College, Agra, for providing us facilities in this work.

Department of Zoology, Agra College, Agra, August 12, 1977.

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VOMITING RESPONSE IN THE INDIAN FROGS *RANA TIGRINA* (DAUD) AND *RANA CYANOPHLYCTIS* (BOULENGER)

Introduction

RETROGRADE expulsion of stomach contents by vomiting is very well known in birds and in a large number of mammals. Pigeons of both sexes develop 'cytogenic crop glands' in their crops under the influence of *gonadotropic pituitary hormones*. Crop glands produce the 'pigeon's milk' which is regurgitated into the mouths of the chickens. Lower Chordates such as the lancelets (*Branchiostoma*) have the capacity to produce a '*rejection current*'. When after a couple of minutes of filtering the food from the water current, sand particles begin to obstruct the incoming water current. This rejection current is produced by the sudden contraction of transverse muscles of the atrial floor, causing the atrial opening to close and raising the atrial floor, thereby compressing the pharynx, which in turn ejects the water through the enterostome. There is a long evolutionary gap between the lower chordates and the birds and the mammals so far vomiting is concerned[1,6]. Attempts were made[8,12] to establish vomiting in the newts, frogs and toads. Both workers experimented upon fully pithed frogs after opening of the abdominal cavity. They also used subcutaneous and intragastric injections of certain emetics to bring about vomiting in frogs and toads in which brain and spinal cords were damaged. They, however, failed to induce vomiting in the newt *Triturus*. By extirpating the brain and the spinal cord[8] it was concluded that Anurans (at least *Rana* and *Bufo*) did not have a vomiting centre in the central nervous system The present author, by using well-fed, healthy and live frogs (*Rana tigrina* and *Rana cyanophlyctis*) has observed that like mammals, these frogs also have a vomiting control centre in the medulla and that vomiting is not uncommon in frogs in the experimental and probably also in the natural environs.

Material and Method

Frogs collected locally were well-fed and were given subcutaneous injections of various concentrations of emetics. Out of the stimulants and emetics tried (nicotine, atropine, suprarenal extract, sodium hydrate, apomorphine, and tartar emetic) best results were obtained with apomorphine and tartar emetic[4–5]. Pithed and chloroformed frogs were dissected for intragastric injections of emetics and local applications of emetics on the wall of the stomach.

Observations and Conclusions

Thirty frogs were given subcutaneous injections through the dorsal lymph sinus and quickly left under glass bell jars for observations. Another 20 frogs were first narcotized with 1 ml of 1% urethan solution injected subcutaneously through the dorsal lymph sinus and dissected soon after. The stomach was split open. Emetics and stimulants were applied directly over the mucosa of the stomach with the help of cotton buds to observe peristalsis of the gastric muscles. Observations are recorded in Table I.

It may now be noted that vomiting was brought about by apomorphine and tartar emetic. Stimulants like suprarenal extract, atropine, nicotine and sodium hydrate could induce peristalsis only. Effects of mild and small doses of apomorphine and tartar emetic injections were amazingly spontaneous. The injected frogs became highly excited, they opened and closed their mouths in quick successions and the tongue was shot out quite frequently. Throat exhibited strong gulping movements apparently to swallow the vomitus back down to the stomach. These responses continued for about 8 minutes, after which they slowed down. In about 30 minutes from the time of injection the frogs became unconscious. It was also observed that large and strong doses of apomorphine and tartar emetic inhibited vomiting, probably causing paralysis of muscles[5,7] and in the dogs.

In frogs with damaged brains, no vomiting could be induced either by injecting the emetics or by direct application of emetics on the gastric mucosa, but peristalsis was produced by a similar treatment with

TABLE I
Vomiting and gastric peristalsis in frogs

Chemical	Concentration	Quantity injected or applied	Observations
Suprarenal extract	0·1%	0·5 ml	no response,
	0·3%	0·5 ml	slow peristalsis in stomach,
	0·5%	0·5 ml	peristalsis increased,
	1·0%	1·0 ml	peristalsis inhibited, no vomiting
Atropine	0·1%	0·5 ml	no response,
	0·3%	0·5 ml	slow relaxation of stomach muscles,
	0·5%	0·5 ml	relaxations advanced,
	1·0%	1·0 ml	relaxations advanced, no vomiting
Nicotine	0·1%	0·5 ml	immediate peristalsis,
	0·3%	0·5 ml	persitalsis advanced,
	1·0%	1·0 ml	peristalsis inhibited, no vomiting
Apomorphine	0·1%	0·5 ml	spontaneous reverse peristalsis in stomach and vomiting,
	0·3%	0·5 ml	peristalsis and vomiting,
	0·5%	0·5 ml	vomiting advanced,
	1·0%	1·5 ml	inhibitory effect on vomiting, muscles of the entire body paralysed in 15 minutes
Tartar emetic	0·1%	0·5 ml	slow reverse peristalsis and vomiting,
	0·3%	0·5 ml	vomiting advanced,
	0·5%	0·5 ml	vomiting advanced,
	1·0%	1·5 ml	all muscular activity paralysed in 12 minutes
Sodium hydrate	1 in 20,000 parts	0·1 ml	spontaneous contractions in the stomach, but no vomiting,
	do.	0·5 ml	all muscular activity paralysed

stimulants like sodium hydrate, nicotine and atropine. In frogs whose spinal cord alone was damaged, vomiting but not peristalsis, could be induced with the same chemicals. This was suggestive of the fact that reflexes could travel through the vagus nerve supplying the stomach while spinal nerves III, IV and V supplying the stomach were inactivated thus peristalsis was out of the question. Lastly, in frogs whose entire central nervous system was damaged no treatment of emetics or stimulants could induce either vomiting or peristalsis.

These experiments suggest a central control of vomiting somewhere in the medulla of the brain. Several workers[1,3,4,7] have demonstrated a vomiting centre and a chemoreceptor trigger zone in the medulla of the dog. A similar vomiting centre if not also a chemoreceptor trigger zone is suggested in the frogs used by this author.

The author is grateful to the management of Lucknow Christian College for all the encouragement.

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PROLINE ACCUMULATION UNDER ZINC DEFICIENCY IN MUSTARD

A STRIKING accumulation of the imino acid proline occurs in many plants in response to water deficit[1,2] and salinity stress[3,4]. In the present paper it has been reported that there is accumulation of proline by deficiency of a micronutrient,