Respiratory mechanics of vomiting in decerebrate cats

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1974—Observations were made on the changes in thoracic venous pressure, abdominal venous pressure, and arterial blood pressure associated with vomiting induced by veratrum alkaloids (50-150 µg/kg, iv). Tracheal and intrapleural pressures were also examined for comparison with the vascular indicators. Electromyograms were recorded concurrently from the diaphragm and/or the body wall muscles of the thorax and/or abdomen. Retching was characterized by a growing series of brief negative intrathoracic pressure pulses mirrored by positive pressure pulses in the abdomen. Expulsion then followed as a more sustained abdominal contraction with a sudden reversal of intrathoracic pressure from negative to positive. Remarkably, however, both retching and expulsion were effected by the same set of muscles according to their EMG profiles. Results obtained with spinal cord section at T and phrenicotomy support the conclusions: 1) that the diaphragm, acting in concert with the inspiratory muscles against a closed glottis, is responsible for the negative intrathoracic pressure produced in retching; and 2) that the abdominal musculature is responsible for the positive pressure generated in the abdomen during retching and expulsion, and which in the latter phase is transmitted into the thorax by an upward shift of the diaphragm.

Methods

Experiments were performed on 20 adult cats, decerebrated under halothane anesthesia. Carotid arterial blood pressure and thoracic and abdominal venous blood pressures were recorded by means of Statham P-23 transducers. The venous catheters were inserted into the superior and inferior vena cava through the jugular and femoral veins, respectively. Intrapleural pressure was measured when required from a T tube inserted low in the cervical trachea. Care was taken to leave intact at least three tracheal rings below the thyroid cartilage so as not to interfere with the glottis. Intrapleural pressure was recorded in one experiment through a fluid filled, open-ended catheter inserted with the aid of a large, blunt, hypodermic needle at an acute angle between two middle ribs. Bipolar electromyograms were obtained from the internal and external intercostal, diaphragm, rectus abdominis, and external oblique muscles by means of fine flexible copper-wire electrodes, sewn and anchored into the muscles by direct visualization. Generally, the sixth through eighth intercostal spaces were chosen as recording sites for thoracic muscles while diaphragmatic electrodes were placed, from an abdominal approach, high on the dome of the left hemidiaphragm away from the chest wall. Diaphragmatic activity was spontaneous and thoracic and abdominal sites were tested by temporarily occluding the airway or by inducing coughing. Physiologic activities were recorded on a Brush rectilinear recorder. The four most pertinent variables in each experiment were chosen for simultaneous recording on a Hewlett-Packard magnetic tape recorder for later playback and detailed analyses.

In most cases the animals were suspended from a horizontal bar by stout lines sewn through the paravertebral muscles and ligaments at the pectoral and pelvic girdles. The head was strung with another line passed under the horizontal bar by stout lines sewn through the paravertebral muscles and ligaments at the pectoral and pelvic girdles. Retching was characterized by a growing series of brief negative intrathoracic pressure pulses mirrored by positive pressure pulses in the abdomen. Expulsion then followed as a more sustained abdominal contraction with a sudden reversal of intrathoracic pressure from negative to positive. Remarkably, however, both retching and expulsion were effected by the same set of muscles according to their EMG profiles. Results obtained with spinal cord section at T and phrenicotomy support the conclusions: 1) that the diaphragm, acting in concert with the inspiratory muscles against a closed glottis, is responsible for the negative intrathoracic pressure produced in retching; and 2) that the abdominal musculature is responsible for the positive pressure generated in the abdomen during retching and expulsion, and which in the latter phase is transmitted into the thorax by an upward shift of the diaphragm.

THAT THE RESPIRATORY MUSCLES PROVIDE THE MOTEIVE FORCE IN VOMITING IS BY NO MEANS A NEW CONCEPT. As early as 1686, Chirac (cited by Mayo (15)) found a flaccid stomach compressed by the abdominal muscles and diaphragm with each vomiting effort. John Hunter, who died in 1793, described vomiting in part as being “performed by the abdominal muscles and diaphragm, while those of inspiration are supporting this action” (6). The famous experiment of Magendie (14), in which he substituted a pig’s urinary bladder for a dog’s stomach, confirmed the somatic musculature by itself capable of generating pressures sufficient for vomitting. A complementary role of the gastrointestinal tract has been widely studied especially since the advent of radiography (3). However, neural reflex involvement of the gut is not a functional requirement for vomiting inasmuch as coordinated evacuation of the stomach through the mouth is not prevented by gastrointestinal decervation (22).

A clinical distinction between labored and projectile vomiting has long been recognized. In this connection, Borison and Wang (2) localized separate electrically reactive areas of the brainstem for unproductive retching and for precipitate vomiting in the decerebrate cat, and they defined related behavioral concomitants in intact animals.

Notable efforts have been made over the last half-century to characterize the phasic components of the emetic process by a variety of techniques including electromyography and cineradiography (4, 6-11, 13, 17, 20). The mechanistic difference, however, between retching and expulsion in the act of vomiting has not been satisfactorily resolved. The present study demonstrates dynamic features of retching and expulsion as distinctive events in the vomiting response.
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Characterizations of Retching and Expulsion

At the low amplifier gain required for recording vomiting activity, normal cardiovascular and respiratory variations were barely detectable in the venous pressure recordings. The development of an emetic episode was heralded by a small deflection which could occur first in either the thoracic or the abdominal pressure tracing. Then followed a series of progressively building brief, negative pressure pulses in the thorax mirrored by positive pressure pulses in the abdomen, all of which terminated in a longer combined positive pressure swing in both compartments (Figs. 1, 2). The repetition number of the opposite pressure pulses usually varied between four and six, but as many as 14 were recorded. The thoracic "suction pulse" constitutes the unique physiological action that defines the retching portion of the vomiting response. The retching pulses were generally 0.5 s or less in duration, and at their peak amplitude often exceeded minus 75 mmHg in the thorax coincident with 125 mmHg in the abdomen, adding up to transdiaphragmatic pressures greater than 200 mmHg. These retching movements were not accompanied by opening of the mouth or the forceful discharge of gastric contents. Nevertheless, liquid vomitus did on occasion escape from the mouth during the retching phase.

The expulsion phase of the vomiting sequence was manifested always as a single squeezing action wherein the final abdominal pressure wave was more prolonged and usually greater than the preceding retching pulse. The thoracic component consisted most often of a biphasic negative-to-positive pressure reversal or it could occur simply as a positive excursion following the last negative pulse. The retching pattern appeared typically as a metronomic series of pulses repeating every second or so. The expulsive event typically occurred as a premature wave that cut short the terminal cycle of the pulse train. In contrast to the retching phase, the final expulsion phase was accompanied by wide opening of the mouth, strong flexion of the spine, and forceful ejection of the upper gastrointestinal contents.

Measurements of pressure variations in the cervical trachea were made in some experiments for comparison with the venous pressure observations (Fig. 3). The involvement of the glottis in vomiting, especially in the generation of the negative thoracic pressure during retching, is shown in Fig. 4. On opening a T tube previously tied into the trachea, the negative thoracic venous pressure excursions in the retching phase were markedly reduced yet essentially no change was obtained in the succeeding positive-pressure wave generated in the expulsion phase. This was confirmed in another experiment by a direct intrapleural pressure recording. On the other hand, the positive-going abdominal venous pressure excursions were facilitated in both phases of the vomiting episode, evidently as a result of the freer descent of the diaphragm permitted by the open airway.

Phasic EMG Activity

Intercostal muscles and diaphragm. The external (inspiratory) intercostal muscles and the diaphragm fired in synchrony during the entire emetic episode, and both were in phase with the negative thoracic pulses generated during the retching phase (Fig. 3). Thus, the expanding effort of the chest wall and the diaphragmatic contraction working against a closed glottis produced the negative thoracic pressure that typifies the single retch. The internal (expiratory) intercostal muscles were electrically active between the retching pulses (Fig. 2), but they generated little or no interpulse positive thoracic pressure.
FIG. 3. Two vomiting episodes showing synchrony of diaphragmatic and external (inspiratory) intercostal EMG activities in retching and expulsion. Tracheal pressure went negative with each retching pulse, but was not sustained in positive conversion during expulsion as was more prolonged abdominal compression (see text and other figures for relating effects of tracheal pressure to thoracic venous pressure changes). BP signifies arterial blood pressure. Venous pressure tracing characteristics as in Fig. 1.

We were surprised to find that the positive thoracic pressure wave during the expulsion phase was characterized by the same EMG profile of the intercostal muscles and diaphragm, as were the preceding negative-pressure retching pulses (Figs. 2 and 3). The positive pulse in the trachea verified the positive thoracic venous pressure during the expulsive portion of the emetic response. This seemingly paradoxical EMG observation was consistent in every emetic response obtained under all appropriate experimental conditions and is, therefore, considered to be a valid physiological manifestation of the vomiting act. It means that the diaphragm shifts during active contraction from the descended to the ascended position when retching is converted to expulsion. The upward diaphragmatic shift that takes place coincidentally with expulsion was confirmed by cineradiography (16).

Abdominal muscles. Electromyographic monitoring of the rectus abdominis and the external oblique muscles revealed that these muscle groups are active concurrently with the increased abdominal pressure observed in the retching as well as in the expulsion phase of vomiting (Figs. 1 and 4). This correlation of electrical and mechanical events points to the supporting muscles of the abdomen as generating the primary force responsible for the positive pressure that occurs both in the abdomen and in the thorax at the appropriate times during the vomiting act. Indeed, the thoracic muscles of inspiration were electrically silent in expulsion as well as in retching (Fig. 2). The cardinal role of the abdominal muscles in vomiting is further substantiated by the following results obtained with spinal cord section and phrenicotomy.

Effects of Neural Sections on Emetic-Response Pattern

Spinal cord section. Interruption of the spinal cord at T11, which spared most of the innervation to the muscles concerned in vomiting, was performed in one animal 24 h prior to the emetic test. No qualitative effect of the cord section could be seen in the thoracic and abdominal pressures recorded during either phase of the vomiting response by comparison with the intact spinal cord preparation. A subsequent cord transection was performed at T6 in the same animal. The second procedure severely compromised the positive abdominal pressure changes that normally occurred during both phases of emesis, and it all but eliminated the negative and positive pressure pulses in the thorax despite the remaining action of the diaphragm (Fig. 5). The failure of the diaphragm to generate significant pressures in either the abdomen or in the thorax during vomiting can be attributed to the lack of “splinting” by the abdominal muscles and by the long ribs which allowed the abdomen to distend and the chest to retract upon descent of the diaphragm. Nevertheless, small but appropriate tracheal pressure variations were still in evidence particularly during the retching phase (Fig. 5). It has been our experience in chronic low-cervical cord-sectioned animals being tested with emetic drugs that the vomiting act is facilitated by wrapping a binder around the abdomen (12).
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FIG. 5. Retching and expulsion after spinal cord transections. Left: 2 vomiting episodes showing attenuated (in comparison with cord-intact animals) but phasically typical thoracic and abdominal venous pressure changes after cord section at T11. Right: effect on vomiting dynamics resulting from a 2nd cord section at T5. Tracheal pressure was recorded for its relationship to venous pressures in thorax and abdomen. Venous pressure tracing characteristics as in Fig. 1.

Phrenicotomy. After low-cervical section of the phrenic nerves in one animal, we found during retching that no negative pulses occurred in the thoracic pressure tracing and that the abdominal pressure deflections were considerably reduced by comparison with results obtained with the innervated diaphragm. Furthermore, retching was initiated repeatedly in an abortive manner before expulsion took place. Eventually, a herculean abdominal squeeze followed the retching prodrome, thereby generating an intrathoracic pressure greater than 180 mm Hg and a simultaneous positive-pressure wave in the thorax with an effective expulsion of vomitus (Fig. 6). Thus, contraction of the diaphragm is essential during retching for the generation of negative thoracic pressure and, at the same time, contributes importantly to the generation of the opposing abdominal pressure pulse. The resulting push pull transdiaphragmatic pressure differential undoubtedly serves to move gastric contents into the thoracic esophagus in preparation for the terminal expulsive event. On the other hand, it is still possible when the diaphragm is paralyzed for the abdominal muscles operating against a thoracic air cushion to bring about evacuation of the stomach through the mouth.

DISCUSSION

The use of venous pressure recordings made it possible for us to determine the separate mechanics of the retching and expulsion phases of vomiting in the thorax and abdomen without disturbing the processes under examination. The mechanisms concerned in these processes will be considered for each phase in each visceral compartment.

Retching

Thoracic compartment. Several early authors appreciated the important role of the muscles of inspiration in vomiting, particularly to produce a suction effect in the thorax during retching. However, it has been a matter of contention whether air movement occurs simultaneously with retching. Gold and Hatcher (4) employed a face-mask arrangement in unanesthetized dogs and cats and they showed that no pulmonary ventilation takes place during retching, whereas Jimenez-Vargas et al. (9) recorded air movements with a spirometer attached to a tracheal cannula in decerebrate dogs. The present work in decerebrate cats indicates that a closed glottis prevents respiratory air movement during the retching suction pulse since bypassing the glottis reduced markedly the peak negative thoracic pressure developed in this phase of vomiting. Effective generation of negative intrathoracic pressure during retching requires the concurrent activities of the diaphragm and external (inspiratory) intercostal muscles. The internal (expiratory) intercostal muscles were found to discharge only between retching pulses. Phrenicotomy alone essentially eliminated the retching pulses in the thorax even though the inspiratory intercostal muscles continued to discharge appropriately. Similarly, high-thoracic spinal cord section, which inactivated the muscles between the long ribs, reduced considerably the negative pressure pulses despite the continuing activity of the diaphragm. Thus, the effect of retching in the thorax is to provide repetitively, against a closed glottis, a brief but strong inspiratory effort that serves as the pulling counterpart of a unidirectional push-pull motion for the transfer of gastric contents across the diaphragm into a dilated esophagus. Indeed, cineradiographic observations made by McCarthy et al. (16), and other studies as well (7, 10, 11, 13), have revealed that during retching the fundus of the stomach itself was drawn through the diaphragmatic hiatus with marked cephalad displacement of the esophagogastric junction. However, from the present evidence, we cannot decide whether the forward movement of the stomach is entirely a passive response to external pressures or is effected partly by active shortening of the esophagus (10, 11).

Abdominal compartment. Positive venous pressure pulses observed in the abdomen during retching were closely synchronized with the negative pressure pulses seen in the thorax.
In no instance was a negative pressure deflection recorded from the abdomen. After repeated vomiting, apparently owing to fatigue, a slight lag sometimes developed in the abdominal pulse peak relative the thoracic pulse. However, at no time did the abdominal pulse lead the thoracic pulse during retching. This signifies that contraction of the diaphragm starts off the retching event. The muscles of the abdominal wall provide the major compressing force since phrenicotomy did not noticeably diminish the positive venous pressure peaks. On the other hand, high-thoracic spinal cord section which inactivated the abdominal wall muscles all but eliminated the retching compressions. It is evident that the large negative pressure pulse normally generated in the thorax against a closed glottis places a significant restraining force on the diaphragm (see Fig. 4). In strong contrast to the concomitant inhibition observed in the internal (expiratory) intercostal muscles, the rectus abdominus and external oblique muscles acted in opposition to the diaphragm. Thus, unlike the primary thoracic muscles of respiration which behaved at all times as breathing synergists, the abdominal accessory muscles were controlled as breathing antagonists during the act of vomiting.

**Expulsion**

**Thoracic compartment.** The most remarkable feature of thoracic motor behavior in the expulsion phase of vomiting was the full reversal from maximum negative to maximum positive intrathoracic pressure while the diaphragm and external (inspiratory) intercostal muscles again discharged simultaneously as in the retching phase. Furthermore, the internal (expiratory) intercostal muscles remained silent as well. However, unlike the exact coincidence in onset of the negative pressure pulse with the diaphragmatic EMG in retching, the EMG activity started well in advance of the positive thoracic wave in expulsion. Close examination of the diaphragmatic EMG often revealed a momentary hesitation in the electrical activity upon the transition of intrathoracic pressure from negative to positive. Gold and Hatcher (4) from their work in animals, and Paillard (17) from his work in humans, believed that the diaphragm relaxed during the expulsive event. Jiménez-Vargas et al. (8, 9), on the other hand, maintained that the abdominal muscles and diaphragm exercised opposing forces on the stomach although they did not make a clear distinction between retching and expulsion. We have found during expulsion, as in retching, that the diaphragm continues to be active and the ribs hold in the inspiratory position, except that upon expulsion the diaphragm suddenly shifts its position upwards thereby transmitting into the thorax the pressure that has already been built up in the abdomen. The action of the diaphragm in delaying the intrathoracic pressure conversion to a positive force becomes evident after phrenicotomy where it can be seen (Fig. 6) that the onset of the intrathoracic positive pressure wave during expulsion now coincides exactly with the abdominal pressure wave as well as with the external (inspiratory) intercostal EMG.

Comparison of tracheal and intrathoracic pressures suggested to us the occurrence of airway collapse. The pressure wave in the thorax was sustained during expulsion despite the presence of a tracheostomy (Fig. 4). We interpret this to mean that air was trapped in the lungs. It can be seen in Fig. 3 that the positive tracheal pressure excursion, as contrasted with the abdominal pressure, was not well sustained during expulsion which is consistent with a collapse of the airway between the lung parenchyma and the cervical trachea caused by the sudden development of an inordinately high intrathoracic pressure, i.e., greater than 30 mmHg (18, 19). In fact, the brief positive pressure deflection in the trachea is extinguished at the peak of the positive pressure wave in the thorax. A sustained tracheal pressure rise was observed after spinal cord section (see Fig. 5) since presumably the residual abdominal contraction did not raise the intrathoracic pressure sufficiently to produce complete airway collapse. The involuntary crow that often accompanies expulsion can now be explained as resulting from the forced evacuation of dead space on collapse of the airways rather than from a deflation of the lungs. Indeed, the air cushion formed by the sealed lungs provides the thoracic counterpressure to the abdominal squeeze for ejection of the cephalic and gastric contents.

**Abdominal compartment.** Magendie (14) was the first to show that vomiting could be effected by either the diaphragm or the muscles of the abdominal wall acting independently. That observation has been verified many times and is again confirmed in the present experiments. Nevertheless, the fact that remarkable pressures can be generated in the large body cavities after phrenicotomy, coupled with the fact that the expiratory intercostal muscles are silent during the expulsion phase of vomiting, leads inescapably to the conclusion that the positive pressure wave generated in both the abdomen and thorax originates from a single source, namely the contraction of the abdominal body wall muscles.

In conclusion: 1) The salient distinction between retching and expulsion phases of vomiting is the direction of intrathoracic pressure change, namely, negative in retching and positive in expulsion; the abdominal pressure change is positive-going in both phases. Nevertheless, the electromyograms from the diaphragm and body-wall muscles of the thorax and abdomen do not differ in their spatial pattern of discharges during retching and expulsion. 2) Retching is accounted for by the external (inspiratory) intercostal muscles briefly contracting synergistically with the diaphragm in a periodic fashion as the glottis is held closed, while the abdominal muscles contract phasically in opposition to the diaphragm. 3) Expulsion is accounted for by a more persistent contraction of the abdominal muscles during the course of which a sudden upward shift of the diaphragm transmits into the thorax the pressure already generated in the abdomen. 4) Pulmonary airway collapse evidently occurs as a result of the large positive pressure surge delivered to the thorax in the expulsion phase of vomiting.

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