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EsoTensive Colic: The Screaming Infant

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Abstract

Background: Infantile Colic is considered a benign process in which an infant has paroxysms of inconsolable crying for more than three hours per day, more than three days per week, for longer than three weeks. It affects approximately 10% to 40% of infants worldwide and peaks at around six weeks of age, with symptoms resolving by three to six months of age. The incidence is equal between the sexes, and there is no correlation with the type of feeding (breast *vs.* bottle), gestational age, or socio-economic status. Some colicky infants have attacks of screaming with associated motor behaviours such as flushed face, furrowed brow, clenched fists and legs pulled up to the abdomen. Crying occurs in prolonged bouts. The infants do not respond to normal comforting techniques. Crying onset is unpredictable, spontaneous and unrelated to environmental events. The cause of infantile colic is unknown. It is proposed that there is a form of infant colic, resulting from Mallory Weiss tears near the esophageous-stomach junction, caused by distension of the stomach cardiac orifice during vomiting.

Physiology: Projectile vomiting is associated with severe colic. Very high intra-abdominal pressures are required to propel vomit several feet, greatly exceeding those involved in Gastro-oesophageal reflux disease. These pressures are generated by simultaneous maximal contraction of diaphragm and abdominal wall muscles, compressing abdominal contents. Pressurised chyme overwhelms the lower esophagus sphincter and enters the lower esophagus, distending and possibly tearing it.

Conclusions: A) There is a form of colic caused by Mallory-Weiss tears at the gastro-esophageal junction during violent vomiting. Subsequently, any reflux flowing over the exposed wound will cause sudden pain. B) Screaming will commence largely unrelated to external events, and last until the reflux disperses. C) Diet will have no effect, unless it induces vomiting. D) There will be no gender bias.

Keywords: Shaken baby; Pyloric stenosis; Venous Hypertension; Gender bias; Subdural hematoma; Retinopathy; Crying

Introduction

Johnson et al. [1] describe infantile colic as: *A benign process in which an infant has paroxysms of inconsolable crying for more than three hours per day, more than three days per week, longer than three weeks. It affects approximately 10% to 40% of infants worldwide and peaks at around six weeks of age, with symptoms resolving by three to six months of age. The incidence is equal between the sexes, and there is no correlation with type of feeding (breast vs. bottle), gestational age, or socio-economic status. The cause of infantile colic is not known.*

Roberts et al. [2] states that *estimates of the occurrence of colic in community based samples vary from 5 to 25% of infants. Fussing and crying are normal aspects of development during the first three months of life. During this time, infants cry an average of 2.2 h per day, peaking at six weeks of age and gradually decreasing.*

Colicky infants have attacks of screaming in the evening with associated motor behaviours such as flushed face, furrowed brow and clenched fists. The legs are pulled up to the abdomen, and the infants emit a piecing, high pitched scream. Colic typically begins at two weeks of age and usually resolves by 4 months of age. Crying is concentrated in the late afternoon and evening, occurs in prolonged bouts, and is unpredictable and spontaneous. It appears to be unrelated to environmental events. The child cannot be soothed, even by feeding.

Many authors have developed hypotheses based on various gastrointestinal disorders but none seem to account for the ineffectiveness of normal comforting procedures. This study considers the possibility of mucosal injury occurring during violent emesis or retching.

Vomiting

Vomiting is a fundamental protective mechanism. It has its own dedicated control system in the brainstem medulla oblongata [3]. This has inputs from many sensors, chemical, mechanical, and systemic. Chemical inputs sense noxious substances in the blood; mechanical sensors report distensions of the alimentary tract, such as caused by obstruction by a swallowed foreign object, muscular spasm, or malformation of the tract in the new born. The vomiting centre can assert its own control over the normal respiratory system to ensure the threat is removed. When it calls for maximum effort from the diaphragm and abdominal muscles the vomit may travel several feet, (projectile vomiting). This indicates a very high intra-abdominal pressure which will also act on all blood vessels and reservoirs in the abdomen, and hence to arteries and veins leading out of the abdominal space. Arteries are developed to withstand over pressure but veins are not. Venous Hypertension can result in subdural haemorrhage,

Valsalva Retinopathy, and diffuse cerebral edema so such action is not without risk. However evolution has found that for our species the risk is worth taking.

Mallory-Weiss Tears: a source of pain

Tearing of the cardiac orifice of the stomach and the adjoining esophagous during vomiting [4] was discovered by Mallory and Weiss during a study of alchoholic adults in whom vomiting was frequent. Weiss describes their findings as: *The chief clinical manifestations of these lesions were persistent vomiting and retching, frequently precipitated by alcoholic debauches and associated with massive hematemesis. The history of these patients revealed similar previous attacks. In four of the patients the hemorrhage was so severe that shock and death followed. Autopsy revealed from two to four fissure-like lesions of the mucosa, characteristically arranged around the circumference of the cardiac opening, along the longitudinal axis of the esophagus. The lesions extended up into the esophagus or down into the cardiac opening of the stomach. In some of the sections ruptured arterioles were observed: in others small veins were found.*

In analysing the development of these lesions, it was concluded that pressure changes in the stomach during the disturbed mechanism of the coordinated motor changes which accompany vomiting, and continuous regurgitation of gastric juices over the mucosa of the cardia are the most significant responsible factors.

Gastroesophageal junction mechanics

The upper part of the esophagus descends between the pericardium and the spine in the mediastrum to which it is attached. The esophagus passes through its own aperture in the diaphragm. It is loosely located by elastic fibres [5,6] in the phrenoesophageal ligament [7]. The phrenoesophageal ligament is a relatively compliant and elastic structure which allows the esophagus to slide relative to the diaphragm. In the adult the gastroesophageal junction moves approximately 2 cm in the cranial direction during a swallow.

Below the diaphragm, the esophagus is attached to the cardia aperture of the stomach. This junction is not a simple end-to-end join, it is more of a splice. The junction follows a zig-zag path around the esophageal and gastric cardia tubes. This junction is known as the Z-line (6). In the adult this transition region extends over 3-4 cm. Locally, histology of the Z-line, marks an abrupt structural change. The esophageal side has a stratified epithelium, but the gastric side has a columnar epithelium [6,8]. This means that tissue on each side of the Z-line will have different stretch characteristics. A stress concentration will be set up whenever the esophagus is stretched. If the diaphragm pulls the stomach down, the gastroesophageal junction will get pulled down with it, stretching the esophagus longitudinally.

The lower end of the esophagus is surrounded by muscles forming a sphincter [5]. This is not a true sphincter, i.e. a ring of smooth muscle cells like the pyloric sphincter, it is a collection of muscles with a sling-like action which combine to form a sphincter-like action [8]. Normally this sphincter only has to withstand a small pressure drop for which this arrangement is adequate.

Figure 1a represents the normal situation and Figure 1b the situation early in a vomiting event. In Figure 1b the diaphragm has descended. This may have been preceded by constrictions of the small intestine (duodenum, ileum) bringing their contents back into the stomach to be ejected [3]. When the diaphragm is maximally shortened, (maximum tension) the lungs get sucked down. Some authors describe this as a "deep breath" [9], but this not a normal inspiration, it is under the control of the vomiting centre.

The site of injury
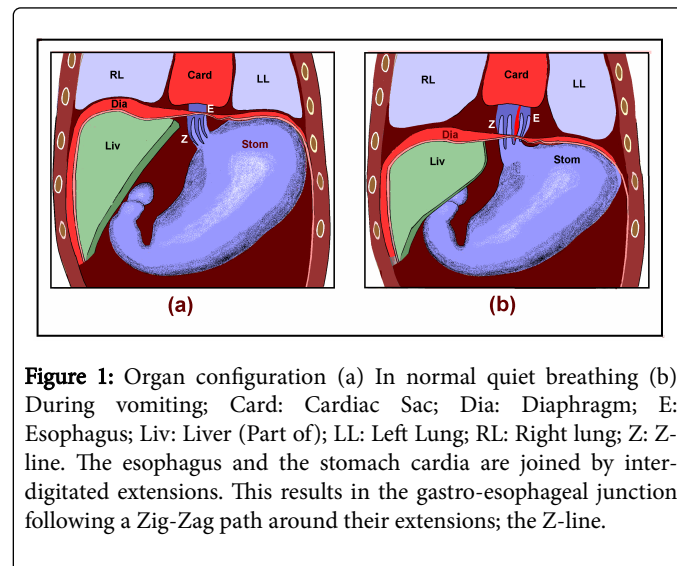
Figure 1: Organ configuration (a) In normal quiet breathing (b) During vomiting; Card: Cardiac Sac; Dia: Diaphragm; E: Esophagus; Liv: Liver (Part of); LL: Left Lung; RL: Right lung; Z: Z-line. The esophagus and the stomach cardia are joined by inter-digitated extensions. This results in the gastro-esophageal junction following a Zig-Zag path around their extensions; the Z-line.

The abdominal walls are also tensioned to form a container in which to generate maximum expulsive pressure on the abdominal contents. Normally the lower esophageal sphincter is closed unless eating or drinking occurs. However, with the diaphragm and abdominal muscles at maximum tension the intra-abdominal pressure will be very high, forcing the lower sphincter open. Chyme at high pressure can then force its way into the esophagus and dilate it. This stretches the walls of the stomach cardiac orifice and the attached local esophagus. The walls of the stomach are lined with rugae which can unfold to accommodate stretch in the associated muscle layers. The mucosas in the walls of the esophagus are lined with plicae [10]. Plicae cannot unfold, and so are less elastic. Such joins of materials of different elasticities are known to produce stress concentrations when stretched. When the Z-line region is excessively stretched by the lumen pressure the mucosa may tear (Mallory-Weiss tears) [11-14]. Once the mucosa is torn it will heal in a few days if undisturbed [11], but if another surge occurs the wound will be re-opened. In the mean-time any reflux will bring acid stomach contents into contact with the wound leading to erosion [14], as Weiss found [4].

Discussion

Various hypotheses suggest that the lower esophageal sphincter relaxes to allow chyme to enter the esophagus during projectile vomiting. However, when the diaphragm and abdominal muscles are simultaneously driven hard by the vomiting centre in the medulla oblongata, extremely high intra-abdominal pressures are produced. It would be difficult, and probably unethical, to attempt to measure intra-abdominal pressure in an infant during projectile vomiting, but its magnitude is demonstrated by the several feet distance the thick liquid vomit is thrown. To achieve the necessary exit velocity from the mouth must require considerable intra-abdominal pressure to drive it.

Such pressure will be more than enough to overcome the relatively weak lower esophageal sphincter. This is logical, since the vomiting centre exists to deal with emergencies and cannot afford to be impeded

by any other malfunctioning control system. Such pressure will distend the cardial orifice and the lower esophagus causing Mallory-Weiss tears. Thereafter any minor reflux will be sufficient to produce profound "heart-burn" and start the infant screaming. The infant will continue to scream whenever stomach acid refluxes over previously torn mucosa. The time at which this happens is determined by internal events with little or no dependency on external factors. The initial tear will have occurred during an intense expulsive event, which, if the upper esophagus is clear would have been seen as projectile vomiting.

If the esophagus lumen is narrow or obstructed the vomit cannot escape fast enough and the full force the diaphragm and abdominal muscles exert will continue to produce a sustained high intra-abdominal pressure. This will force blood out of the abdominal organs into the systemic circulation, raising systemic venous pressure, as demonstrated by the 'red' [15] or 'flushed' [2] face reported in descriptions of projectile vomiting.

During the first few months of post-natal life the pylorus is transforming from a relatively long thin walled tube into a very muscular shorter length of alimentary tract [16]. During this development, spasm of the pyloric muscle is known to simulate pyloric stenosis, even before the pyloric sphincter is fully formed [17,18].

Medication

No medications have yet been found to treat excessive crying in infants [2], except for a possible decrease in its incidence produced by antispasmodics. This is to be expected since once the tears have occurred antispasmodics will have little effect on the reflux of acid over the wound. Some form of protective coating similar to that developed for peptic ulcers might help.

Conclusions

A) There is a form of colic caused by Mallory-Weiss tears at the gastro-esophageal junction during violent vomiting. Subsequently, any reflux flowing over the exposed wound will cause sudden pain, initiating crying.

B) Screaming will commence, largely unrelated to external events, and last until the reflux disperses.

C) Diet will have no effect, unless it induces vomiting.

D) There will be no gender bias.

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