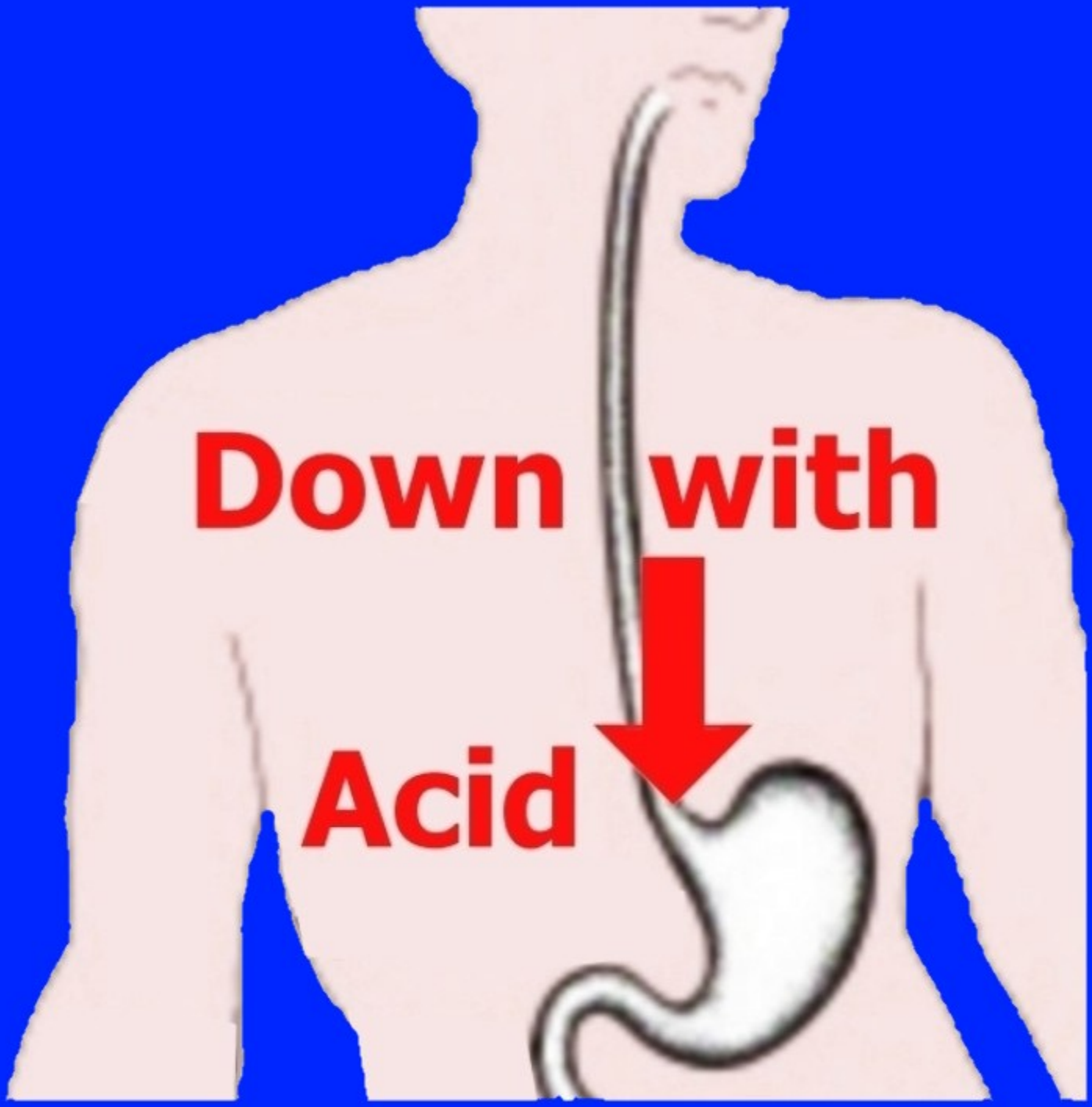
Down with Acid



lite

By Chris Robinson

Down With Acid *Lite*

A selection from the popular encyclopaedia, www.DownWithAcid.org.uk

Contents

The Down With Acid encyclopaedia first printed in 2015 but has now grown too large, and updated too frequently, to be printed in full.

This slimmed down edition attempts to provide the most popular pages in a handy sized book.

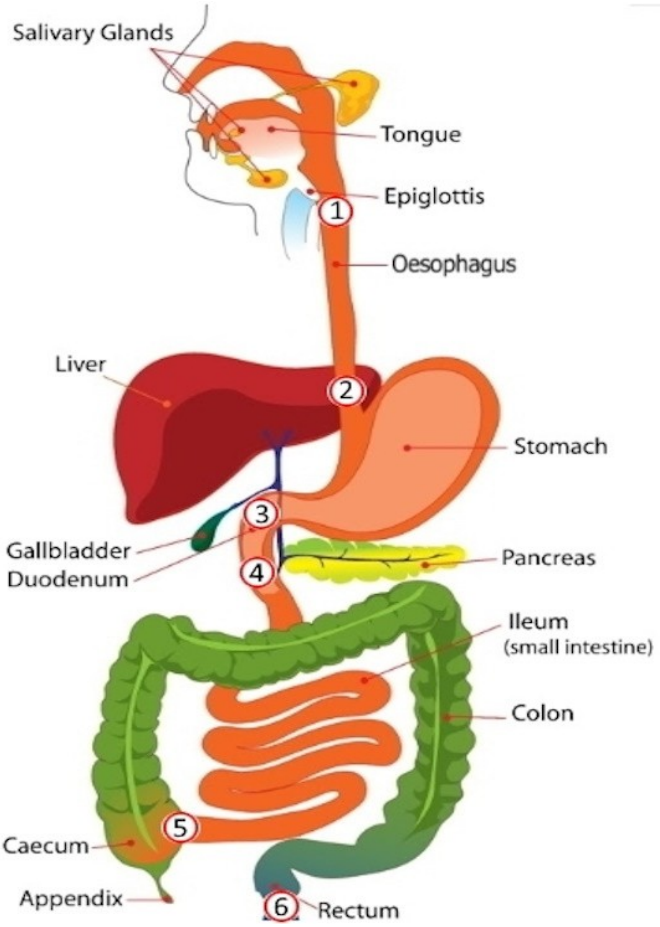
To learn more about the chapters in this book and for information regarding newer techniques and understanding, visit the online version which also includes links to abstracts of the scientific papers quoted.

Disclaimer

The content of this book is intended for patients and health care providers and is provided for information only. Information provided should be discussed with a qualified medical practitioner if readers have or suspect they have a health problem as it is not a substitute for direct professional medical advice or diagnosis.

Digestion Story

The GastroIntestinal Tract:



KEY

1 Upper Oesophageal Sphincter

2 Lower Oesophageal Sphincter

3 Pyloric Sphincter

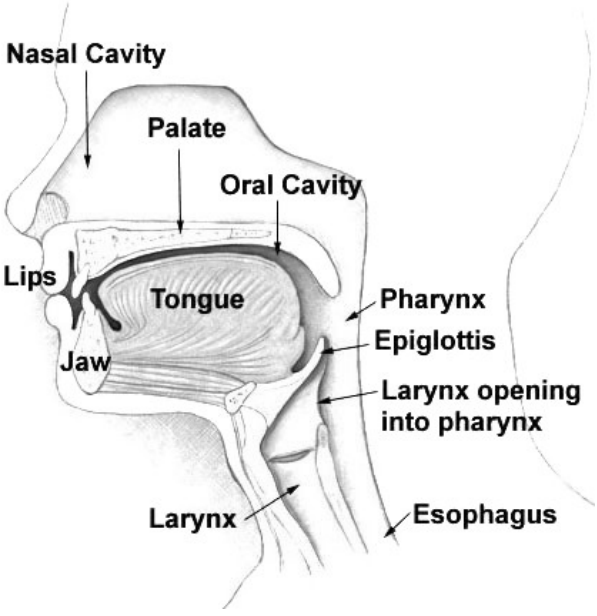
4 Sphincter of Oddi

5 Ileocecal Sphincter

6 Anal Sphincters

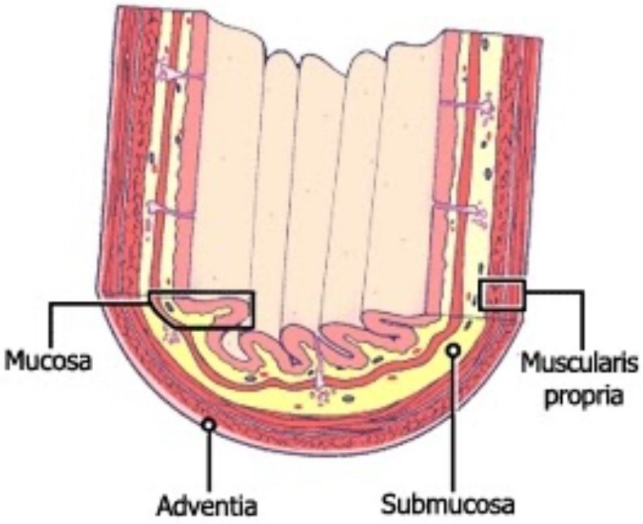
Part 1: We chew food and mix it with saliva, passing it as a bolus through the pharynx and the Upper Oesophageal sphincter in to the oesophagus.

Stimulated by how much you chew, some of the enzyme amylase is produced with the saliva to help break down starches.

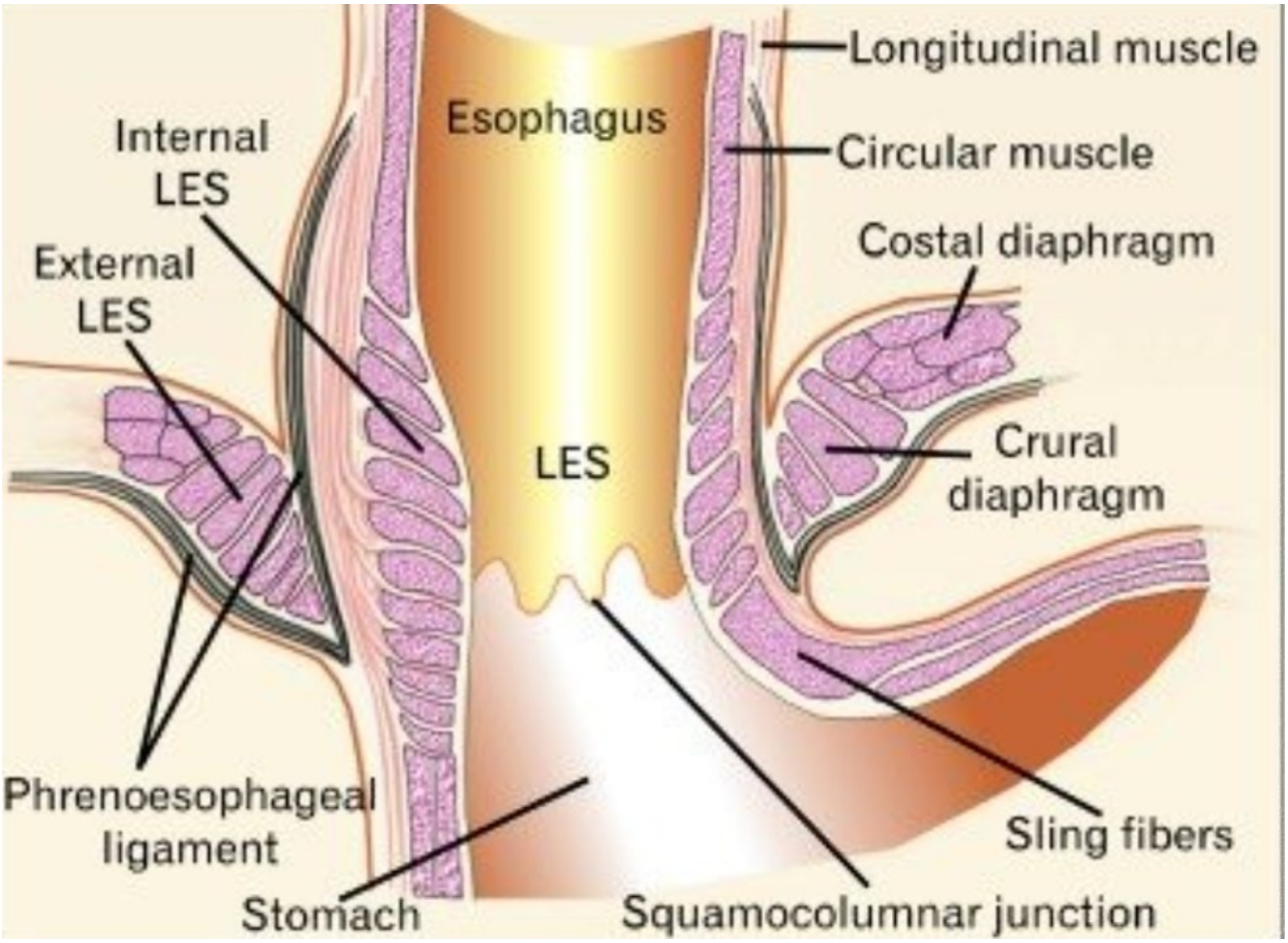


Part 2: The bolus slides down the oesophagus with the help of muscles in an action known as peristalsis,

Muscles above the food bolus contract while muscles below relax to help propel the food along the oesophagus.



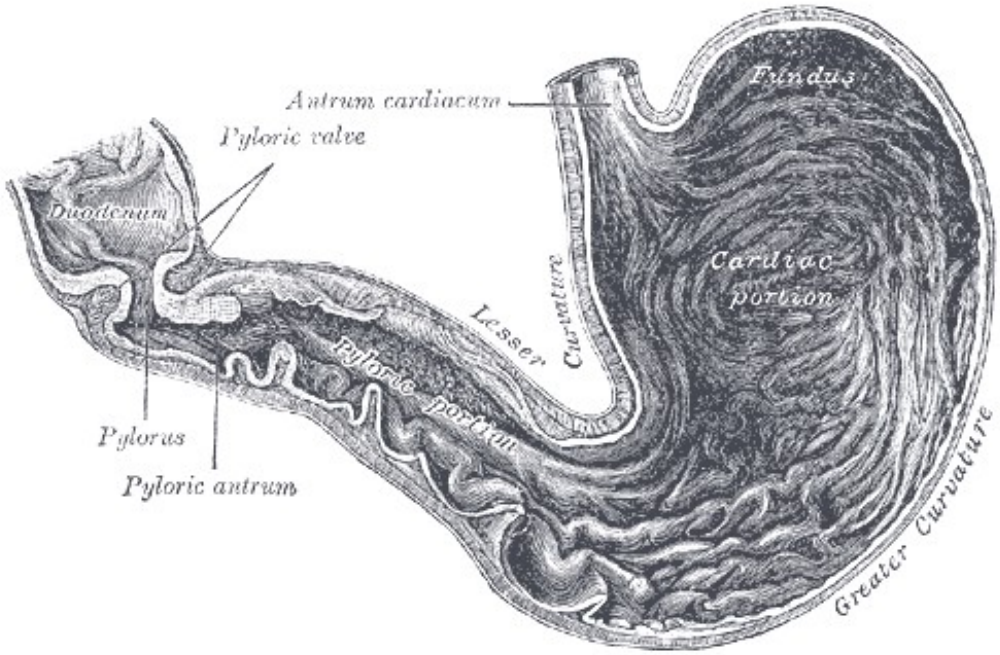
to the Lower Oesophageal Sphincter (LOS / LES) which relaxes to permit the bolus to enter the stomach.



This is the sphincter usually thought of when people speak of Acid Reflux or Gastro-Osophageal Reflux Disorder (GORD).

Part 3: In the stomach the bolus is churned with strong acid and some of the enzyme pepsin to break it down to a thick liquid chyme to pass on through the pyloric sphincter to the duodenum and small intestines where the most important processes of digestion, assimilation, take place.

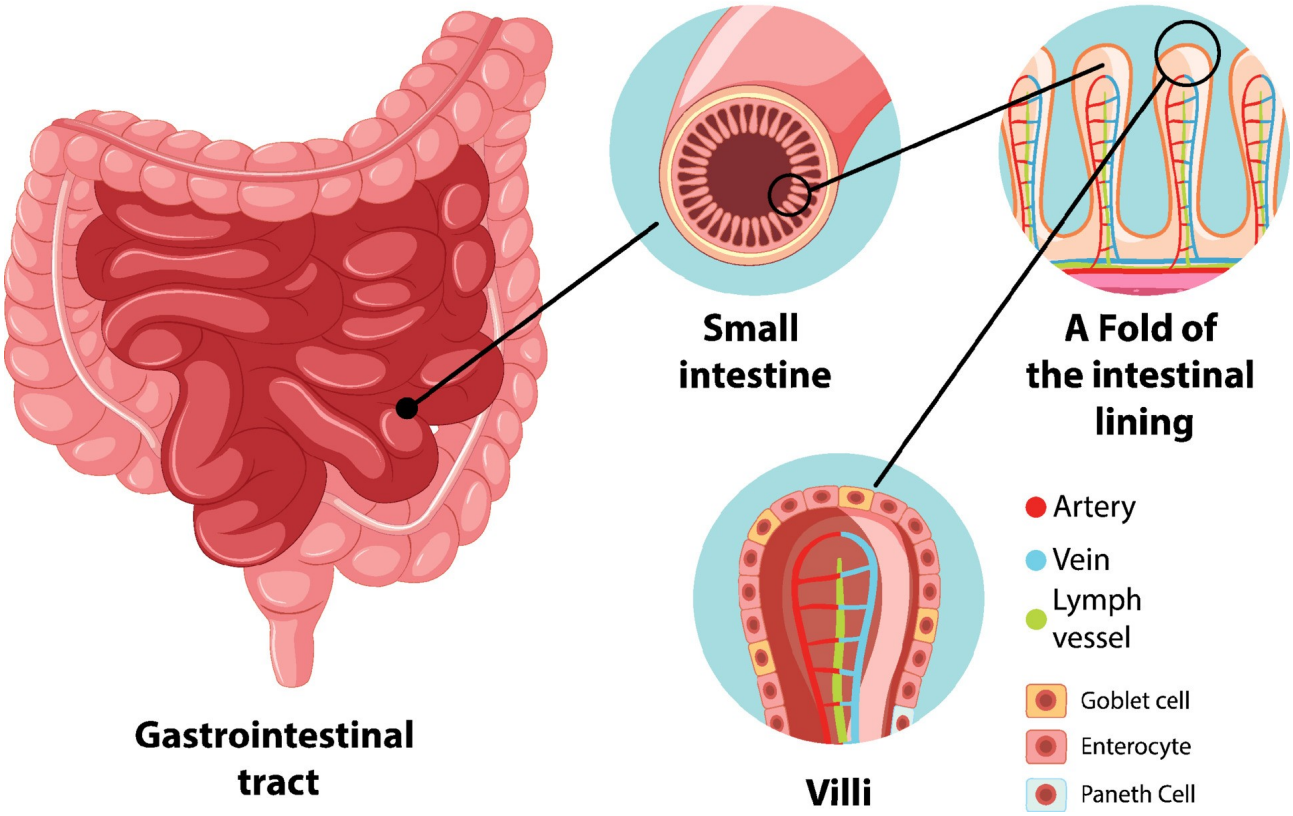
Food can spend up to 4 hours in the stomach depending on its consistency.



Stomach with Pyloric Sphincter (Pylorus) to the right.

Part 4: In the duodenum, digestive enzymes produced in the pancreas, including amylase to help break down carbohydrates and protease to help break down proteins along with bile from the gallbladder where it is stored, enter via the Sphincter of Oddi to commence the real part of digestion. Bile helps break down fats, acting like a detergent as acid doesn't dissolve fats, and neutralising any excess remaining acid, with help of sodium bicarbonate also produced by the pancreas.

It then passes into the intestines which are lined with millions of villi, microscopic hairlike protuberances where nutrients are transferred to the small blood vessels they contain so they may be transported to the kidneys and liver to filter and send to the parts of the body that require them.



Chyme can take upto 4 hours traversing the small intestines.

Part 5: The Ileocecal sphincter marks the end of passing nutrients into the blood to the beginning of receiving waste products from the blood to add to the remaining indigestible material that continues through the intestines.

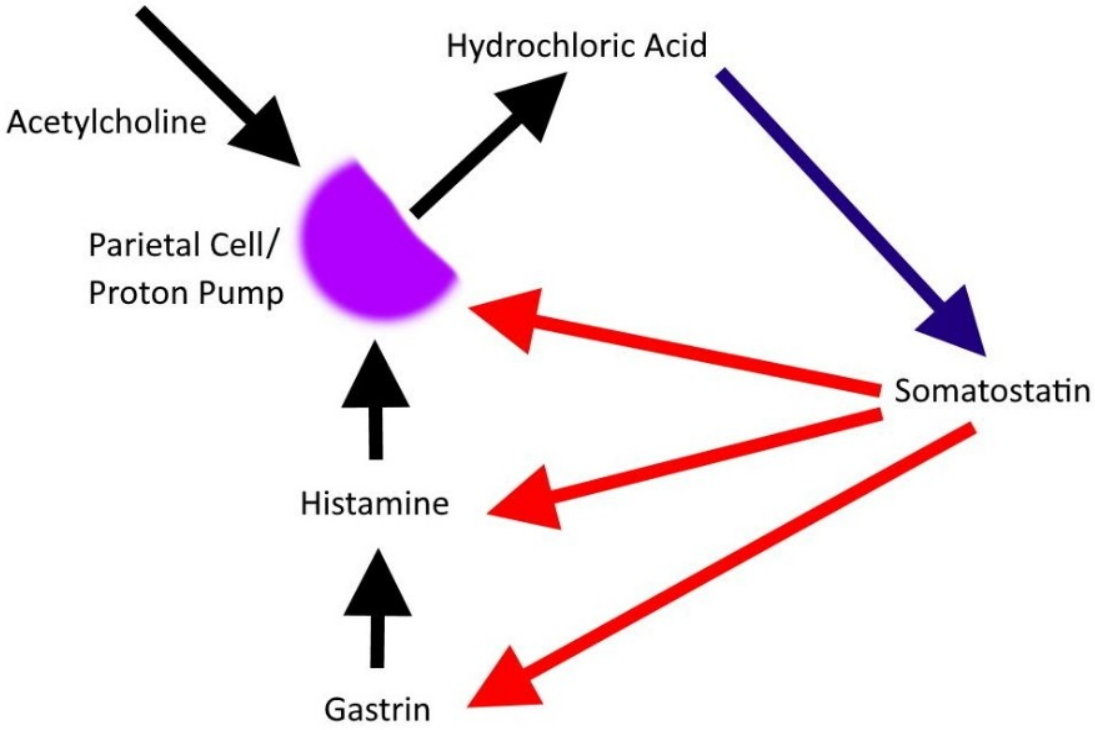
Part 6: Waste builds up and is contained until we are ready to excrete it via the Anal Sphincters**.**

Acid

The stomach is lined with parietal cells.

In response to sight, smell, taste on thought of food, the brain sends signals to activate acid production involving the neurotransmitter acetylcholine which causes the cells to change, becoming Proton Pumps that release hydrogen ions and make Hydrochloric Acid.

As food enters the stomach, the hormone, Gastrin is released due to the stretching of the stomach and the peptides and amino acids in the food. In turn Gastrin triggers Histamine release which, picked up by H2 receptors in the parietal cells, also stimulates acid production.



Somatostatin acts to inhibit acid production to maintain the correct balance of acidity by acting directly on the proton pumps as well as gastrin and histamine production.

For most people suffering acid reflux, the amount of acid produced is probably not the problem, it’s that the acid is refluxing to an unprotected location.

Reflux of acid is not due to too much acid but a weak Lower Oesophageal Sphincter.

Reducing Acid

Reflux of acid from the stomach is via an incompetent Lower Oesophageal Sphincter into the oesophagus which it burns causing oesophagitis inflammation which is the source of heartburn symptoms.

Once oesophagitis exists, some foods passing over it may further irritate it. These are your particular trigger foods that can vary from person to person but typically include acidic drinks and spicy foods.

Reducing heartburn

Simple **antacids** will neutralise acid in the oesophagus to provide fast relief but they are only a temporary measure, like putting a sticking plaster on a wound that needs a stitch. A permanent solution requires the oesophagitis inflammation to heal.

Note: Antacids neutralise acid in the oesophagus *not* the stomach!

To permit oesophagitis to heal:

- Avoid any foods you have identified as a particular trigger for you.
- Take an acid suppressant: either an “**H2 blocker**” such as famotidine, a “Proton Pump Inhibitor (**PPI**)" such as omeprazole or a Potassium Competitive Acid Blocker (**PCAB**) such as vonoprazan.
  Note. These are powerful drugs that must be taken strictly in accordance with the printed patient information leaflet and for no longer than a couple of weeks without a doctor’s approval. It’s not that the drugs are harmful but they can mask the symptoms of oesophageal cancer.
  These medicines do not reduce reflux - they just make it less damaging.
- Reduce reflux by lifestyle modifications.

Antacids & Suppressants

Antacids

These work immediately on excess acid in the oesophagus. They do not prevent acid reflux occurring.

They are drugs that neutralise the acid. Most commonly they are made of

1. **Chalk**, calcium carbonate. Examples are Tums or Rennie. Chemically, this reaction takes place:

$CaCO_3 + 2HCl \rightarrow CaCl_2 + H2O + CO_2$

(Calcium Carbonate + Hydrochloric acid gives Calcium Chloride (a harmless salt) plus water and carbon dioxide).

Warning. Excess calcium is filtered out of the blood by the kidneys where it may accumulate to form kidney stones. Excess calcium can also lead to calcification of the arteries which can cause myocardial infarction, heart attack.

2. **Baking Soda** (Sodium Bicarbonate) based, Example Alka Seltzer.

$NaHCO_3 + HCl \rightarrow NaCl + H_2O + CO_2$

(Sodium bicarbonate (baking soda) + Hydrochloric (stomach) acid gives Sodium Chloride (common table salt) plus water and carbon dioxide gas).

Warning. Too much sodium in the diet can lead to **high blood pressure, heart disease, and stroke**.

Alginates

Gaviscon is the brand name of the white milky liquid that floats on the stomach contents as oil floats on water to reduce the possibility of reflux whilst also providing a temporary protective film to the lower oesophagus and neutralising the acid with an antacid component. (Some generic versions are now available, such as Peptac.)

Acid Suppressants

Unlike antacids, these do not neutralise acid already produced but prevent the stomach producing more.

They are best taken when the stomach is low on acid and about to make more. Half an hour or so before breakfast is optimal.

They do prevent acid reflux occurring.

H2 Blockers (or Histamine H2 Receptor Antagonists, H2RAs)

Examples: ranitidine (Zantac), famotidine, cimetidine.

These work to block the H2 receptors that receive histamine signals that tell the stomach to produce acid working proactively to reduce the amount of acid by preventing histamine signals reaching the parietal cells telling them to make acid.

They are not an instant antacid and are often prescribed to be taken in the evening to reduce nighttime acid.

They are generally effective for up to 12 hours.

In 2019, some batches of Zantac were found to have a higher level of NDMA than they should. Although there is no evidence to prove it, NDMA (N-nitroso dimethylamine) is thought to be carcinogenic. Ranitidine was removed from the shelves. The levels of NDMA found, however, were no higher than found on grilled meat. Further investigaton showed the samples were tainted by incorrect storage.

Proton Pump Inhibitors (PPIs)

Examples: omeprazole, lansoprazole, pantoprazole, esomeprazole (Nexium) – Research evidence has shown all PPIs are as effective as each other when taken in equivalent dose. (20 mg omeprazole = 30 mg lansoprazole = 40 mg pantoprazole = 20 mg esomeprazole.)

Side Effects

Side effects are rare but the most common include headaches, diarrhoea and constipation.

If affected, switching to an alternative brand usually works.

Associations

There have been claims PPIs are associated with more serious side effects, however, PPIs have been shown *not* to be the cause.

Adverse claims have been propagated by the sensationalist media and lawyers but a correlation is not a causation. It is merely a “Umbrella Conundrum”.

The most commonly propagated myths are described below:

Cardiovascular risks are mostly caused by excess weight which can also promote acid reflux, hence the PPIs.

A paper from the American Journal of Gastroenterology found no link between PPI use and risk of cardiovascular events, the meta-analysis of randomised controlled events, Researchers included data from 52 placebo-controlled trials, with 14,988 patients finding "Cardiovascular outcomes were infrequent in randomized trials of PPIs."

Dementia is related to aging. As we age, we lose muscle tone which can result in more noticeable acid reflux, hence a greater likelihood of taking PPIs.

A study, "Association of Proton Pump Inhibitor use with Incident Dementia and Cognitive Decline in Older Adults" concluded, "In adults ≥ 65 years of age, PPI and H2RA use were not associated with incident dementia, CIND, or decline in cognition over time. These data provide reassurance about the safety of long-term use of PPIs among older adults."

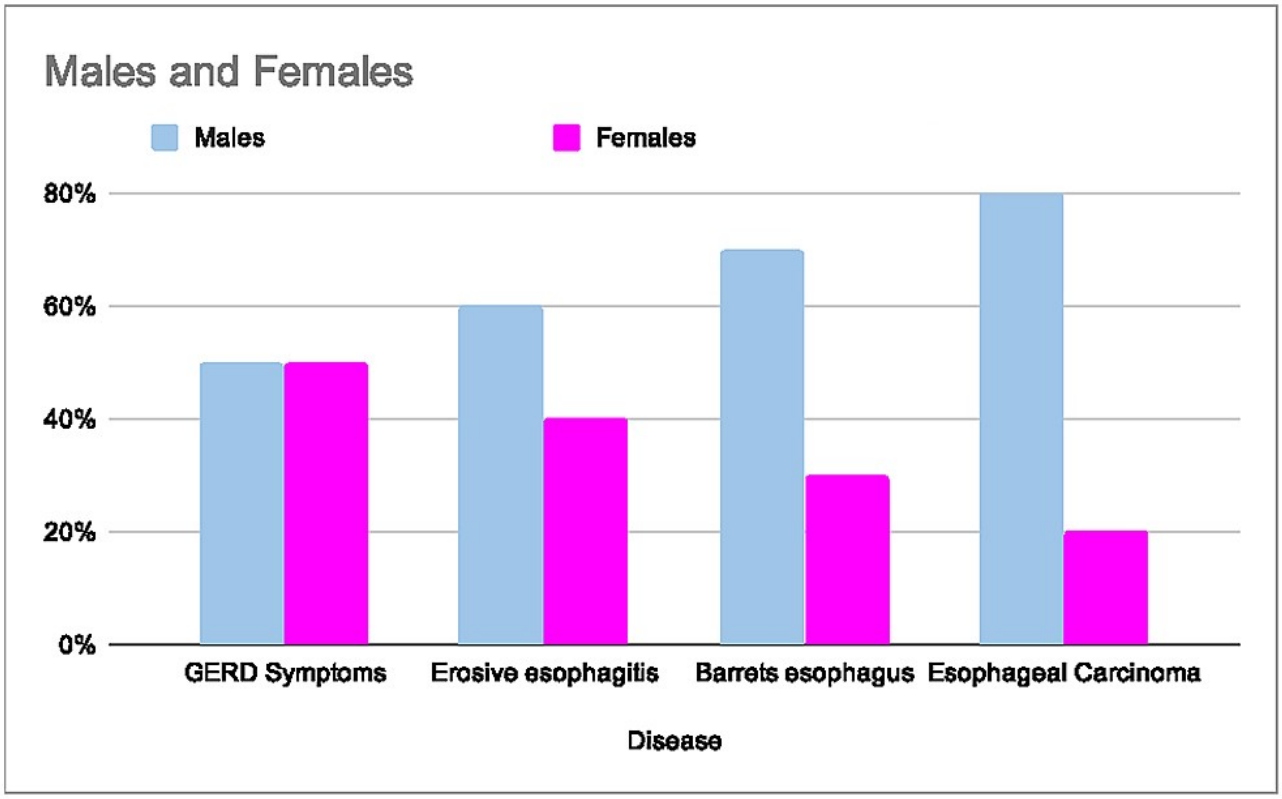
Kidney diseases may be similarly caused and carry acid reflux symptoms prompting PPI usage.

"The causality between PPI use and increased mortality and disease risk can be questioned since most studies are observational. Confounding

variables can greatly affect an observational study and explain the wide-ranging associations with the use of PPIs. Patients on PPIs are generally older, obese, sicker with a higher number of baseline morbidities, and on more medications than the compared PPI non-users. These findings suggest that PPI users are at a higher risk of mortality and complications based on pre-existing conditions."

Osteoporosis occurs most frequently in women around the menopause when oestrogen levels fall. That hormone protects the bone but also against acid reflux which will occur during pregnancy. As oestrogen levels fall, women start noticing heartburn and start taking PPIs at the same time as experiencing bone loss.

A paper, Is Estrogen a Curse or a Blessing in Disguise? found " Estrogen can be used to protect the mucosa from GERD induced injury and its complications like metaplasia and cancer."



PPIs and cancer protection.

There has been research that shows PPIs have a chemo-protective effect. One suggesting, "PPI use was associated with a 71% reduction in risk of OAC."

Another found, “PPI use to be associated with 41% lower risk of Barrett's oesophagus progression to OAC after adjusting for multiple confounders."

The AspECT trial (Aspirin + Esomeprazole Cancer protection Trial) found, "people who took this combination for at least seven years were 20% less likely to develop oesophageal cancer than if they had been untreated."

And another paper found, “The proton pump inhibitor pantoprazole disrupts protein degradation systems and sensitizes cancer cells to death under various stresses” looking at the possible mechanism for this.

PPIs have also been found to have a possible chemo-protective effect on some other cancers.

"Basic research studies ... suggested that **PPI may even have a protective effect against ColoRectalCancers**"

A Nested Case-Control Study of 23 Million Individuals" found "The use of PPIs was significantly associated with **reduced risk of breast cancer** and **ovarian cancer**" and "PPI exposure was associated with a significant **decrease in cervical and endometrial cancer risks**."

Potassium Competitive Acid Blockers (PCABs)

Examples: Vonoprazan, Tegoprazan.

These are the latest type of acid suppressant with many claims they could be better than PPIs as they don’t need an acid environment to activate them: “it can be taken without food because it is quickly absorbed. Vonoprazan accumulates in parietal cells under both acidic and neutral conditions. It does not require an acidic environment for activation.”

“In this rapidly evolving field, novel drugs such as potassium-competitive acid blockers (P-CABs) show promising potential.”

“The efficacy of vonoprazan was comparable with PPIs for the treatment of peptic ulcers following Endoscopic Mucosal Disection.”

There have been a number of studies comparing efficacy of PCABs with PPIs with the most common conclusion being they are “non-inferior” to PPIs.

From AGA Clinical Practice Update: “P-CABs Can Help When PPI Therapy Fails, "Clinicians generally shouldn’t use potassium-competitive acid blockers (P-CAB) as first-line therapy for acid-related conditions, nonerosive gastroesophageal reflux disease (GERD), or peptic ulcer disease,. ... However, P-CABs are recommended in place of proton pump inhibitors (PPIs) for most patients with *Helicobacter pylori* and other conditions where patients haven’t responded to PPIs."

Whether or not PCABs demonstrate a similar chemo-protective benefit to PPIs is yet to be determined.

Acid Reflux

Reflux of acid from the stomach is via an incompetent Lower Oesophageal Sphincter (LOS) into the oesophagus which it burns causing oesophagitis inflammation which is the source of heartburn symptoms.

Some common misconceptions regarding acid reflux.

1. It is *not* from excess acid “overflowing”. (Stomach acid is made and regulated as required by the action of hormones.)
2. It is *not* caused by low stomach acid. (A theory suggested by a chiropractor wanting to sell useless supplements. It is obviously incorrect as stomach acid refluxing is sufficient to cause oesophagitis damage.)
3. It is *not* caused by acidic foods - though they can cause heartburn symptoms by irritating oesophagitis.
4. It is *not* caused by common drugs found in foods, like caffeine or alcohol, or even nicotine.

For many people, the Lower Oesophageal Sphincter doesn't work as well as it should permitting stomach contents to flow back into the oesophagus.

Hiatus Hernia

One of the most common reasons for reflux is a **hiatus hernia**.

The hiatus is the hole in the diaphragm the oesophagus passes through just above its junction with the stomach. In many people, the top of the stomach can push up through this hole. Known as a hiatus hernia, we don't always know why this happens. Some people may have been born with a larger hiatus enabling herniation to occur at a young age. For others, the upward pressure caused by excess body fat may be the contributing factor.

For most people a hiatus hernia will cause no problems but for others, their sphincter muscles may not be strong enough to keep the end of the oesophagus tightly closed.