Mechanisms of airway protection during retching, vomiting, and swallowing

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Lang, Ivan M., Nicole Dana, Bidyut K. Medda, and Reza Shaker. Mechanisms of airway protection during retching, vomiting, and swallowing. Am J Physiol Gastrointest Liver Physiol 283: G529–G536, 2002; 10.1152/ajpgi.00062.2002.—We investigated the mechanisms of airway protection and bolus transport during retching and vomiting by recording responses of the pharyngeal, laryngeal, and hyoid muscles and comparing them with responses during swallowing and responses of the gastrointestinal tract. Five dogs were chronically instrumented with electrodes on the striated muscles and strain gauges on smooth muscles. Retching and vomiting were stimulated by apomorphine (5–10 ug/kg iv). During retching, the hyoid and thyroid descending and laryngeal abductor muscles were activated; between retches, the hyoid, thyroid, and pharyngeal elevating, and laryngeal adductor muscles were activated. Vomiting always occurred during the ascending phase of retching and consisted of three sequential phases of hyoid and pharyngeal muscle activation culminating in simultaneous activation of all recorded elevating and descending laryngeal, hyoid, and pharyngeal muscles. Retrograde activation of esophagus and pharyngeal muscles occurred during the later phases, and laryngeal adductor was maximally activated in all phases of the vomit. During swallowing, the laryngeal adductor activation was followed immediately by brief activation of the laryngeal abductor. We concluded that retching functions to mix gastric contents with refluxed intestinal secretions and to impart an orad momentum to the bolus before vomiting. During retches, the airway is protected by glottal closure, and between retches, it is protected by ascent of the larynx and closure of the upper esophageal sphincter. The airway is protected by maximum glottal closure during vomiting. During swallowing, the airway is protected by laryngeal elevation and glottal closure followed by brief opening of the glottis, which may release subglottal pressure expelling material from the laryngeal vestibule.

cricopharyngeus; stylopharyngeus; geniohyoideus; thyroarytenoideus

Whereas many of the mechanisms that prevent aspiration during swallowing, i.e., vocal cord closure, aryepiglottic approximation, and laryngeal elevation (23), and during gastroesophageal reflux, i.e., contraction of the upper esophageal sphincter (18), have been described, the motor responses that contribute to airway protection during vomiting are unknown. During vomiting, some of these airway protective mechanisms either do not occur, e.g., contraction of the upper esophageal sphincter, or would provide little airway protection, e.g., aryepiglottic approximation.

In prior studies (10, 11, 16), the intestinal, gastric, esophageal, pharyngeal, and some respiratory muscle responses that occur during retching and vomiting were characterized and quantified, but the motor responses of the intrinsic laryngeal muscles and the muscles that elevate and lower the pharynx, larynx, and hyoid are largely unknown. The responses, during vomiting, of some upper airway muscles have been examined in the decerebrate cat (24), but many significant muscles were not examined. No studies have been performed using intact unanesthetized animals, and the relationship between the digestive tract motor responses and the responses of the upper airway and hyoid muscles have not been investigated. Understanding the activation of the laryngeal, pharyngeal, and hyoid muscles is important for understanding the mechanisms both of airway protection and of the transport of the bolus from esophagus to oral cavity during vomiting.

The aims of this study were to 1) characterize and quantify the laryngeal, pharyngeal, and hyoid muscle responses during retching and vomiting; 2) correlate the activation of laryngeal, pharyngeal, and hyoid muscles with the digestive tract responses; and 3) compare the responses of these muscles during retrograde transport, i.e., vomiting, with responses during anterograde transport, i.e., swallowing, to understand the mechanisms of bolus transport and airway protection.

METHODS

Animal Preparation

Experiments were performed on five mixed breed dogs of either sex weighing between 16 and 23 kg. All dogs were

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implanted with recording devices on the gastrointestinal tract, diaphragm, and various pharyngeal, laryngeal, and hyoid muscles of the neck using aseptic surgical techniques under Halothane (1–1.5%) anesthesia. The devices were implanted during two separate surgeries at least 2 wk apart, and the abdominal devices were implanted first. The gastrointestinal tract was implanted with strain-gauge force transducers situated on the gastric antrum, duodenum, and at least three sites in the jejunum 50 cm apart. The abdominal side of the diaphragmatic hiatus was implanted with a bipolar silver wire electrode. The wires from the strain gauges exited the abdomen through a stainless steel cannula as previously described (10). The following muscles of the neck were implanted with bipolar silver wire electrodes as previously described (11): supahyoid muscles: geniohyoideus (GH), mylohyoideus (MH), and hyoglossus (HG); infrahyoid muscles: thyrohyoideus (TH), sternohyoideus (SH), and sternothyroideus (ST); intrinsic pharyngeal muscles: thyropharyngeus (TP), cricopharyngeus (CP), and hyopharyngeus (HP); extrinsic pharyngeal muscles stylopharyngeus (SP); extrinsic laryngeal muscle: cricothyroideus (CT); laryngeal adductor muscle: thyroarytenoideus (TA); laryngeal abductor muscle: cricoarytenoideus dorsalis (CD); and esophagus at 1 (E1) and 4 (E4) cm from the CP. These electrodes were composed of wires imbedded into a thin Silastic backing. The Silastic backing was used to sew the electrodes onto the muscle and to provide electrical insulation from other electrodes and other muscles. The left jugular vein was cannulated with a Silastic catheter, and the other end of this catheter was fitted with an intravenous injection plug. This jugular vein catheter was implanted subcutaneously, which allowed for intravenous administration of emetic agent by a subcutaneous injection. This catheter avoided the trauma of venipuncture at each experimental session. Experiments were begun at least 14 days after the last surgery.

Electromyography

The implanted bipolar electrodes consisted of 18-gauge silver wires spaced 5 mm apart and exposed for a length of 3 mm embedded in a thin (2 mm) Silastic rubber backing. The electrodes were sutured to the muscle along the long axis of the muscle fibers. The electrodes were connected to an Amphenol plug by Teflon-coated stainless steel wires (Cooner Wire AS636), and the plug was embedded in a dental acrylic base. For electromyographic (EMG) recording, electrical activity was first band-pass filtered (0.1–1.0 kHz) and then amplified (1,000 ×) by a differential amplifier (A-M Systems model 1700). The amplified signal was acquired and stored in a computer using CODAS (Dataq Instruments) hardware and software.

Contractile Activity

The strain-gauge force transducers consisted of precision strain-gauge elements (EA-06–031DE-120; Micromeasurements Group, Raleigh, NC) glued onto a copper-beryllium shim, waterproofed using polysulfide coating (M-coat JL, Micromeasurements Group) and embedded into Silastic rubber. The strain gauges were connected to an Amphenol plug by Teflon-coated silver-plated copper wires. Each strain gauge was connected electrically to a quarter Wheatstone bridge circuit before amplification by transducer amplifier (CWE, model PM-1000). The amplified signal was acquired and stored using CODAS hardware and software.

Stimulation of Retching and Vomiting

The dogs were fasted overnight for 15–18 h, and vomiting was stimulated by intravenous injection of apomorphine (5–10 ug/kg) through the jugular vein catheter. This dose of apomorphine activated, at most, two vomits, and vomiting was stimulated, at most, twice per week. Each dog was studied two to six times. Retching was identified as the period of rhythmic heaving caused by contraction of the diaphragm and abdominal muscles (11, 16). In prior studies, Lang et al. (11) found that retching was associated with alternating periods of simultaneous activation and inhibition of pharyngeal muscles and esophagus. Vomiting was identified as the period during which gastrooral reflux occurred when the diaphragmatic hiatus ceased to be active (11, 16) and the GH was maximally activated (11).

Data Acquisition, Storage, and Analysis

Amplified signals were acquired, stored, and quantified on a computer using CODAS hardware and software. The stored EMG signals were acquired at 1,000 Hz, and the strain gauge signals were acquired at 25 Hz. The magnitude of EMG signals was quantified by the following advanced CODAS functions: full-wave rectification, moving average at 1/3 s smoothing factor, and area under the curve.

RESULTS

Relationship Between the Laryngeal, Pharyngeal, and Hyoid Muscle Responses with the Gastrointestinal Motor Responses

The first motor event we observed to occur in association with vomiting was the retrograde giant contraction (RGC) of the gastrointestinal tract (Fig. 1). The RGC was not associated with any response of the esophagus or pharyngeal, laryngeal, or hyoid muscles. Retching always (20 of 20 trials) began after the arrival of the RGC to the gastric antrum and usually (18 of 20 trials) after the peak of the contraction (Fig. 1). The average time delay between the arrival of the peak of the RGC in the gastric antrum (3 cm from the pylorus) to the first retch and the vomit was 7.8 ± 1.2 and 18.4 ± 1.3 s, respectively.

Laryngeal, Pharyngeal, and Hyoid Muscle Responses During Retching and Vomiting

Three distinct stages were observed during the retching and vomiting sequence: preretch, retch, and vomit.

Preretch. Just before retching, significant changes in resting activity of some of the pharyngeal, hyoid, and laryngeal muscles occurred (Fig. 1 and Table 1). Before the first retch, the CP, esophagus, TH, SH, ST, and CD were tonically activated for a duration ranging from 4.8 to 14.1 s. This tone increased from 2.8 to 13.8 times the spontaneous activity before administration of the emetic agent (Table 1). The changes in tone of these muscles increased steadily until the beginning of retching, and no (0 of 20 vomiting episodes) swallows occurred during this time (Fig. 1).

Retching. The average number of retches per vomit was 10 ± 1 (range 1–22), and they occurred at 0.9 ± 0.02/s. The time during the retch (0.57 ± 0.01 s) was
not different \((P > 0.05)\) from the time between retches \((0.56 \pm 0.02 \text{ s})\). During the entire retching period, the tone of the TA increased \((1,300 \pm 110\%)\) and tone of the CD decreased \((54 \pm 2\%)\). During and between retches, different sets of muscles were activated simultaneously (Figs. 1 and 2). During retching, the hyoid (SH) and thyroid (ST) descending muscles were strongly activated, and between retches, the hyoid (MH, GH, HG), thyroid (TH), and pharyngeal (SP) elevating muscles, pharyngeal constricting muscles (TP, CP, and HP), and the esophagus were strongly activated. The HP and SP were activated during both phases of retching, but primarily between retches.

**Vomiting.** Vomiting began at \(0.30 \pm 0.02 \text{ s}\) after the end of the last retch, which was significantly \((P < 0.001)\) sooner than the next expected retch \((0.56 \pm 0.02 \text{ s})\), sometimes \((3 \text{ of 45 trials and } 2 \text{ of 5 animals})\) began at the end of the interretch interval, and never \((0 \text{ of 45 trials and } 0 \text{ of 5 animals})\) began during or immediately after a retch (Fig. 2).

**Three Phases of Muscle Activation Occurred During Vomiting**

**Phase I.** During the first phase of the vomit, which began with activation of the diaphragm, a laryngeal adductor muscle, TA, a pharyngeal elevating and distending muscle, SP, and a laryngeal descender muscle, ST, were activated simultaneously with relaxation of the intrinsic pharyngeal muscles, TP and CP (Table 2 and Fig. 2, A and C). The responses of the TA, SP, and ST lasted the duration of the vomit for \(2.12 \pm 0.11 \text{ s}\).

**Phase II.** The second phase of the vomit began \(~0.85 \text{ s}\) after the beginning of the vomit and was characterized by activation of the suprahyoid muscles: HP, HG, GH, and MH (Table 2 and Fig. 2, B and C).

**Phase III.** The third phase of the vomit began \(~1.5 \text{ s}\) after the beginning of the vomit and was characterized by simultaneous strong activation of the TH and SH. (Table 2 and Fig. 2, B and C).

A retrograde contraction propagated from the proximal esophagus to the CP and TP in a sequential manner at a rate of \(~6 \text{ cm/s}\) during the second and third phases of the vomit (Table 3 and Fig. 2, A and C). This retrograde peristalsis was often followed by an anterograde peristalsis of the same muscles (Fig. 2A). This anterograde peristalsis appeared similar to peristalsis associated with a swallow except that the CP activation was not preceded by inhibition (Fig. 2A).

**Comparison of the Laryngeal, Pharyngeal, and Hyoid Muscle Responses During Retching, Vomiting and Swallowing**

**Laryngeal muscles.** During retching as well as vomiting, the TA, a laryngeal adductor muscle, was activated during the time when the CP was inhibited, and
was strongly activated just after TA activation during swallowing (Fig. 2, A and B).

Pharyngeal constrictor muscles. During retching and vomiting, the intrinsic pharyngeal muscles, HP, TP, and CP were activated (between retches) or inhibited (during retches and the vomit) simultaneously (Fig. 2). In contrast, the intrinsic pharyngeal muscles were activated in a caudally sequential manner during swallowing (Fig. 2). The magnitudes of the responses of these muscles were not much different from responses of these muscles during swallowing (Table 4). During phase III of the vomit, a sequential aboral activation of the pharyngeal muscles occurred similar to that which occurred during swallowing, but it was not accompanied by relaxation of the CP, which is a prominent feature and important action during swallowing (Fig. 2).

Hyoid and thyroid muscles. During retching, the hyoid (GH) and thyroid (TH) elevating and descending (SH, ST) muscles were activated alternatively, and during vomiting, they were activated simultaneously. In contrast, during swallowing, the hyoid (GH) and thyroid (TH) elevating muscles were strongly activated simultaneously and the hyoid (SH) and thyroid (ST) descending muscles were inhibited (Fig. 2B). During retching, the muscle activated most strongly (over 3 times) relative to its response during swallowing was the ST; during vomiting, all hyoid muscles were more strongly activated (over 3 times) than during swallowing, except the TH (Table 4, Fig. 2, B and C).

Pharyngeal Elevator and Dilator Muscle

The pharyngeal elevator and dilator muscle, SP, was activated between retches as well as during vomiting and swallowing (Fig. 2C). The responses during vomiting and swallowing occurred at times when the bolus would be expected to be in the pharynx. The SP was more strongly activated (~2.5 times) during vomiting than swallowing.

DISCUSSION

This study examined the laryngeal, pharyngeal, and hyoid muscle responses during retching and vomiting in relation to swallowing as well as the motor responses of the digestive and respiratory tracts to obtain a more complete understanding of the vomiting pro-

Table 2. Phases of the vomit with time delays of specific muscle responses

<table>
<thead>
<tr>
<th>Phase</th>
<th>Muscles</th>
<th>Mean delay</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>ST: 0.25±0.09 s</td>
<td>0.24±0.06 s</td>
</tr>
<tr>
<td></td>
<td>SP: 0.30±0.10 s</td>
<td>0.86±0.15 s</td>
</tr>
<tr>
<td></td>
<td>TA: 0.18±0.05 s</td>
<td>1.56±0.18 s</td>
</tr>
<tr>
<td>II</td>
<td>GH: 0.89±0.18 s</td>
<td></td>
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<tr>
<td></td>
<td>MH: 0.68±0.10 s</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HP: 0.82±0.32 s</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HG: 1.03±0.33 s</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>SH: 1.38±0.16 s</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TH: 1.74±0.20 s</td>
<td></td>
</tr>
<tr>
<td></td>
<td>MH: 0.68±0.10 s</td>
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</table>

Values are means ± SE of time delays from the beginning of vomit (diaphragm activation and/or CP inhibition) to the start of muscle activation. SP, stylopharyngeus; TA, thyroarytenoideus; GH, geniohyoideus; MH, mylohyoideus; HP, hyopharyngeus; HG, hyoglossus.
cess. Study findings corroborate prior reports (10, 11) that the process of vomiting has three stages: preretch, retch, and vomitus expulsion; and for the first time, the laryngeal, pharyngeal, and hyoid muscle responses during each stage of the vomiting process were investigated (Fig. 3).

Before retching, there is an increase in the rate of swallowing and licking (11), but for ~14 s before retching, swallowing and licking ceased and the tone of the CP, esophagus, TH, SH, ST, and CD increased (Fig. 3). The increase in tone of the CD was both short lasting and relatively low amplitude and may have been due to the change in response characteristics rather than amplitude. During this period, the tone of the CD was rather constant, whereas during the control period, the tone fluctuated with licking. The physiological significance of the activation of this laryngeal abductor muscle during this time is unknown.

The increase in tone of the CP and esophagus before retching was previously (11) found to be the EMG correlate of longitudinal contraction of the esophagus, which acts to pull the proximal stomach orad (8, 13, 20), thereby eliminating one of the anatomical impediments (incisura cardiaca) to gastroesophageal reflux. The simultaneous low-level tonic activation of the ST, SH, and TH before retching may reflect the change in posture of the dogs as they extend their necks and lower their heads at this time in preparation for retching and vomiting. The voluntary nature of this posture may explain the lack of similar findings with regard to the TH in decerebrate cats (24).

During retching, there was alternating activation of hyoid and thyroid elevating and hyoid and thyroid descending muscles, which probably acted to pull the larynx caudally during the retch and to pull the larynx cranially between retches (Figs. 3 and 4). We found that the activation of these muscles was relatively constant, whereas in decerebrate cats, the TH was activated in a decrementing fashion (24). It is unknown whether this difference was due to species or technical differences. In addition, we found that the pharyngeal muscles and esophagus contracted between retches and relaxed during retches, which probably acted to elevate the proximal stomach between retches and allow the proximal stomach to fall during retches. These combined muscle actions would have resulted in the larynx, pharynx, esophagus, and proximal stomach moving caudally during each retch and cranially between retches, such that the entire gastroesophagopharyngolaryngeal apparatus would move up and down in synchrony with the diaphragm (Fig. 4).

Gastric contents may reach as far orad as the caudal border of the upper esophageal sphincter during the retching process (7, 20), but neither esophagopharyngeal reflux nor aspiration occurs. Esophagopharyngeal reflux and aspiration probably do not occur during retches, because the gastroesophagopharyngolaryngeal apparatus (and its contents) and diaphragm move caudal (as discussed above), thereby moving the bolus away from the larynx and pharynx (Fig. 4). On the other hand, between retches, when the gastroesophagopharyngolaryngeal apparatus (and its contents) and diaphragm move cranially (Fig. 4), the pharynx contracts, pull the proximal stomach orad during retches, and relax during retches, whereas the pharynx relaxes and pushes the proximal stomach caudally between retches (Figs. 3 and 4).

Table 3. Time delays of responses from esophagus to pharynx during vomiting

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>Muscle</th>
<th>Delay of peristalsis from vomit (s)</th>
<th>E4</th>
<th>E1</th>
<th>CP</th>
<th>TP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharyngeal</td>
<td>HP</td>
<td>0.80 ± 0.32</td>
<td>1.41 ± 0.16</td>
<td>1.51 ± 0.08</td>
<td>1.64 ± 0.11</td>
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<td></td>
<td>TP</td>
<td>0.60 ± 0.09</td>
<td>1.04 ± 0.14</td>
<td>1.05 ± 0.25</td>
<td>1.07 ± 0.14</td>
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<tr>
<td></td>
<td>CP</td>
<td>0.53 ± 0.10</td>
<td>1.05 ± 0.25</td>
<td>1.06 ± 0.25</td>
<td>1.08 ± 0.25</td>
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<tr>
<td></td>
<td>SP</td>
<td>1.46 ± 0.54</td>
<td>2.50 ± 0.42</td>
<td>2.52 ± 0.42</td>
<td>2.54 ± 0.42</td>
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<tr>
<td></td>
<td>CD</td>
<td>0.82 ± 0.20</td>
<td>1.48 ± 1.61</td>
<td>1.50 ± 1.61</td>
<td>1.52 ± 1.61</td>
<td></td>
</tr>
<tr>
<td>Laryngeal</td>
<td>TA</td>
<td>4.60 ± 0.92</td>
<td>11.50 ± 1.07</td>
<td>11.52 ± 1.07</td>
<td>11.54 ± 1.07</td>
<td></td>
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<tr>
<td>Hyoid</td>
<td>MH</td>
<td>1.31 ± 0.39</td>
<td>3.82 ± 0.38</td>
<td>3.84 ± 0.38</td>
<td>3.86 ± 0.38</td>
<td></td>
</tr>
<tr>
<td></td>
<td>GH</td>
<td>0.78 ± 0.06</td>
<td>5.22 ± 0.60</td>
<td>5.24 ± 0.60</td>
<td>5.26 ± 0.60</td>
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<tr>
<td></td>
<td>TH</td>
<td>1.75 ± 0.44</td>
<td>1.63 ± 0.40</td>
<td>1.65 ± 0.40</td>
<td>1.67 ± 0.40</td>
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<tr>
<td></td>
<td>SH</td>
<td>0.78 ± 0.35</td>
<td>5.85 ± 2.13</td>
<td>5.87 ± 2.13</td>
<td>5.89 ± 2.13</td>
<td></td>
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<tr>
<td></td>
<td>ST</td>
<td>3.50 ± 1.26</td>
<td>15.82 ± 6.27</td>
<td>15.84 ± 6.27</td>
<td>15.86 ± 6.27</td>
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Values are means ± SE of time delays from the beginning of vomit (see Table 2 for definition) to the beginning of observed responses. E4, esophagus at 4 cm; TP, thyropharyngeus.

Table 4. Magnitude of responses of pharyngeal, laryngeal, and hyoid muscles during retching and vomiting relative to swallowing

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>Muscle</th>
<th>Retch</th>
<th>Vomit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharyngeal</td>
<td>HP</td>
<td>1.49 ± 0.37</td>
<td>4.68 ± 1.45</td>
</tr>
<tr>
<td></td>
<td>TP</td>
<td>0.69 ± 0.07</td>
<td>1.04 ± 0.14</td>
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<tr>
<td></td>
<td>CP</td>
<td>0.53 ± 0.10</td>
<td>1.05 ± 0.25</td>
</tr>
<tr>
<td></td>
<td>SP</td>
<td>1.46 ± 0.54</td>
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<td>5.85 ± 2.13</td>
</tr>
<tr>
<td></td>
<td>ST</td>
<td>3.50 ± 1.26</td>
<td>15.82 ± 6.27</td>
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</table>

Values are means ± SE of ratio of EMG response during retch or vomit/EMG response during swallowing.
Fig. 4. Illustration of movements of important digestive and respiratory structures based on the observed muscle responses during retching and vomiting. A: movement of larynx/pharynx during retching and vomiting. Thicker lines between structures indicate stronger muscle responses, and arrows indicate force vector. Note that vomiting occurs only after the interretch period and results in greatest distraction of pharynx/larynx away from spinal column and greatest opening of upper esophageal sphincter. B: movement of pharynx, larynx, esophagus, and stomach relative to the diaphragm during retching and vomiting. The actions of diaphragm, esophagus, and stomach are taken from current study and the Refs. 10, 11, and 17. Note that retching causes rhythmic orad and caudal movement of the entire pharyngeal, laryngeal, esophageal, and gastric apparatus. During vomiting, the esophagus and stomach are maximally stretched, perhaps to eliminate any obstruction to gastroesophageal reflux, and the diaphragmatic dome compresses the stomach with relaxation of the hiatus, which allows gastroesophageal reflux. sh, suprahyoid muscles; HB, hyoid bone; PE, pharynx and esophagus; sh/P, superior hyoid and pharyngeal muscles; L/P, larynx and pharynx; D, diaphragm; S, stomach, E, esophagus.

Close as the pharyngeal constrictors (CP, TP, and HP) are strongly activated, and the glottis closes as the laryngeal adductor (thyroarytenoideus) muscle is strongly activated simultaneously with relaxation of the laryngeal abductor (CD) muscle. Therefore, both strongly activated simultaneously with relaxation of the laryngeal adductor (thyroarytenoideus) muscle is strongly activated, and the glottis closes as the pharyngeal constrictors (CP, TP, and HP) closes as the pharynx; D, diaphragm; S, stomach, E, esophagus.

The primary motive force for vomitus expulsion is the pressure gradient between the stomach and esophagus generated by diaphragmatic and abdominal muscle compression of the stomach with a closed pylorus (4, 8, 14). We found that this force is always applied between retches when the cranially directed force of contraction of the hyoid, pharyngeal, and esophageal muscles pull the proximal stomach orad (7, 8, 11, 13, 14, 20). As the proximal stomach is pulled orad and enters the thoracic cavity (7, 8, 14, 20), the gastric contents are thrown orad such that the gastric contents sometimes move as far orad as just below the upper esophageal sphincter (7, 20). The effect of orad movement of the bolus before the strong gastric compression during vomiting may be to amplify the effects of the compressive forces of the diaphragm and abdominal muscles by imparting momentum to the bolus.

We found that vomiting may be separated into three groups of sequential motor responses based on the actions of the laryngeal, pharyngeal, and hyoid muscles (Fig. 3). Once a muscle or group of muscles was activated, it stayed activated for the duration of the vomit. During phase I of the vomit, a pharyngeal elevator and dilator muscle was activated simultaneously with a thyroid descender and relaxation of the intrinsic pharyngeal muscles. The effect of these muscle responses would have been to begin the opening of the pharynx and upper esophageal sphincter. Prior studies (11, 16) have found that, at this time, the diaphragm and abdominal muscles maximally contract and the lower esophageal sphincter is relaxed (11). We found that all of this occurs during the ascending phase of the retch when the elevating muscles of the larynx, pharynx, esophagus, and proximal stomach had already been activated and gastric contents would have already begun moving orad.

Phases II and III of the responses of the pharyngeal, hyoid, and laryngeal muscles during vomiting may function to maximally open the upper esophageal sphincter and pharynx and to move the vomitus from esophagus to pharynx. During phase II of the vomit, the suprahoid muscles were activated very strongly and simultaneously. The consequences of activation of these muscles along with the continued activation of the laryngeal descender muscle (from phase I) would have been to strongly pull the hyoid bone anteriorly and superiorly away from the larynx. The hyoid bone is attached to the larynx through the TH, but at this time, the TH had not begun to be activated. The action of these suprahoid muscles may have been to move and lock the hyoid in a superior and anterior position, where the action of the TH would be used to assist vomiting in phase III. During phase III of the vomit, maximal superior and inferior tension on the larynx occurred, because, in addition to the prior activation of the hyoid elevator and thyroid descender muscles, the SH and TH were activated strongly. The result of activation of the TH would have been to move the larynx anteriorly toward the position of the hyoid. The activation of the SH would provide the final and maximal anterior and inferior traction on the larynx (SH,
through the TH). It was at this time that the larynx was probably maximally distracted from the spinal column and the upper esophageal sphincter and pharynx were maximally pulled open (Fig. 4). Bolus movement through the aerodigestive tract during this time was probably accomplished by the previously (11) characterized and quantified vomit-related retrograde peristaltic contraction of the esophagus and pharynx (Fig. 3). We found in the current study that this retrograde peristaltic contraction occurred during phases II and III of the vomit.

The airway was probably protected throughout all phases of vomiting by the maximal activation of the laryngeal adductor and inhibition of the laryngeal abductor muscles. It is unlikely that laryngeal elevation contributed to airway protection, because laryngeal descending muscles were strongly activated throughout all stages of the vomit.

Three phases of laryngeal and pharyngeal muscle activity during vomiting were also identified in the decerebrate cat (24), and, in general, the findings were similar to those obtained in the current study. Significant differences were found with regard to the laryngeal adductor, CD, and pharyngeal dilator, SP, muscles. In the decerebrate cat (24), the CD was activated during vomiting as it was during retching and the SP was not activated until phase III of the vomit. However, considerable variability in responses of both of these muscles was observed in the decerebrate cats (24), because responses sometimes changed in the same animal and some responses were observed in only about one-half of the animals. This variability may have been due to the decerebrate preparation.

We found that the vomit-related retrograde contraction of the esophagus and pharynx was often followed immediately by secondary peristalsis (Fig. 3). The combination of TP activation by the retrograde contraction followed by secondary peristalsis made it appear as if a swallow had occurred. However, this set of responses was not a swallow (3, 9), because it was not accompanied by inhibition of the CP (i.e., upper esophageal sphincter relaxation) or activation of laryngeal, hyoid, or other pharyngeal muscles characteristic of a swallow.

We compared the responses of the laryngeal, pharyngeal, and hyoid muscles during retching and vomiting to the responses of these muscles during swallowing, a function that moves contents in an opposite direction, to better understand the function of these muscles (Fig. 3). The mechanisms of bolus transport during phase I of vomiting and swallowing were different. During the first stage of vomiting, the bolus is transported by forceful expulsion of the bolus from the stomach through the maximally relaxed lower esophageal sphincter. On the other hand, during swallowing the bolus is transported by sequential activation of muscles in an orderly fashion throughout its travel. The mechanisms of bolus transport when the bolus traverses the aerodigestive tract during vomiting (phases II and III) and swallowing were similar but differed in a few specific ways. In both functions, the bolus was propelled by peristaltic contractions of the esophagus and pharynx; the upper esophageal sphincter was opened by relaxation of the CP and TP, and distraction of the larynx was by activation of superior hyoid muscles. However, during vomiting, an additional set of muscles was activated. The infrahyoid muscles were also strongly activated, and the combined supra- and infrahyoid muscle activation would have produced a force vector perpendicular to the larynx. This force would have maximally distracted the larynx from the spinal column and caused maximal opening of the upper esophageal sphincter without much superior or inferior movement of the larynx (Fig. 4).

The mechanisms of airway protection during vomiting and swallowing differed significantly. Airway protection during swallowing is accomplished by vocal cord closure, aryepiglottic approximation, and laryngeal elevation (18). The muscle responses we observed that contribute to these mechanisms included activation of the laryngeal adductor and the laryngeal elevating muscles. However, we found that during all phases of vomiting, laryngeal elevation is not likely to occur because of the strong activation of laryngeal descending muscles. The only airway protective mechanism operative during vomiting is probably glottal closure, because throughout the vomit, the laryngeal adductor is maximally activated and the laryngeal abductor is relaxed.

Another difference in upper airway muscle responses between swallowing and vomiting was that during swallowing, the laryngeal adductor muscle was activated briefly but vigorously just after the laryngeal adductor activation, whereas no such activation of the adductor occurred after adductor activation during vomiting (Fig. 3). This pattern of activity during swallowing was also seen in decerebrate cats (24). The function of this response is unknown, but this finding may be related to swallow-related subglottic pressure changes. It has been observed in humans and cats (5, 19) that an increase in subglottic pressure occurs during swallowing, and this pressure drops briefly during elevation of the larynx. The increase in pressure seems to be important to the timing of the swallow and may help prevent deglutitive aspiration (5). This activation of the CD may open the glottis during laryngeal elevation, causing the drop in subglottic pressure. The release of air from the trachea to the laryngeal vestibule and pharynx may function to expel material from the laryngeal vestibule, thereby serving a role in airway protection.

The relationship between the digestive tract correlates of vomiting and the pharyngeal, laryngeal, hyoid, and thyroid muscle responses has not previously been examined. We found that the digestive tract responses associated with vomiting are synchronized with the laryngeal, pharyngeal, hyoid, and thyroid muscle responses, such that the digestive tract responses are completed before retching occurs. In prior studies (10), we found that the digestive tract correlates of vomiting comprise a single well-organized set of responses that can occur independent of retching and vomiting. We (10) concluded from this and other data that the cen-
central pattern generator for vomiting is probably composed of at least two subsets: one controlling the digestive tract correlates of vomiting, and the other controlling retching and vomitus expulsion. In this study, we found that the laryngeal, pharyngeal, and hyoid muscle responses that comprise retching and vomitus expulsion also occur as a single well-organized set of responses. No evidence was found that retching and vomitus expulsion or any subgroup of pharyngeal, laryngeal, or hyoid muscle responses comprised a separate set of responses. Therefore, it is likely that all of the pharyngeal, laryngeal, and hyoid muscle responses associated with retching and vomiting are controlled by the same central pattern generator. The mechanisms that coordinate the two central pattern generators of vomiting are unknown. Considering that the two sets of responses comprise different types of muscles: smooth (digestive tract responses) vs. striated (pharyngeal, laryngeal, hyoid muscles), which are controlled by different brain stem nuclei, it is possible that the two central pattern generators of vomiting may not be located in the same brain stem nuclei.

In conclusion, we found that retching comprises a rhythmically alternating (about once per second) elevation and descent of the entire pharyngolaryngoesophageal apparatus in synchrony with the movement of the diaphragm. The function of retching may be to mix gastric contents with intestinal refluxate to buffer gastric contents before gastroesophageal reflux and to impart a momentum to the gastric contents before vomiting. Airway protection during retching is accomplished by glottal closure during retches and constriction of the upper esophageal sphincter between retches. Vomiting is composed of three phases of laryngeal, hyoid, and pharyngeal muscle responses. Gastroesophageal reflux occurs during the first phase due to abdominal compression, oral movement of the stomach, and relaxed lower esophageal sphincter. During the second and third phases, retrograde peristalsis of the esophagus and pharynx move the bolus through maximally opened upper esophageal sphincter and pharynx. Airway protection during vomiting is probably accomplished by glottal closure only. Brief glottal opening during swallowing may release an increase in subglottal pressure, which may function to expel material from the laryngeal vestibule, thereby serving a role in airway protection.

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REFERENCES


