



# Duodenal Ulcer-The Baby Version Why Pyloric Stenosis of Infancy (PS) Occurs

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Received: Jul 26, 2023

Accepted: Aug 17, 2023

Published Online: Aug 24, 2023

Journal: Annals of Gastroenterology and the Digestive System

Publisher: MedDocs Publishers LLC

Online edition: <http://meddocsonline.org/>

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**Keywords:** Neonatal hypergastrinaemia; Neonatal hyperacidity; Cause of Pyloric Stenosis of infancy; Immature negative feedback between gastrin and acid secretion at birth.

## Introduction

The connection between Pyloric Stenosis of Infancy (PS) with Duodenal Ulcer (DU) in adults is undeniable.

Both occur predominantly in males and in the same ratio-5/1.

Both have a familial basis [1] and both have hyperacidity [2,3,4] which is fundamental to pathogenesis. In both conditions blood group O-the blood group associated with hyperacidity -predominates [5,6] and presumably reflects a parietal mass at the top of the normal range. Indeed, PS has been shown to occur in new born puppy dogs when gastric hyperacidity is caused by pentagastrin injections [7].

P.S. babies not uncommonly have shallow pyloro-duodenal ulceration equivalent to adult D.U. patients and both have the capacity to develop outlet obstruction for similar but not identical reasons.

The helicobacter pylori infection so common with D.U. presumably unmask the greater parietal cell mass in male patients since the rate of infection is similar in adult females and they have a smaller incidence of hyperacidity and DU.

The big difference between PS and DU patients is of course the early developmental changes necessary in the emerging neonate to safely cope with the challenges of leaving the womb.

The remainder of this manuscript relates to these challenges. The mechanism involved protects most normal babies from gastro-intestinal infections while avoiding the later onset of hyperacidity problems. In this context, the baby who inherits its hyperacidity and is destined to develop PS, may become an inevitable victim of a system designed to protect the majority.

## The New Normal Neonatal Gastro-Intestinal Physiology

Immediately on birth, the gastric contents are neutral or alkaline due to swallowed liquor animi. Within a few hours after



**Cite this article:** Rogers IM. Duodenal Ulcer-The Baby Version Why Pyloric Stenosis of Infancy (PS) Occurs. *Ann Gastroenterol Dig Syst.* 2023; 6(2): 1077.

gastric aspiration there is a temporary short acting wave of gastric acid secretion which is followed by a slow increase in acidity from Day 10 [8] (Figure 1).

Maternal gastrin transfer during labor via the placenta causes the temporary early acidity [9,10]. The later rise in acidity is associated with a progressive rise in neonatal fasting plasma gastrin- at the same time as acidity is increasing. This contravenes normal adult physiology where gastrin and acid have an inverse negative relationship which results in mutual restraint [11].

An increase in gastrin and gastric acidity at the same time is only found when gastrin secretion is unrestrained and is driving acid secretion such as in the Zollinger-Ellison syndrome.

Why should new born babies demonstrate independent and unrestrained gastrin secretion?

In 1974 when we discovered and reported neonatal hypergastrinemia which increased after birth, we proposed that the usual inverse relation between gastrin and acidity was not present and took time to mature [12]. If this were true then neonatal gastrin secretion, pre-maturity, would be always maximal within neonatal capacity to secrete it and the following conditions would be expected.

- A. The usual post-feed gastrin increase would not occur until the negative feedback matured.
- B. At maturity, gastrin would fall and a post-feed rise in gastrin would occur.
- C. At maturity, there would be a peak in acid secretion before both acid (and gastrin) fall.

All three prophecies have been shown to be true [13-15]. This is all the more impressive since these studies were all observational studies, not one of which was designed within a "no negative feedback) hypothesis or indeed any other hypothesis! (Figure 2) [14].

These phenomena are not confined to human babies. Beagle puppy dogs display the same time sensitive changes in fasting and post-feed gastrin as well as acidity in this further simple observational study only.

Before the maturity of the negative feed-back phenomenon (in the Beagle's case at 7 days) pentagastrin did NOT increase acidity. Only after maturity was a response detectable-when acid secretion was no longer being maximally stimulated [16]. Not only does gastrin increase but acid as a consequence is being maximally secreted before maturity as well.

The reader should not be surprised to learn that that PS itself is not confined to human babies. Some breeds of puppy dogs and foals are also affected [17].

Agunod et al in 1969 graphically illustrated the change in acid secretion (and pepsin and intrinsic factor) from birth to 2 months of age in human babies. All parameters showed a temporary peak between 14 and 17 days before falling again-presumably when the feed-back system kicked in. Thereafter a gradual slow rise took place as the gastric mucosa naturally developed with time [15].

**The New Neonatal GI Physiology and PS**

What happens when a baby inherits an enhanced capacity to secrete acid.

The ability of the gastric mucosa to secrete acid appears to be directly related to birth weight [8]. Bigger babies have naturally more acid-and big babies more frequently develop PS [18].

Like their adult DU counterpart, these PS babies will be hungry and feed well in the beginning. Such big, bouncy babies meet the classical description of the baby destined to develop PS.

The pyloric sphincter hypertrophy which causes gastric hold up is caused by repeated vigorous contraction.

Acid entering the duodenum is the most potent cause of pyloric sphincter contraction [19,20]. The high neonatal gastrin levels have a G.I. trophic effect which rapidly transforms repeated contraction into the classical pyloric "tumor".

There are two main stimulants of sphincter contraction

1. **Hyperacidity [19].**
2. **Frequent feeding [21] (Figure 3)**

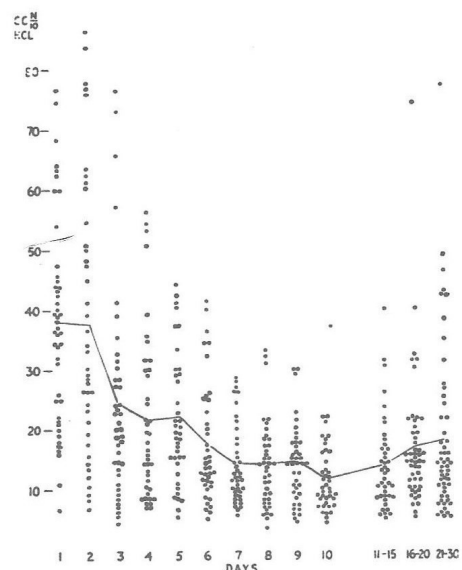
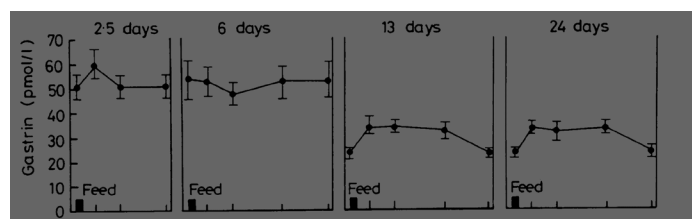


FIG. 1.—The total acidity of the fasting juice during the first month of life, expressed in c.c. N/10 HCl per 100 c.c. stomach contents.

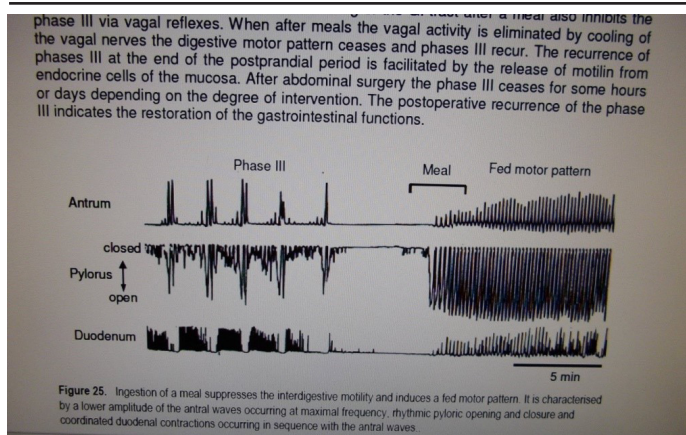
\* Working under the auspices of the Kirk Duncanson Research Fellowship of the Royal College of Physicians, Edinburgh.

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**Figure 1:** The above graph only measures total acidity with an endpoint to phenolphthalein (pH.<>10). The maximum acidity in fasting juice was reached within 24 hours of birth. Reuse granted by kind permission from BMJ publishing. © BMJ Publishing Group Ltd. All rights reserved."



**Figure 2:** In premature human babies, fasting gastrins are high before 13 days of life. After this the levels fall-and-for the first time-a post-feed increase in gastrin can be measured. © BMJ Publishing Group Ltd. All rights reserved."



**Figure 3:** The contractions of the pyloric sphincter are hugely greater in frequency and amplitude after a meal. Ingestion of a feed suppresses the interdigestive motility pattern and induces a fed motor pattern in this experimental model. The fed pattern is characterised by lower amplitude antral waves and pyloric sphincter contractions which are hugely increased in amplitude and frequency. (middle graph). Tutorial on Gastro-intestinal Motility H.J.Ehrlein and M. Schemann by permission from M.Schemann).

### Hyperacidity

PS babies hypersecrete acid [3]. What is even more interesting is that one week after pyloro-myotomy when gastric hold-up is relieved, gastric hypersecretion of acid persists [28].

The theory of cause also states that babies destined to develop PS also are hyperacid. It is only when naso-gastric tubes are required for other reasons that ethical acid studies can be done. The acid secretion of premature infants who require tube feeding have been tested. Premature baby boys secrete much more acid than matched baby girls [22].

### This explains the male preponderance in PS

#### Frequent feeding

First-time mothers are likely to repeatedly feed their vigorous hungry but vomiting baby while more experienced mothers are likely to withhold feeding for a while.

When PS develops, babies who had been fed routinely at 3 hourly intervals, develop symptoms earlier than those fed 4 hourly [23].

The strange first-born preponderance now has a credible explanation. It also explains the increased incidence of PS in summer months (hot babies are likely to drink more milk) and the increase in bottle fed babies. It is easier to overfeed when bottle fed.

The baby who inherits a relatively large parietal mass inherits the potential for hyperacidity and would develop critical hyperacidity during peak acidity time of 3 weeks of age in normal development. The beginnings of repeated sphincter contraction would stem from this point.

Thus-the family history and the classical onset of symptoms at 3-4 weeks should not surprise us. Nor should the occasional spontaneous cure when babies survive long enough for acid secretion to fall when the negative feed-back has become mature [24].

Medical treatment with atropine- like drugs and relative underfeeding does work presumably on the basis of reduced

acidity. Acid blocking drugs such as lansoprazole-quickly quickly restores acid-base status pre-operatively and its temporary use can produce a long-term cure in the early case [25,26].

Long term cure after pyloromyotomy, medical treatment or self-cure-previously rather difficult to understand, now becomes easy.

Hypertrophy requires sphincter contraction. No sphincter-no contraction-no hypertrophy and hence no recurrence. Once negative feed-back has matured-normal service is resumed.

### Darwin and Natural Selection

This combination of a maternal boost to early acid defense and absence of a negative feed-back at birth persists because it offers to the great majority of babies a survival advantage. The early temporary wave of gastric acidity fills the gap in acid defense from a primitive gastric mucosa while it awaits its own gastrin sourced neonatal acid secretion and development.

The unrestrained neonatal hypergastrinemia squeezes out the maximum secreting ability from the early mucosa and the high gastrins also aid mucosal development.

Thus, during this vulnerable time GI infections are reduced because of the maintained acid defense.

The clever aspect of this system is that it does not subject the normal baby to hyperacidity problems in later life.

The baby destined to develop PS from inherited hyperacidity is unfortunately a victim of this otherwise splendid survival mechanism.

When he brings his enhanced parietal mass to this system, the critical hyperacidity created at around 17 days of age, begins the hypertrophic process.

For the PS survivors their only legacy is an increased frequency of hyperacidity problems in late childhood or adult life.

Indeed-finally this baby form of D.U. patient transmutes into his adult counterpart! [27].

Readers who may wish to learn more about this subject may do so by consulting.

The Cause of Pyloric Stenosis of Infancy First Edition 2021. Ian Munro Rogers FRCS Elsevier Publications.

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