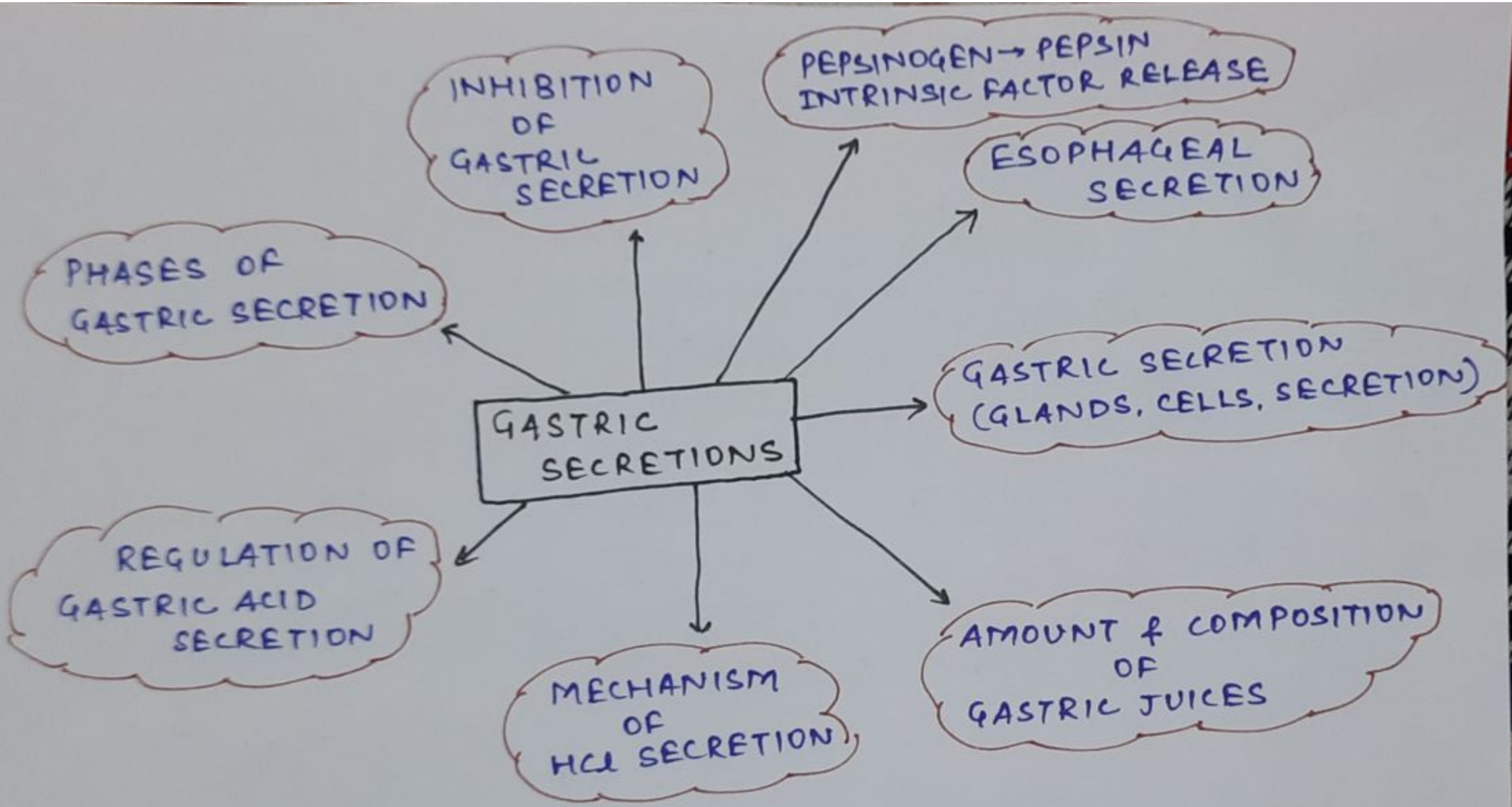
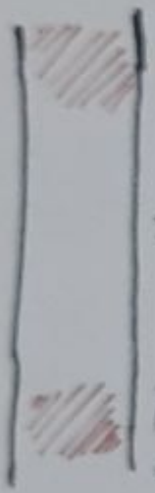

GASTRIC SECRETIONS
INHIBITION OF GASTRIC SECRETION
PEPSINOGEN → PEPSIN
INTRINSIC FACTOR RELEASE
ESOPHAGEAL SECRETION
PHASES OF GASTRIC SECRETION
GASTRIC SECRETION (GLANDS, CELLS, SECRETION)
REGULATION OF GASTRIC ACID SECRETION
MECHANISM OF HCl SECRETION
AMOUNT & COMPOSITION OF GASTRIC JUICES

ESOPHAGEAL SECRETION

⇒ ENTIRELY MUCOUS SECRETION



⇒ INITIAL PORTION OF ESOPHAGUS ⇒ COMPOUND MUCOUS GLANDS ⇑ PREVENTS EXCORIATION FROM NEWLY ENTERED FOOD.

⇒ MAIN BODY OF ESOPHAGUS ⇒ SIMPLE MUCOUS GLANDS

⇒ GASTRIC END OF ESOPHAGUS ⇒ COMPOUND MUCOUS GLANDS ⇓ PREVENTS DIGESTION OF ESOPHAGUS FROM GASTRIC ACID REFLUX.

GASTRIC SECRETION (GLANDS, CELLS, SECRETION)

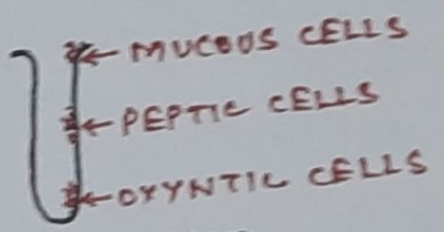
STOMACH MUCOSA HAVE TWO IMPORTANT TYPES OF TUBULAR GLANDS:-

1) OXYNTIC / GASTRIC GLANDS
2) PYLORIC GLANDS
3) SURFACE MUCOUS CELLS

OXYNTIC GLANDS

⇒ LOCATED ON INSIDE SURFACES OF BODY AND FUNDUS OF STOMACH (PROXIMAL 80% OF STOMACH)

⇒ 3 TYPES OF CELLS:-
- (i) MUCOUS NECK CELLS → MUCUS
- (ii) PEPTIC / CHIEF CELL → PEPSINOGEN
- (iii) OXYNTIC / PARIETAL CELL → HCl, INTRINSIC FACTOR

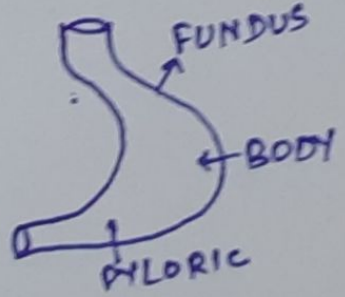


OXYNTIC GLAND

PYLORIC GLANDS

⇒ LOCATED IN ANTRAL PORTION OF STOMACH (DISTAL 20% OF STOMACH)

⇒ 3 TYPES OF CELLS:-
- (i) MUCOUS NECK CELLS → MUCUS
- (ii) PEPTIC / CHIEF CELL → PEPSINOGEN
- (iii) G CELL → GASTRIN



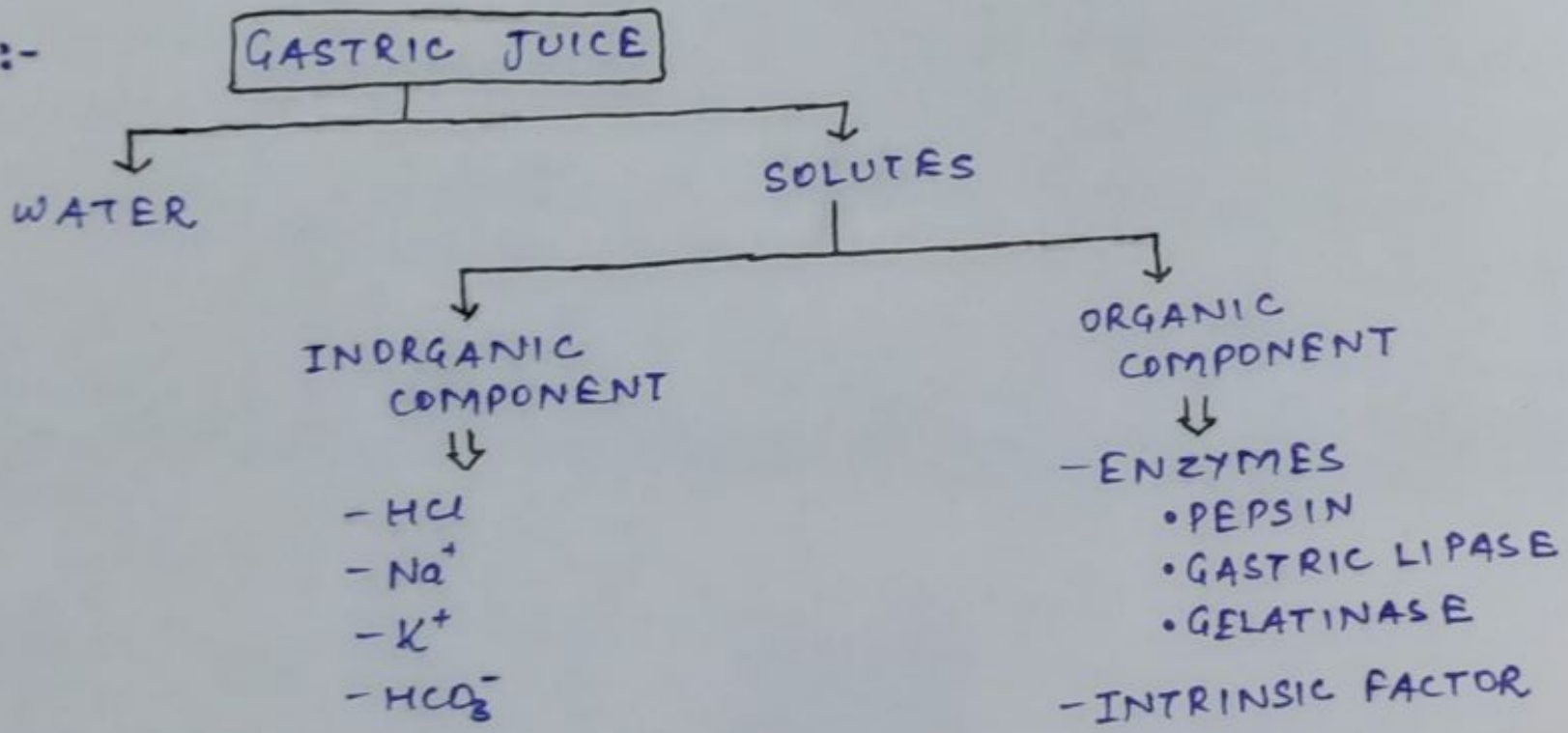
AMOUNT AND COMPOSITION OF GASTRIC JUICES

⇒ **AMOUNT** :- 1·5L TO 2L

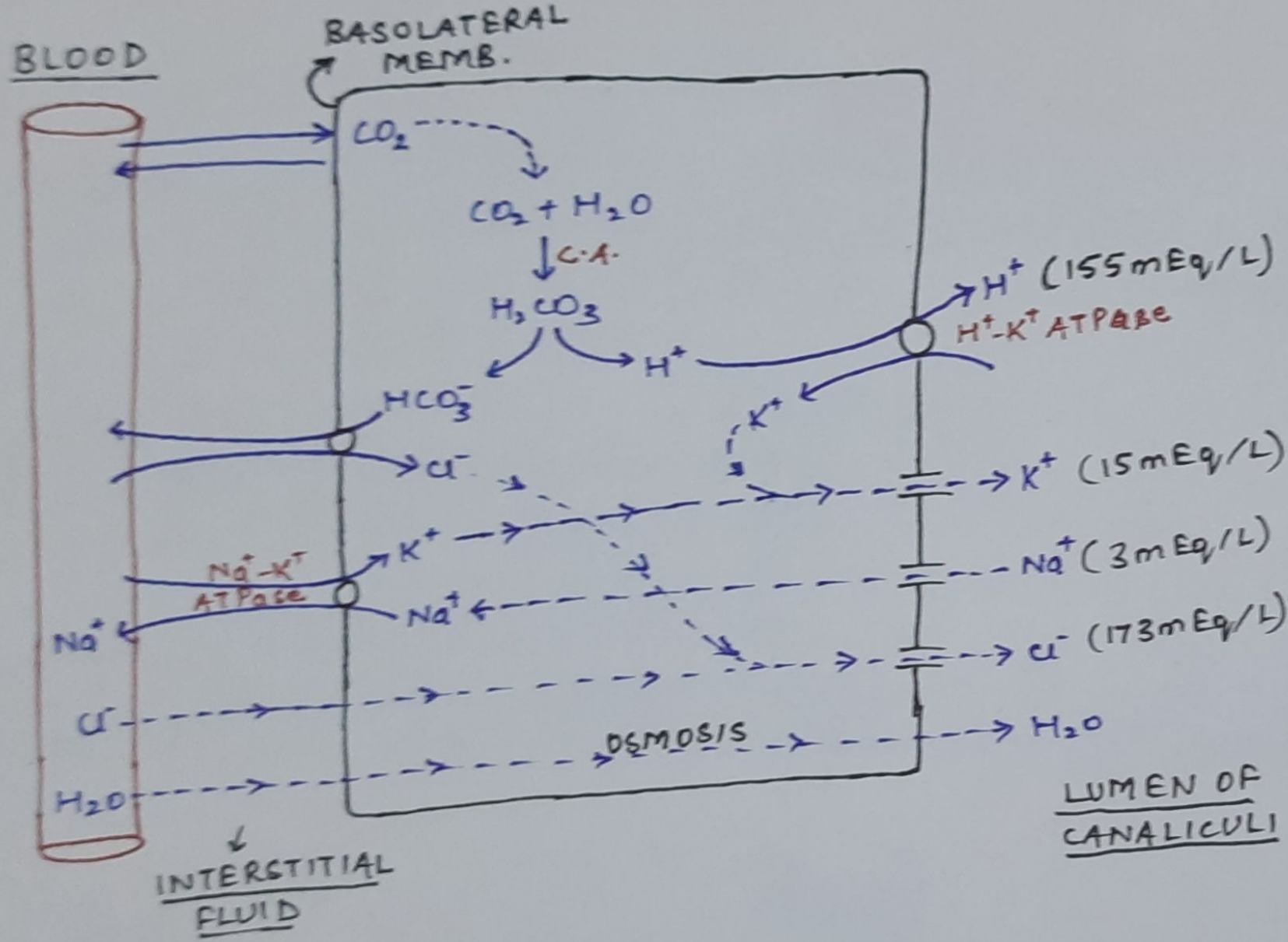
⇒ **pH** :- 1·0 TO 3·5

⇒ **RELEASED FROM** :-
- GASTRIC GLAND
- PYLORIC GLAND
- SURFACE MUCOUS CELLS

⇒ **COMPOSITION** :-



MECHANISM OF HCl SECRETION



NOTE :- AT SAME TIME, THE H^+ ARE SECRETED, HCO_3^- IONS DIFFUSE INTO BLOOD SO THAT GASTRIC VENOUS BLOOD HAS A HIGHER P^H THAN ARTERIAL BLOOD WHEN STOMACH IS SECRETING ACID.

FINAL SECRETION IN CANALICULUS :-

- WATER
- HCl (150-160 mEq/L)
- KCl (15mEq/L)
- NaCl (SMALL AMOUNT)

GASTRIC BARRIER :-

⇒ ALKALINE MUCUS + TIGHT JUNCTION BETWEEN EPITHELIAL CELLS

⇒ PREVENTS BACKLEAK OF ACIDS.

⇒ DAMAGE OF GASTRIC BARRIER (D/T EXCESSIVE USE OF ALCOHOL/ASPIRIN)

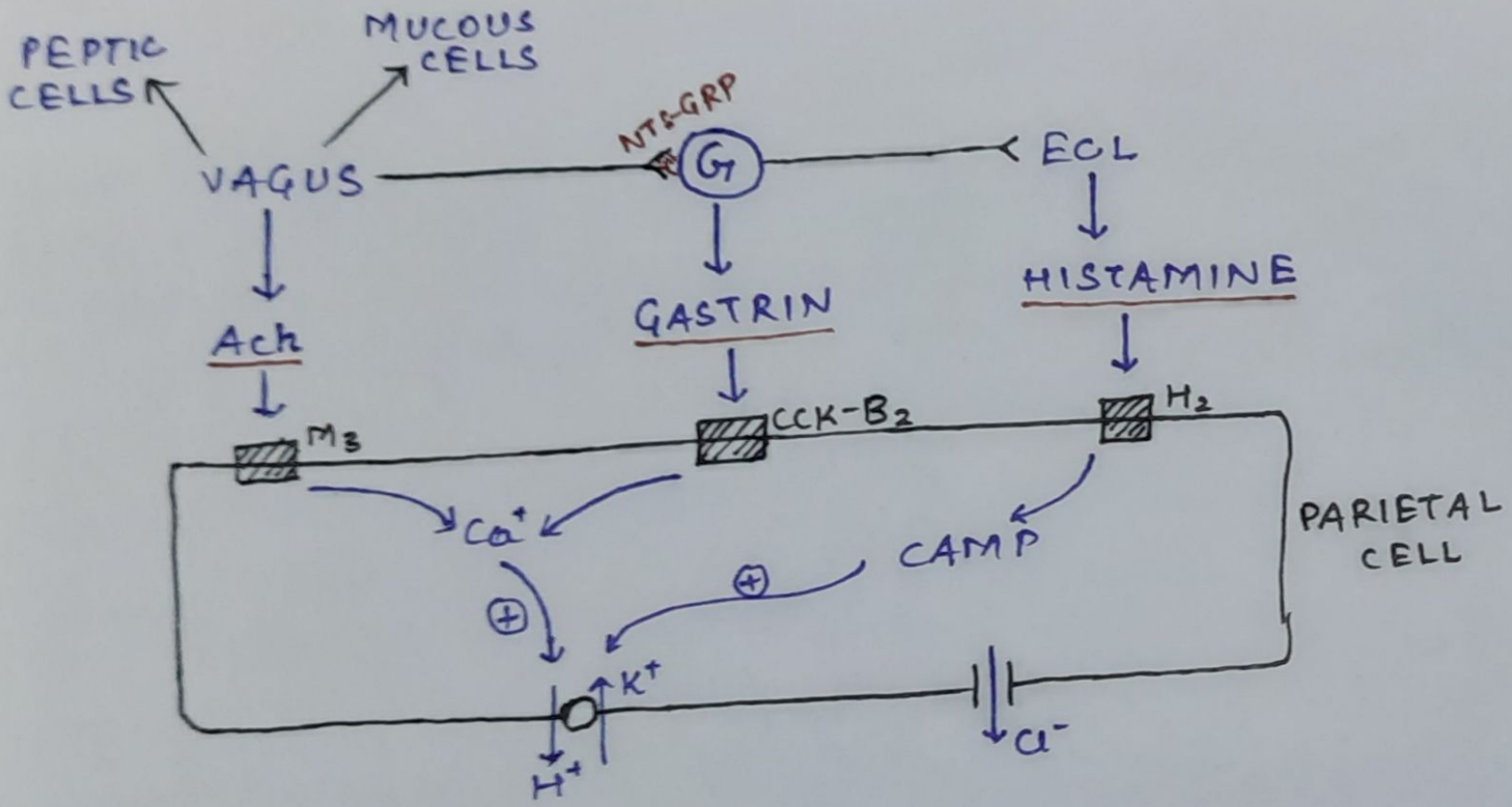
⇓

LEAKAGE OF ACID INTO MUCOSA

⇓

STOMACH MUCOSAL DAMAGE

REGULATION OF GASTRIC ACID SECRETION



* ATROPINE :- M_3 BLOCKER

⇩

BLOCKS ACID SECRETION STIMULATED BY Ach AND NOT BY OTHERS.

GASTRIN-HISTAMINE REFLEX

FOOD CONTAINING PROTEIN, REACHES ANTRUM

⇓

SOME PROTEINS STIMULATES G-CELLS IN PYLORIC GLANDS

⇓

RELEASE OF GASTRIN INTO BLOOD

ECL CELL

⇓

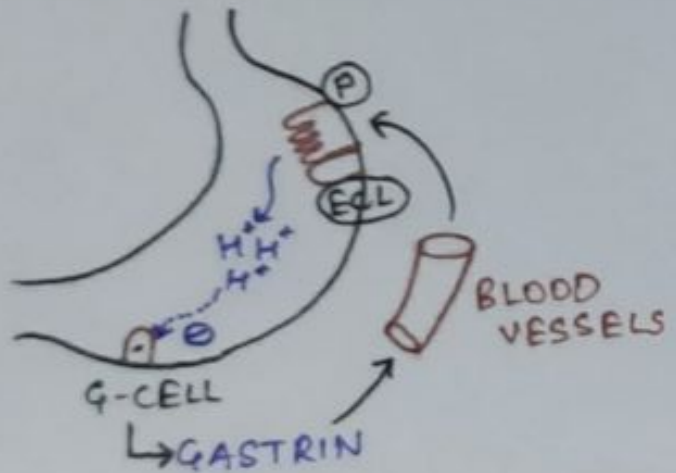
RELEASE OF HISTAMINE INTO BLOOD

⇓

STIMULATES PARIETAL CELLS

⇓

ACID SECRETION ↑



PHASES OF GASTRIC SECRETION

CEPHALIC PHASE:-

⇒ BEFORE FOOD ENTERS STOMACH, WHILE IT IS BEING EATEN
(STIMULUS:- SIGHT, SMELL, TASTE, THOUGHT)

⇒ 30% OF GASTRIC SECRETION

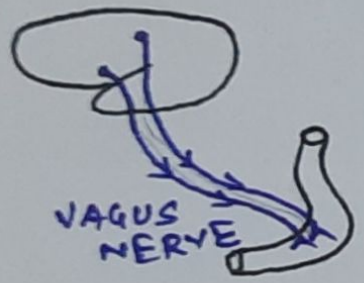
⇒ NEUROGENIC SIGNALS ORIGINATE IN
- CEREBRAL CORTEX
- APETITE CENTRES OF AMYGDALA & HYPOTHALAMUS

⇓

THROUGH VAGUS NERVE

⇓

TO STOMACH



GASTRIC PHASE:-

⇒ DURING FOOD REMAINS IN STOMACH

⇒ 60% OF GASTRIC SECRETION

⇒ REGULATION

(i) LONG VAGOVAGAL REFLEXES
(STOMACH → BRAIN → STOMACH)

(ii) LOCAL ENTERIC REFLEXES

(iii) GASTRIN - HISTAMINE MECHANISM

INTESTINAL PHASE

⇒ FOOD ENTERING IN UPPER PORTION OF SMALL INTESTINE (PARTICULARLY DUODENUM)

⇒ 10% OF GASTRIC SECRETION

⇒ REGULATION

1) NERVOUS MECHANISM

2) HORMONAL MECHANISM (PARTLY BECAUSE OF SMALL AMOUNT OF GASTRIN RELEASED BY INTESTINAL MUCOSA).

INHIBITION OF GASTRIC SECRETION

(I) ENTEROGASTRIC REFLEX (WILL BE DISCUSSED)

(II) INHIBITORY EFFECT OF EXCESS H^+ ON G-CELL

(III) INTESTINAL HORMONES:-

ACID, FAT, PROTEIN BREAKDOWN PRODUCTS, HYPEROSMOTIC / HYPOOSMOTIC FLUIDS / IRRITANTS IN UPPER S. INTESTINE

⇓

SECRETIN, GIP, VIP, SOMATOSTATIN

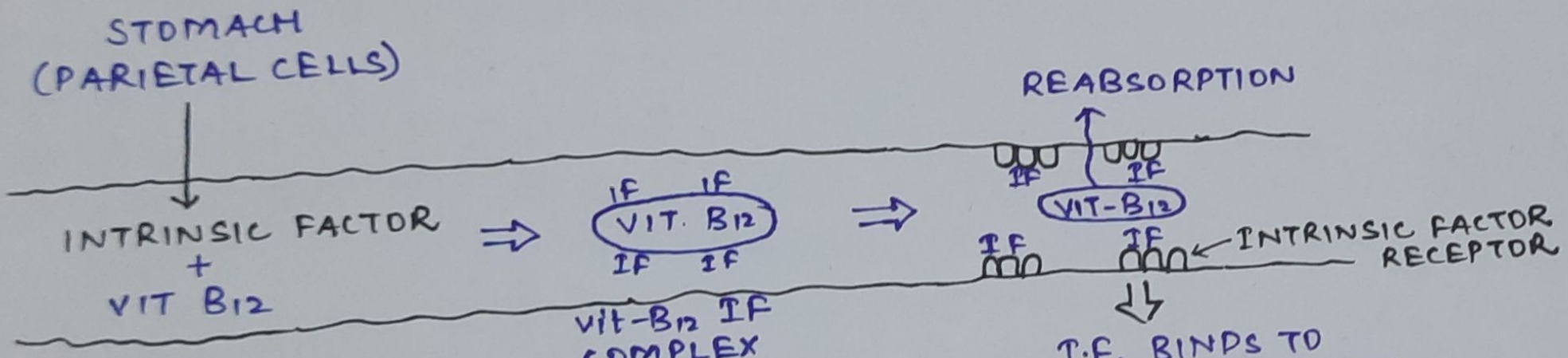
⇓

INHIBITS GASTRIC SECRETION

CONVERSION OF PEPSINOGEN → PEPSIN

PEPSINOGEN (INACTIVE) (NO DIGESTIVE ACTIVITY) —HCl→ PEPSIN (ACTIVE IN ACIDIC MEDIUM) (1·8–3·5) (PROTEIN DIGESTION)

INTRINSIC FACTOR RELEASE



vit-B_{12} IF COMPLEX

⇓

VIT-B_{12} IS PREVENTED FROM BEING DIGESTED OR DESTROYED INTO SMALL INTESTINE

I.F. BINDS TO ITS RECEPTOR ON ILEAL EPITHELIUM SURFACE AND VIT·B_{12} IS ABSORBED