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The Effect of Denervation of Abdominal Musculature on the Prolapse of Esophagus and Stomach in the Vomiting of Frogs

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ABSTRACT

The role of abdominal musculature in the prolapse of esophagus and stomach into the buccal cavity during vomiting of the frog was studied. Vomiting of a normal frog produced by administration of a emetic, antimony potassium tartrate, was always accompanied by temporary prolapse of the esophagus and stomach into the mouth simultaneous to a strong contraction of the abdominal musculature. But a frog with a chronically denervated abdominal musculature failed to produce the prolapse of the esophagus and stomach, and expelling the food was seriously prevented, even if the emetic was administered. This indicates that the prolapse, which is the indispensable behavior of vomiting act in frogs, is induced by the rise of intra-abdominal pressure due to the strong contraction of abdominal musculature.

Introduction

On the occasion of vomiting, a normal frog always shows an anomalous phenomenon, that is, temporary prolapse of the esophagus and stomach into the mouth wide open (Tokita *et al.*, 1953; Noble, 1954; Hukuhara *et al.*, 1973). In the previous paper (Hukuhara *et al.*, 1973), we were led to the conclusion that the prolapse of the esophagus and stomach was assumed to be induced by the sudden rise of intra-abdominal pressure due to the strong contraction of the abdominal musculature. To observe more exactly the role of abdominal musculature in the prolapse of esophagus and stomach, the effect of denervation of abdominal wall on the vomiting of the frog was studied.

Materials and Methods

First, in the interests of clarity, the relationship between expulsion of food from the stomach and contraction of abdominal musculature was followed according to our previous paper (Hukuhara *et al.*, 1973) by means of an electromyogram recording of

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rectus abdominis muscle in Tonosamagaeru, *Rana nigromaculata*. The shematic representation of the method is shown in Fig. 1 described below.

Secondly, the following experiment on the vomiting of denervated frogs was performed, which is the major subject in this paper. Twenty bullfrogs (♀: 12, ♂: 8), *Rana catesbeiana*, weighing 282 g to 590 g were divided into three groups. In the first group, six of them were chronically denervated by cutting *N. spinalis IV*, *V*, *VI*, *VII* and a branch of *N. spinalis VIII* (*N. iliohypogastricus*) as well as branches of *N. spinalis III*, which innervate the abdominal wall (Gaupp, 1899). The spinal nerves *IV* through *VII* were cut out at their stems, leaving the central stems of 1.5 cm to 2.5 cm; and the branches of the spinal nerves *III* and *VIII* were cut out at their branching points. Longitudinal incisions of 2.5 cm to 3.2 cm long for operations were made through the skin and wall along lateral edges of vertebral transverse processes on both sides, and then the nerves were bilaterally cut. The wounds were sutured immediately after cutting the nerves. Thereafter, the frogs were allowed to recover from the wounds. In the second group, five frogs were sham-operated, leaving the nerves intact. The third group of the remaining nine frogs was the control group and received no incisions.

They were kept at temperatures ranging between 10°C and 20°C in containers with shallow tap-water, and were fed 5 g to 6 g ground meat once a week. The sutures were removed one or two weeks after the incisions, and between forty two and fifty days after the operations, the frogs of the first and the second group were used for the experiment. Each frog was given a 4 g piece of calf liver and then, 15 min to 30 min later, they were orally given 1 ml of Ringer's solution with emetic of 2.5% antimony potassium tartrate (Kishida Chemical, Osaka) per 100 g body weight using catheter. After that their behavior was observed at room temperatures ranged between 10.3°C and 19.0°C for 6 hrs to 19 hrs together with frogs of the third group which also had been administered a piece of calf liver and the emetic.

Results

In Tonosamagaeru, it was evidently shown that the ejection of food occurred simultaneously with the electromyogram reading associated with the contraction of rectus abdominis muscle (Fig. 1). Vomiting of this animal induced by the administration of tartar emetic was always accompanied by the sudden prolapse of esophagus and stomach into the mouth.

In bullfrogs, the following results were obtained; the points of them are tabulated (Table 1).

In the normal frogs, oral administration of the tartar emetic led to abrupt and powerful contractions of the abdominal muscles, and to opening the mouth. These occurred in periods of 1 hr 13 min to 10 hrs 22 min after the administration of the emetic; and, always resulted in the vomiting of the piece of liver administered previously from the stomach in periods of between 3 hrs 58 min to 15 hrs 4 min. On

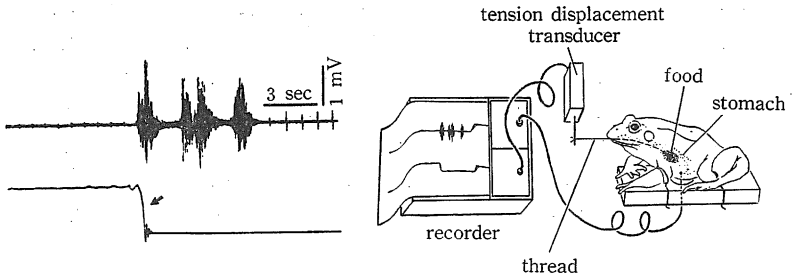

Fig. 1. Relationship between expulsion of the food from the stomach and electromyogram recording of rectus abdominis muscle in the vomiting of Tonosamagaeru, *Rana nigromaculata*. Upper recording: Electromyogram recording of rectus abdominis muscle contraction. Time constant is 0.01 sec. Bottom recording: Tension against the food in stomach. When the food was expelled from the stomach into the mouth, the tension decreased and reading shifted downwards (arrow). The method to record both the electromyogram and the tension was the same as had been used in our previous paper (Hukuhara *et al.*, 1973). The schematic representation is shown on the right. In this case, 0.6 m*l* of Ringer's solution with 0.05% antimony potassium tartrate was orally administered to the frog of 25.8 g (♂). Ejection of food from the stomach occurred 13 min after the administration of the emetic. The prolapse of stomach was observed when the food was expelled. Room temperature was 28.2°C.

Table 1. Effect of antimony potassium tartrate on the vomiting of frogs.

treatment	vomiting act	prolapse of stomach	expulsion of food
not operated	(+)	(+)	(+)
	(+)	(+)	(+)
	(+)	(+)	(+)
	(+)	(+)	(+)
	(+)	(+)	(+)
	+	+	+
	+	+	+
	+	+	+
	+	+	+
sham-operated	(+)	(+)	(+)
	(+)	(+)	(+)
	(+)	(+)	(+)
	+	+	+
	+	+	+
denervated	(+)	(−)	(+)
	(+)	(−)	(+)
	(+)	(−)	(−)
	(+)	(−)	(−)
	(+)	(−)	(−)
	(+)	(−)	(−)

Each symbol indicates one individual.
+: each phenomenon occurred.
−: each phenomenon did not occur.
Parentheses indicate death after experiment from the toxicity of antimony potassium tartrate.

the other hand, in the sham-operated frogs of the second group with the intact nerves, the vomiting act such as described above occurred between 48 min and 4 hrs 42 min after the administration of the emetic, and actual ejection of the food was observed in 50 min to 5 hrs 29 min. In both cases, the ejection of the food from the stomach was induced by the vigorous contraction of the abdominal muscles which was accompanied by an abrupt prolapse of the esophagus and stomach into the mouth. Fig. 2 shows the typical prolapse of esophagus and stomach in a control frog. Immediately after the prolapse, frogs usually gestured as if they were sweeping something

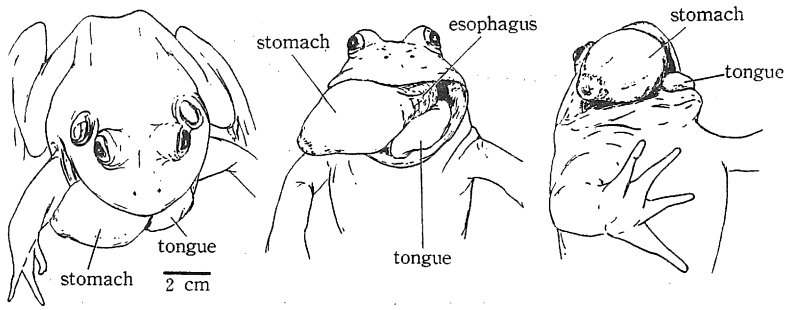

Fig. 2. Prolapse of esophagus and stomach in the vomiting of the bullfrog. Drawn from photographs of the same frog. ♀, 400 g.

from their prolapsed stomach by their forefeet. Within a few minutes after vomiting, however, both the esophagus and stomach were quickly swallowed back, the food having been discarded. Thereafter, the vomiting act described above were repeated on many occasions for a long periods, but the prolapse was repeated only a few times.

In the bullfrogs with the denervated abdominal walls, the prolapse of the stomach did not occur, though only the part of the esophagus which was situated closely to the esophageal orifice slightly prolapsed. They opened their mouths wide and ejected viscous fluids from the esophageal orifices. Their abdomens were distended backward. Two of them expelled a piece of liver without any prolapse of stomach 3 hrs 14 min and 4 hrs 55 min after administration of the emetic respectively. But, the remaining four frogs were unable to eject the food.

Five of nine normal frogs and three of the five sham-operated frogs died about 1.5 days to 16.5 days after administration of the emetic, presumably owing to poisonous action of the emetic. The remaining six frogs of these groups, however, were kept alive for more than 7 months until they were killed. On the other hand, all of the six frogs with the denervated abdominal walls died in 1 day to 4 days. According to the post-mortem examinations, the calf liver remained at the cardia in one of the four frogs that did not vomit, and in the upper lumen of the stomach in the remaining three.

In supplementary experiments, though the same results had been obtained in our previous paper (Hukuhara *et al.*, 1973), it was confirmed that, in spite of the mechanical

stimulation of the buccal cavity or oral administration of antimony potassium tartrate, bullfrogs with intact nerves neither showed a prolapse of the stomach nor expelling of the food when the abdominal cavity was opened.

In the course of the experiments, the esophagus and stomach were turned inside out when they prolapsed both in Tonosamagaeru and in bullfrogs as are shown in Fig. 3.

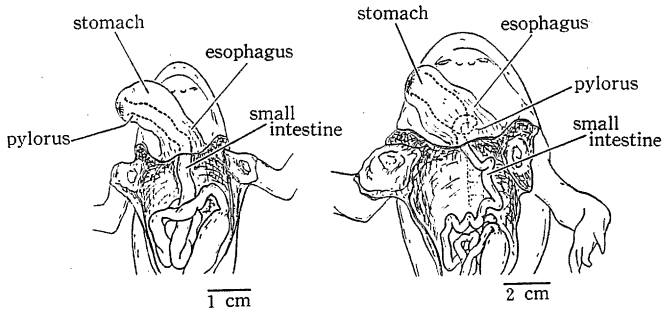

Fig. 3. The esophagus and stomach after they have prolapsed during vomiting. Left: Tonosamagaeru, 42.1 g, ♀. Right: bullfrog, 500 g, ♀. Frogs were immersed in boiling water as soon as vomiting was produced by mechanical stimulation of buccal cavity, and, then, esophagus, stomach and intestine were exposed by removing lower jaw, upper abdominal wall and guts other than gastro-intestinal tract.

Discussion

Vomiting of the frog is easily elicited by mechanical stimulation of the buccal cavity near the esophageal orifice or by oral administration of the emetic of antimony potassium tartrate. The most noticeable feature in this occasion is that the ejection of gastric contents is always accompanied by the sudden prolapse of esophagus and stomach into the mouth (Tokita *et al.*, 1953; Noble, 1954; Hukuhara *et al.*, 1973).

What is the motor force to make them prolapse? This is an interesting problem in a viewpoint of the physiology of gastro-intestinal movements in animals. Hukuhara (1967) postulated that the invagination of small intestine is produced by the contraction of intestinal wall due to the intrinsic mucosal reflex. In the previous paper (Hukuhara *et al.*, 1973), however, we suggested that the prolapse of the esophagus and stomach in the vomiting of frogs was produced not by the contractions of these organs but by the raising of the intra-abdominal pressure due to the contraction of the abdominal musculature, because of following two major reasons, (1) vomiting was always accompanied by a strong contraction of the abdominal musculature, which is also shown in Fig. 1 in the present paper, (2) the stomach, exposed by incision of the abdomen, did not show any particular movement to produce the invagination during vomiting act.

If the abdominal contraction induces prolapse of the stomach as well as the esophagus, it is expected that frogs with paralysed abdominal musculature will fail to show prolapse. The results obtained in the present experiment satisfactorily support the concept, that is to say, prolapse of the stomach did not occur in the frogs with denervated abdominal wall. Two of six frogs with denervated abdominal musculautre expelled the liver food, though the prolapse of stomach was not observed. The expulsion of the food, however, may be caused by the rise of intra-luminal pressure of the stomach due to the stomach contraction, which might be due to antimony potassium tartrate (Hukuhara *et al.*, 1973) and/or extrinsic reflexes where the autonomic nerves are involved.

It is now reasonably concluded that vomiting of the frog is induced, not by the movement of the stomach, but by the prolapse of the esophagus and the stomach into the mouth which is caused by the abrupt rise of the intra-abdominal pressure due to the strong contraction of abdominal musculature. It is assumed that a flexible esophageal wall and a large esophageal orifice easily lead to the prolapse of the esophagus and the stomach when raised intra-abdominal pressure forces them to shift upwards. In short, abdominal musculature, which is an expiratory muscle, plays a major role in the vomiting of the frog. In comparative study, therefore, vomiting of the frog is under the same contribution of respiratory musculature as in the mammals (Hukuhara *et al.*, 1957). The results described here do not support the concept postulated by Mellinger (1881) and by Gruzewski under Thumas (1891). Mellinger reported that vomiting was successfully produced by antiperistalsis of the stomach in the pithed frog with an opened abdomen and Thumas said that Gruzewski confirmed the Mellinger's experiment. Their results were quoted in the monographs by Klee (1927) and by Reeder (1964) without any doubt.

Some of the frogs which had vomited with the aid of the abdominal musculature were likely to continue to live for long periods because the vomiting prevented the ingestion of poisonous emetic of antimony potassium tartrate.

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