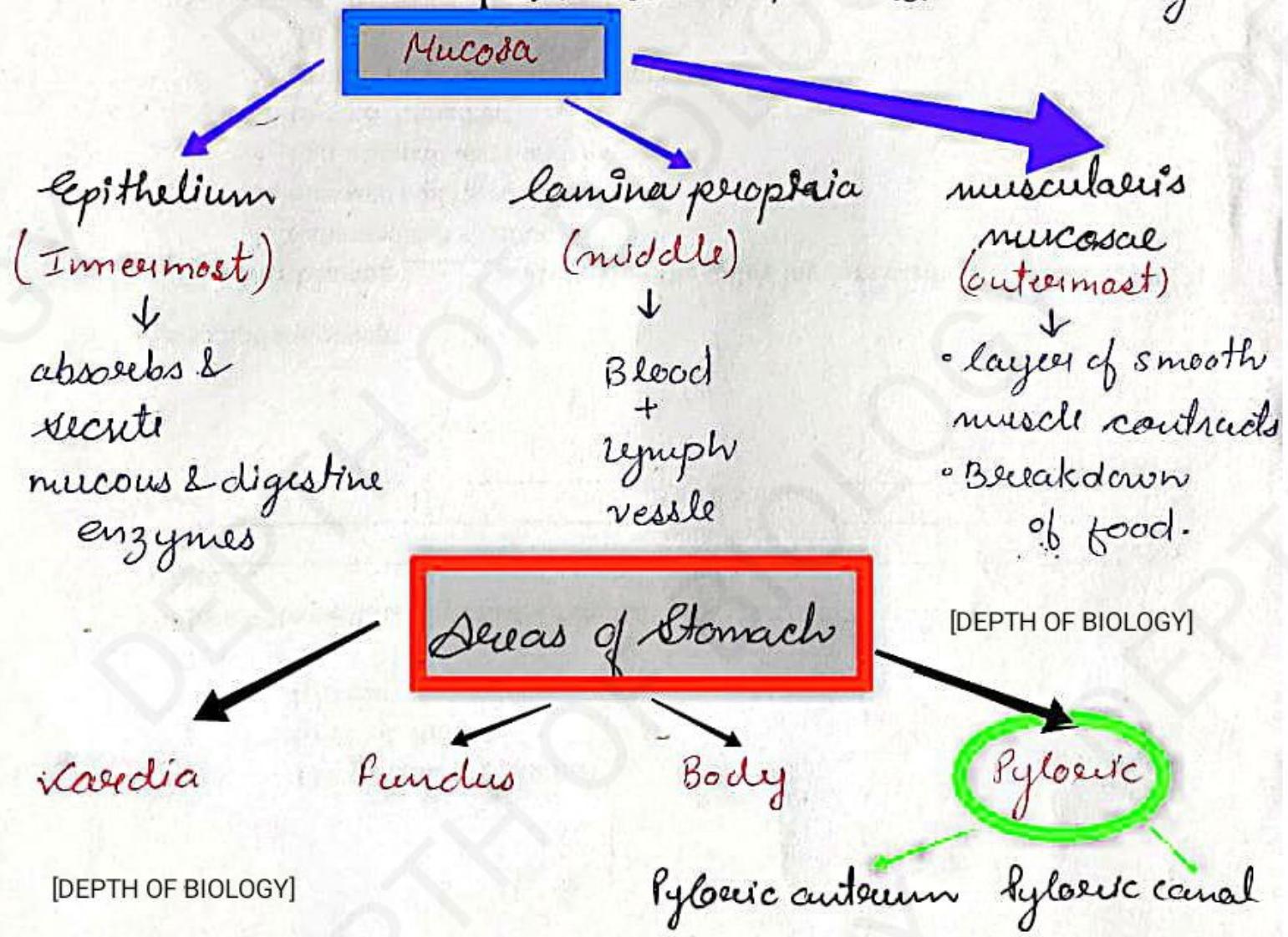


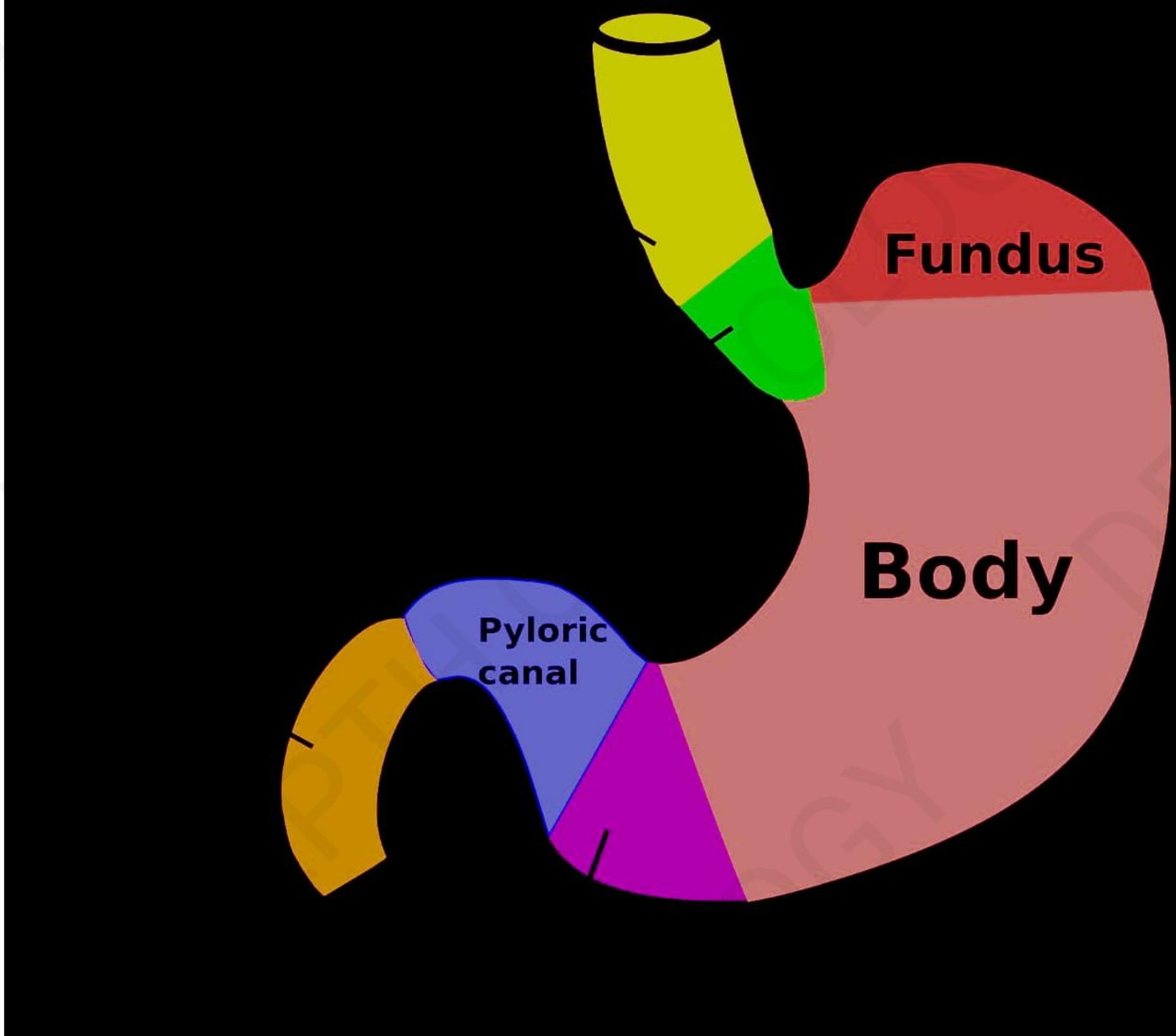
Peptic Ulcer Disease

Peptic → stomach ; Ulcer → sore or break in memb.
Having one or more sores in stomach → Gastric ulcer
If in duodenum → Duodenal ulcers

* The innermost wall of the entire GI tract is lined by



* a sore that develops on the lining of stomach.



* There is a pyloric sphincter at the end of stomach which gets closed while eating to keep food inside [DEPTH OF BIOLOGY] stomach.

1. Cauda — have mostly foveolar cells — secrete mucus (water and glycoprotein)

2. Fundus & Body (corpus) — have mostly parietal cells and chief cells

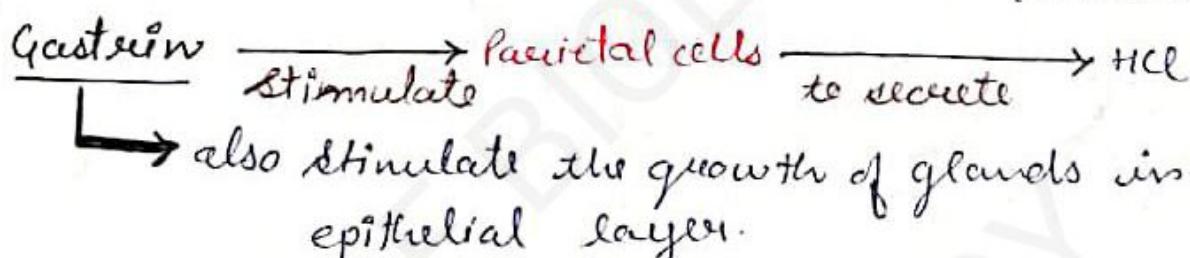
Parietal cells — secrete HCl

Chief cells — secrete pepsinogen → digest proteins

3. Antrum (Pyloric) — has mostly G-cells → secrete Gastrin also found in duodenum & pancreas

Now,

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→ Duodenum has Brunner's gland in submucosa

↓
secrete mucus rich in HCO_3^- into lumen

* Stomach walls are constantly exposed to acid so have thicker mucus layer as compared to duodenum where acid exposure is less and momentarily.

• Duodenum has single layer of mucus built around the MUC2 mucin — this is not attached to epithelium and more permeable to bacteria.

• Stomach has 2 layer mucus system — inner attached — outer unattached, loose

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both inner and outer are built by HUC5AC mucin produced by superficial epithelium.

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- * In addition, to the momentarily exposure of acidic duodenum — the blood flowing stomach and duodenum have more HCO_3^- → neutralize acid.
 Finally, small signalling molecules called Prostaglandins gets secreted in stomach and duodenum
 they stimulate mucus and bicarbonate secretion.

If vasodilate nearby blood vessel → Blood flow ↑↑
 - This promote the growth of new epithelial cells and inhibits the acid secretion.

Causes of Gastric ulcer and Duodenal ulcer

Main cause → infection by *H. Pylori bacteria* (gram-negative)
 especially in low income countries and settings
H. Pylori bacteria colonize in gastric mucosa
 → and release adhesions.

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↓
 helps them to adhere → Parietal cells.

→ and also release proteases cell
 → damage the mucosal cells

* It cause patchy pattern of damage.

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H. Pylori cause damage in Antrum (Pyloric region) and spread to most of stomach and eventually to duodenum.

→ Over time, the damage goes deeper and deeper in mucosa that eventually cause ULCER

Another cause,

(NSAID) Non-steroidal AntiInflammatory drugs



inhibit the enzyme cyclooxygenase

(which is involved in synthesis of Inflammatory
Prostaglandins)

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* Reduced level of prostaglandins over a prolonged period of time leave the gastric mucosa susceptible to damage and over time ulcers starts to develop.

Peptic ulcer results in →

- ① small, punched out hole in mucosa
- ② clean base → ∵ HCl secretion and constant churning acts as dishwasher actually keeping debris out of ulcers.
- ③ Typically beneath the base layer of scar tissue and blood vessel.
- ④ ulcers can bleed if erosion goes deep.

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Symptoms

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- ① epigastric pain - aching or burning in upper abdomen.
- ② Bloating
- ③ Belching
- ④ Vomiting (emesis)

* Gastric ulcer pain increases while eating - due to physical presence of food in stomach.
- as well as HCl production gets stimulated with eating.
Duodenal ulcer pain ↓↓ with eating.
Maybe that's why

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- Gastric ulcers are associated with weight loss.
- Duodenal ulcers are associated with weight gain

Diagnosis

- ① Upper endoscopy — tube is inserted in stomach and to proximal duodenum through mouth in order to visualise ulcers.
but during the procedure
- ② Biopsy — also done
 - To find signs of malignant cells.
 - To see and figure out H. Pylori infection.

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Treatment.

If H. pylori infection occurs —

- ① Combination of antibiotics —
metronidazole + tetracycline or metronidazole + Amoxicillin
- ② Acid lowering agents (Antacids)
- ③ Proton pump Inhibitors

Do not consume → Alcohol, Tobacco and caffeine
In extreme cases surgery can be done.