreceptor trigger zone (2) in the medulla. However, the regulation of these medullary structures by the higher centers is still rather hazy. Routinely, neither hypothalamic (3, 4) nor cerebral cortex (5) stimulation has been shown to evoke vomiting. The present research is a continuation of observations noted by this investigator while studying somatic movements in response to electrical stimulation (6).

Material and Methods. Twenty-seven *Caiman sklerops* were anesthetized by placing them in a refrigerator at an average temperature of 7° for 15–20 min. The skin over that area of the skull to be opened was removed and a craniotomy was performed. A small incision in the dura readily exposed the brain. Monopolar electrodes were implanted in forebrain regions and in the optic tectum. These electrodes were stainless steel wire 60–90 μ in diameter encased in PE 10 polyethylene tubing. The electrodes were kept in

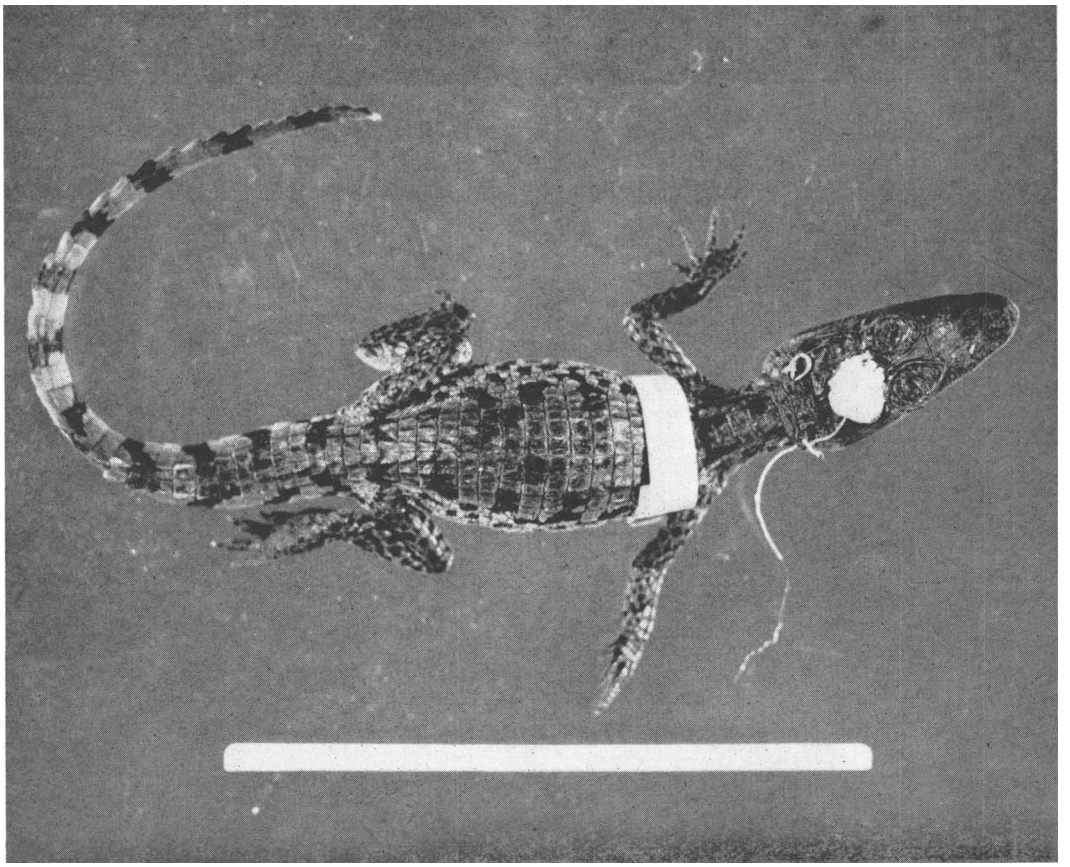

FIG. 1. *Caiman sklerops* with implanted electrode.

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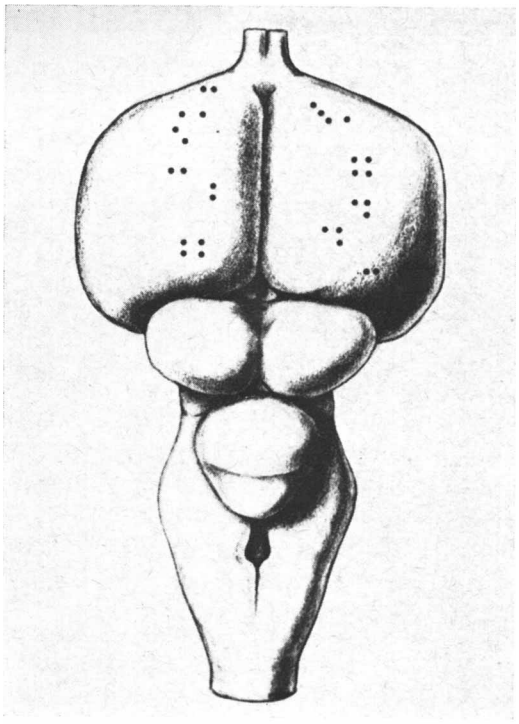

FIG. 2. Loci in the striatum that evoked retching.

place and the skull was sealed with dental cement (Fig. 1). The position of each electrode was verified histologically with the thionin stain. Two to five electrodes were implanted in some of the *Caiman*. A stainless steel indifferent electrode was implanted into the neck muscles and sutured to the skin. An "ident-a-band" was placed around the neck of each *Caiman* for identification purposes.

The stimulation studies were always carried out on intact, unanesthetized and unrestrained *Caiman* in which electrodes had been implanted 4–5 days previously. Electrical stimulation was delivered by a Montgomery–Ward (7) constant current, 60 cycle, sine-wave stimulator. The intensity of the current delivered in these experiments was in the range of 0.05–0.50 mA for a duration of 5–10 sec with a 30-min interval between each stimulation. The responses of the *Caiman* to these electrical stimulations were observed visually and recorded by the experimentor while the animal was moving freely in a glass aquarium.

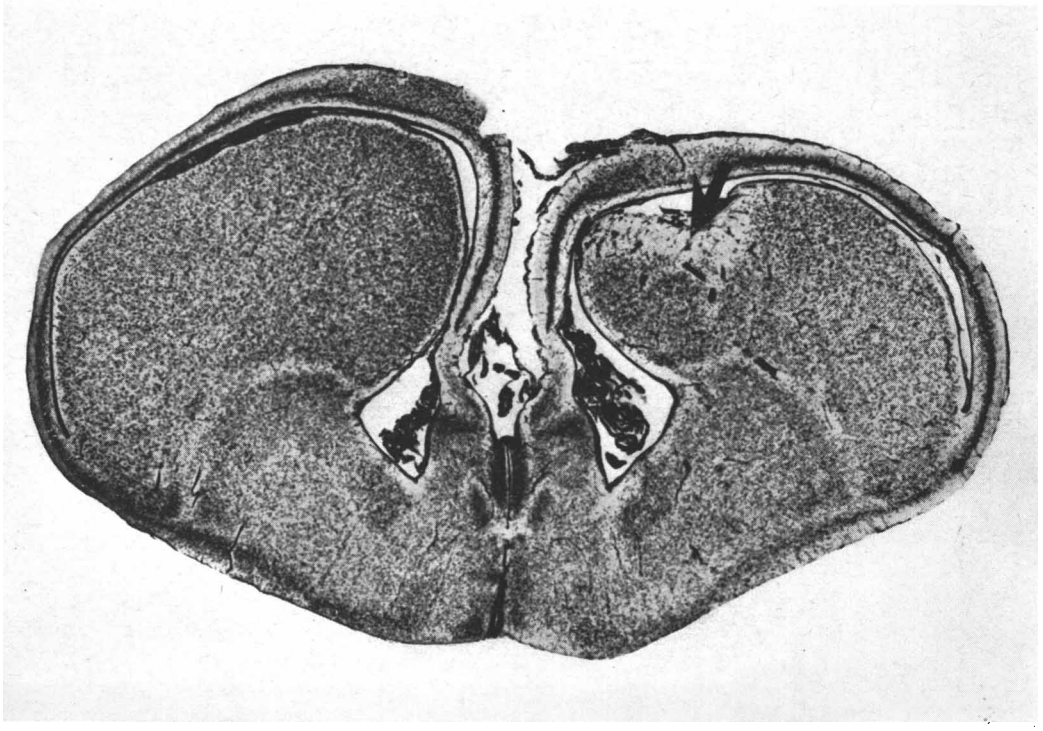

FIG. 3. Histological representation of a locus in the striatum that evoked retching.

TABLE I. Retching Responses in *Caiman sklerops* Elicited by Central Nervous Stimulation.

Region stimulation	No. of loci	No. of Caiman	Stimulus strengths (mA)	Stimulus duration (sec)	Latent period for onset of retching (min)	Incidence of retching
Dorsolateral region of striatum	34	16	0.05–0.40	5–10	2–5	30
Dorsolateral region of striatum without dorsal pallium, hippocampus and pyriform cortices	6	5	0.20–0.50	5–10	2–5	6
Dorsal pallium	20	15	0.10–0.50	5–10	—	0
Hippocampal cortex	6	5	0.10–0.50	5–10	—	0
Pyriform cortex	7	5	0.10–0.50	5–10	—	0
Optic tecta	14	8	0.10–0.50	5–10	—	0

Results. Electrical stimulation of 30 loci in the dorsolateral region of the striatum in 16 *Caiman* (Figs. 2, 3) evoked, after a 1–5-sec latent period, locomotion to the ipsilateral side which lasted as long as the stimulation continued. After a 2–5-min latent period, the *Caiman* would raise up on their forelimbs and rhythmic abdominal movements would begin that ended in retching. The position of the electrode in the rostral, middle, or caudal portions of the dorsolateral area had no observable effect on the stimulus threshold or on the quality of the retching (Table I).

In each of five *Caiman* most of the dorsal pallium, part of the hippocampal cortex, and part of the pyriform cortex were removed unilaterally by suction and six electrodes were placed in the underlying striatal region. A period of 4–5 days were allowed for recovery. No visible motor deficits were observed. The threshold for retching in this experimental group was slightly raised. Stimulation of the underlying striatal region evoked an immediate ipsilateral locomotion that was followed, after a 2–5-min latent period, by retching (Table I).

No retching responses could be elicited by electrical stimulation of loci in the dorsal pallium (20 loci in 15 *Caiman*), the hippocampal cortex (six loci in four *Caiman*) the pyriform cortex (seven loci in five *Caiman*) or the optic tecta (14 loci in eight *Caiman*) (Table I).

Discussion and Summary. In the present study on *Caiman*, the lack of retching observed when the dorsal pallium, the hippocampal cortex or the pyriform cortex was electrically stimulated agreed with the observations of Eliasson (5) in which a systematic electrical exploration of the entire cerebral cortex in cat elicited no vomiting. Eliasson also recorded that stimulation of the orbital surface of the cerebrum resulted in gastric motor inhibition while Borison and associates (8) reported that the emetic effect of pilocarpine depended upon the integrity of this part of the frontal cortex. However, vomiting by direct electrical stimulation of the cerebral cortex has not been encountered.

In this study, electrical stimulation of the striate region of the unanesthetized *Caiman* caused retching after a 2–5-min latent period. These emetic responses to electrical stimulation could be produced with regularity, whereas, Hess (9) reported occasional vomiting upon electrical stimulation of thalamic and hypothalamic regions in the unanesthetized cat. Hess also noted a latent period of seconds to a couple of minutes from the time of stimulation until the onset of the emesis.

Thus, while this study demonstrates the influence of the forebrain upon the hindbrain in a poikilothermic species, no definite answer is forthcoming on the long latency observed between forebrain stimulation and retching. An intervening process is undoubtedly involved in this long latent period; how-

ever, the data seems to exclude the dorsal pallium, the hippocampal cortex and the pyriform cortex from having a direct role in the response.

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Immunoglobulin in Fetal Lamb Lung and Its Fluid* (33460)

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The fetal lamb lung was shown to produce a fluid (1) which completely fills the airway passages before the onset of respiration. Surface active material is normally present in fetal lung fluid before the first breath (2, 3). Lung extracts of patients dying with hyaline membrane disease have been shown to lack surface activity (4, 5). Thus, the physicochemical properties of the fetal lung fluid may be important during the onset of respiration. It seems possible then, that under conditions of fetal distress, changes in the composition of fetal lung fluid may occur, favoring the formation of hyaline membranes. Fetal lung fluid contains at least five protein fractions (6), including a globulin with gamma mobility, as detected by immunoelectrophoresis. The purpose of the present study was to determine the immunological properties and the source of the gamma globulin fraction previously described.

Materials and Methods. Eleven lambs at different gestational ages, ranging in weight from 1.4 to 5.2 kg were delivered by cesarean section. Special precautions were taken to maintain the placental circulation intact (2). The methods for handling and monitoring the mother and the fetus were identical to those previously described (6). Following removal of the lung fluid, the lambs were sacrificed and multiple lung biopsies were performed on six fetuses. In addition, sections were made of other fetal organs (i.e., thymus, liver, parotid, and kidney) as well as the lung in one.

Lung fluid. Immediately after collection, specimens were frozen without addition of preservatives and maintained at —20°. The total amount of fetal lung fluid collected was 323 ml. Prior to pooling and lyophilization, 2 ml of each specimen were removed and set aside for vacuum ultrafiltration concentration to approximately 3 g/100 ml (7). These 2 ml samples were later pooled. Prior to concentration, the total protein of the nonlyophilized samples was determined (8) to be 12.7 mg/100 ml.[1] Lyophilization was performed in the remaining 300 ml which rendered 4 ml of total protein residue.

Lung biopsy. Frozen biopsies were stored at —75°. Frozen sections, 4 μ in thickness, were cut on a Harris-International cryostat

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[1] The amount of total protein in tracheal fluid reported by one of us (FHA) previously was erroneously calculated and should have been about 0.1 the value, i.e., 30 mg/100 ml. (2).