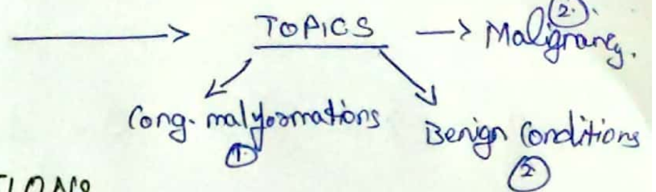
PANCREAS



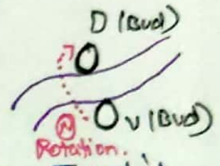
BENIGN CONDITIONS

I CONG. ABNORMALITY / MALFORMATION :-

* There are 2 embryonic malformation c can occur.

1.) ANNULAR PANCREAS :-

* Two Pancreatic Buds are (f) by the duodenum * DORSAL BUD, * VENTRAL BUD.



↓

* Ventral Bud rotates around Duodenum by 5th wk. of I.U Life.

↓

* If this ROTATION DOES NOT occur then, it forms an ANNULAR PANCREAS. (usually around the 2nd Part of Duodenum).

* C/F :- Pt. / child (usually) Presents c,

1.) Bilious Vomiting (Starts since Birth)

(Acc. to latest Study in 22 children c Annular Pancreas, 9 had Bilious vomiting & 13 had non bilious vomiting).

(I/o Cong. hypertrophic Pyloric Stenosis -> Non-Bilious vomiting will occur 3-4 wks after. Birth)

2.) X-ray Abd. / CxR. -> DOUBLE BUBBLE Sign (also seen in Duodenal Atresia)

* IOC = CECT
* MX => Duodeno - Duodenostomy (same for Duodenal atresia).

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NORMALLY:-

2:) PANCREAS DIVISUM :- * when the Dorsal + Ventral Buds fuse their ducts also fuse. & There is Proper drainage of Pancreas.

* DORSAL BUD	* VENTRAL BUD
* DUCT = DUCT OF SANTORINI	* DUCT = DUCT OF WIRSUNG.
* IN UTERO => Maj. OF Drainage is through this Bud.	
* IN ADULTS =>	Maj. of Drainage is by the Ventral Bud / Duct of WIRSUNG.

∴ Problem is -> If fusion does not occur then -> No Proper drainage -> these Pt. are @ a Higher Risk of "PANCREATITIS."
(who is an ADULT(me))

(BCZ in this Pt. (ADULTS) only Maj. Drainage is still through DUCT of Santorini).

ACUTE PANCREATITIS.

* Latest theory of Acute Pancreatitis development =>

> There is INTRA-ACINAR Activation of Trypsinogen

↓ Causes

> Inflammatory Response

↓ bcz of this

> ↑↑(IL-1, IL-6) are Raised. (also Seen in "SIRS")
(SIRS was Coined in Acute Pancreatitis only)

↓ bcz of this

Signs & Symptoms of ACUTE PANCREATITIS.

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③ * CAUSES OF ACUTE PANCREATITIS :-

1.) (mc) Cause => Gall Stones. (Not Alcohol)

2.) 2nd (mc) " => ALCOHOL.

3.) Mechanical Causes :- • Gall stones, • Pancreatic Divisum,

• ERCP induced Pancreatitis (A/k/as) IATROGENIC Pancreatitis.
  → A side view duodenoscope is used to cannulate the Ampolla.
  → PANCREATITIS - is the (mc) Complication of ERCP.

4.) Metabolic Causes :- • Alcohol, • Hypercalcemia, • ↑ed Triglycerides,

• Drugs => * Anti retro viral.
  * valproate.
  * Furosemide.

5.) Infectious Causes :- • Mumps, • CMV (viral), •

6.) BLUNT Trauma - is the (mc) Cause of Acute Pancreatitis in CHILDREN.

7.) Scorpion Bite.

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* C/F of Acute Pancreatitis :- Pt. presents c̄,

1.) Epigastric pain c̄ Radiates to the Back (mc).

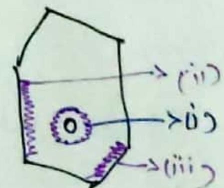
2.) Nausea & Vomiting (±)

3.) 3 Ⓧ Signs of Acute Haemorrhagic Pancreatitis,

i) CULLEN's Sign = Discoloration around the umblicus.

ii) GREY TURNER Sign = Discoloration in FLANKS.

iii) Fox Sign = Discoloration in Inguinal Region.



(4)

So now, this pt. c̄ Pancreatitis comes to our Emergency. How to Δse it's Pancreatitis?

* Δsis => I Investigation = (1) 1st inves. => S. Amylase

* If S. Amylase 4 × (N) value => Suggestive of Acute Pancreatitis

* But S. " can be Raised In,
  - Perforations
  - Volvulus
  - Mesenteric ischemia
  - Torsion of ovary.

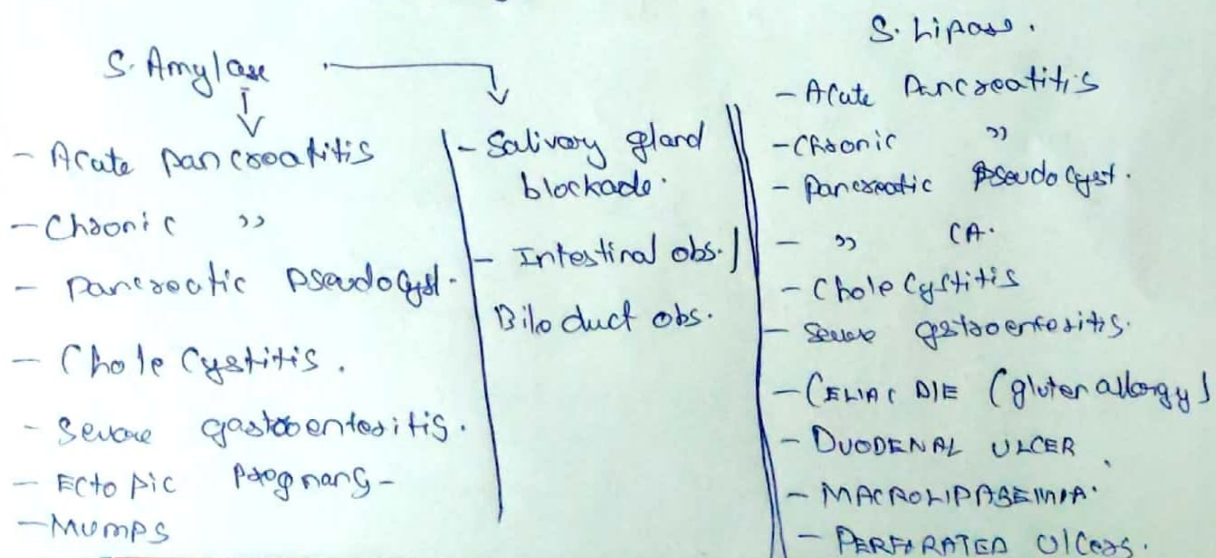
(N) S. Amylase = 25–125 U/L.
(N) S. Lipase = 0–160 U/L.

∴ S. Amylase is SENSITIVE but NOT SPECIFIC Δsis of Acute Pancreatitis.

(2) S. LIPASE => More Specific for " " .

(*) The Values of S. Amylase & S. Lipase DOES NOT Correlate c̄ the SEVEARITY OF ATTACK of Pancreatitis.

∴ (These 2 values are not included in any " Scoring Systems)



Causes of High Level of

S. Amylase
- Acute pancreatitis
- Chronic "
- Pancreatic Pseudocyst.
- Chole Cystitis.
- Severe gastroenteritis.
- Ectopic Pregnancy
- Mumps
- Salivary gland blockade.
- Intestinal obs.
- Bile duct obs.

S. Lipase.
- Acute Pancreatitis
- Chronic "
- Pancreatic Pseudo cyst.
- " CA.
- Chole Cystitis
- Severe gastroenteritis.
- CELIAC DIS (gluten allergy)
- DUODENAL ULCER.
- MACROLIPASEMIA.
- PERFORATED Ulcers.

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(5.)

II CECT → can be done after 3-4 days of the attack
- Tells us about the SEVERITY of the Attack.
- used in Scoring the D/E.

* BEST SCORING System for ACUTE PANCREATITIS :-

Q

BALTHAZAR Score / CT Severity INDEX.

> Takes into account 2 things,

(1) Appearance on CT

GRADE		SCORE
A	- (N) appearance	= 0
B	- Focal / Diffuse enlargement	= 1
C	- peri pancreatic inflammation	= 2
D	- single fluid collection around pancreas	= 3
E	- Multiple fluid collection	= 4.

(2) Amount of Necrosis (F).

	SCORE
> No Necrosis	= 0
> <30% "	= 2
> 30-50% "	= 4
> >50% "	= 6.

* when we club Both we get a score upto 10.

(*) * " " of ≥6 = Q ACUTE SEVERE PANCREATITIS

* Now we have Asod & grade the condition Next,.

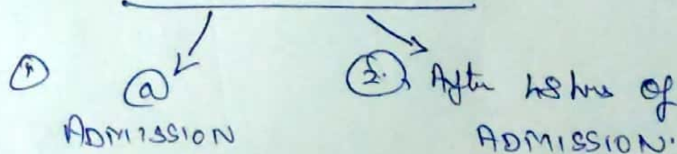
SEVERITY SCORES

Q

(1) *(mc) RANSON's Criteria for Acute Pancreatitis.

(There are separate " " Alcohol & gall stone induced Pancreatitis).

- Criteria's measured



P.T.O.

(1) @ ADMISSION :-
- Age > 55 yrs.
- WBC > 16,000.
- S. LDH > 350 IU/L
- S. AST > 250 IU/L
- B. Sugar > 200 mg/dl.

(2) After 48 hrs :-
- Fall in Hematocrit by > 10%.
- Base deficit > 4 mEq/L. (ACIDOSIS)
- ↑ in Blood urea N_2 by 5 mg/dl. (BUN)
- Hypo calcemia = < 8 mg/dl.
- Fluid Sequestration = > 6L.
- PO_2 < 60 mm Hg.

(*) (S. Amylase & S. Lipase values not included in any scoring systems here)

∴ If Ranson's criteria ≥ 3 = Acute Severe Pancreatitis.

Other Severity scoring Systems :-

(2) Glascow Scoring System = ≥ 3 ⇒ Acute Severe Pancreatitis.

(3) APACHE Score = (Acute Physiology & Chronic Health evaluation Score)
- → Apache II Criteria = used for Acute Pancreatitis
- → ≥ 8 = Acute Severe Pancreatitis.

* APACHE "O" = Apache + obesity (is an independent Poor Prognostic factor for Acute Pancreatitis).

(4) BISAP Score :- Bedside index For Severity of Acute " .

4) B - BUN > 25mg/dL.

I - Impaired mental status.

S - SIRS (if > 2 criterias are there)

A - Age > 60yrs.

P - pleural effusion ⊕.

* BISAP Score of > 3 = Acute Severe Pancreatitis.

Other indicators of Acute Severe Pancreatitis:-

5.) CRP > 150 IU/L

6.) Procalcitonin can also be measured.

Atlanta Classification of Acute Pancreatitis.

1.) Acute Pancreatic fluid Collection (APFC)

when,
> Pancreatic juice collected around Pancreas, (This occurs in 4 wks of the attack)

2.) Acute Pancreatic Necrotic Collection (APNC)

when,
> Infected collection around Pancreas (in 4 wks of attack).

3.) Pseudo cyst => collection & is walled by Granulation tissue
> occurs after 4 wks. of attack.

Complications Of Pancreatitis.

- Local
- Systemic

P.T.O.

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* Local Comp. :- • APFC, • APNC, • Pseudocyst, • Pseudoaneurysm

formation = occurs bcz of Elastase (E) + Splenic vessels are the ones

c̄ are commonly involved, • Portal / Splenic vein thrombosis → Can

give rise to Lt. sided Portal HTN. , • Lt. sided pleural effusion.

(Mx of Lt. sided » » = Splenectomy).

* Systemic Comp. :- • Hypovolemic shock (mc), • Hyperglycemia,

• Hypocalcemia, • ARDS, • ATN (Acute Tubular Necrosis, • Chronic Heart failure.

---

* Pseudocyst of Pancreas. = Is the Collection c̄ occurs after 4 wks & lined by granulation tissue.

(Not by true epithelium ∴ pseudocyst).

>(mc) situated in — Lesser Sac. behind the stomach.

> this presses the stomach against the Ant. gastric wall.

* C/F – • Epigastric Swelling, • Nausea & vomiting.

• Pseudo cyst are (mc) c̄ Chronic Pancreatitis >> Acute Pancreatitis.

* IOC = CECT (confirmatory).

De'Egedio's class. for Pseudocyst of Pancreas.

Type I = • found in Acute Pancreatitis,

• Have (N) Anatomy of duct.

• No fistula btwn. the duct & the Cyst.

Type II = • found in Acute on chronic Pancreatitis,

• Abnormal Duct anatomy Can be (+), but
  c̄out Strictures.

» III = • found in chronic Pancreatitis c̄ abnormal duct anatomy
  c̄ Strictures

P.T.O.

- have fistulous communication.
- type of a Retention Cyst.

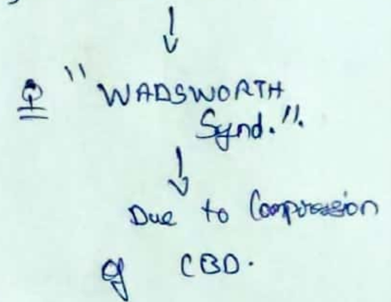
* Fate of Pseudocyst of Pancreas :- 1.) Can show spontaneous Resolution → if it Drains into the gut.

2.) if it Ruptures into the peritoneal cavity → then there can be peritonitis.

3.) it can Rupture into a vessel → giving rise to Bleeding.

4.) Can become infected → Infection is the (mc) comp. of a Pseudo pancreatic Cyst.

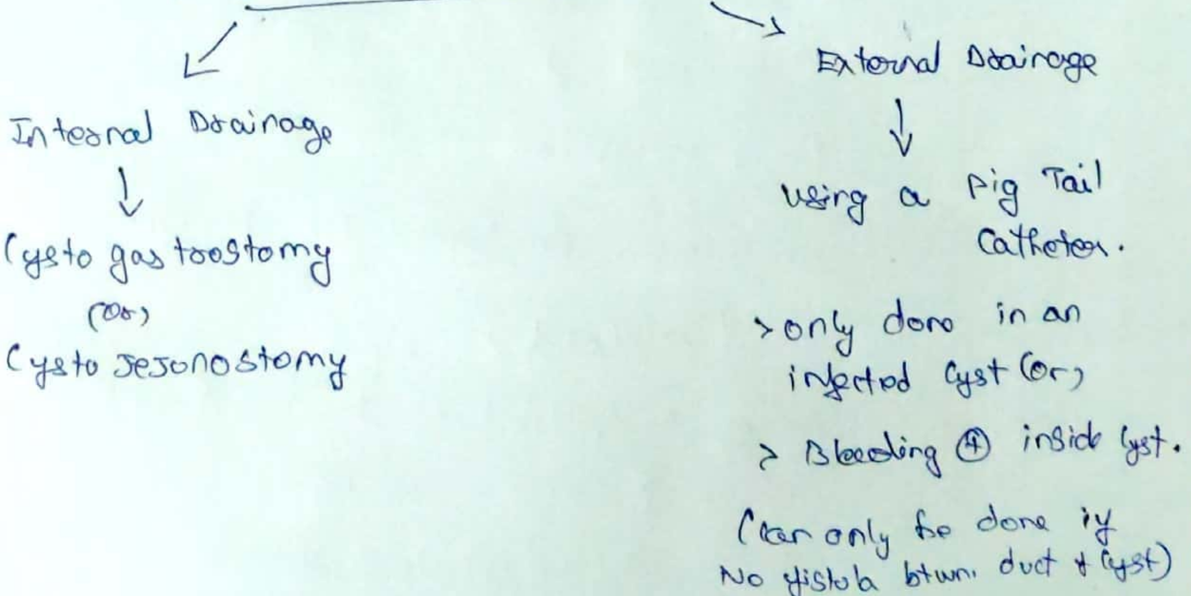
5.) there can be mechanical obs. BCZ of the Cyst & this can give Rise to → i) Gastric outlet obs., ii) obs. Jaundice.



Mx of Pseudo Pancreatic Cyst :-

1.) Majority of the Cyst Resolves Spontaneously

2.) Those c̄ are > 6cm in size & > 6 wks old & > 6mm wall thickness



*(mc) Comp. of Pseudocyst Sx → Haemorrhage.

GRADING OF ACUTE PANCREATITIS

Based on,

	Local Comp.		Systemic Comp.
MILD	No		No
Moderate	Sterile Collection	(or)	Transient organ failure.
Severe	Infected Collection	(or)	Persistant " " .
Critical	" "	+	" " " .

TPN = Total Parenteral Nutrition

* Mx of Acute Pancreatitis :-

1.) Mainly Supportive in Nature.

- aggressive fluid Resusitation
- Nutrition (initially TPN given But Early initiation of Enteral Nutrition Reduces the mortality in Acute Severe Pancreatis).
- No Role of prophylactic Antibiotics (only to be used if Infec. is ⊕)
  (I.V Meropenam is the DOC).
- Somatostatin Analogues can be given (octreotide)
- Mx of Complications has to done.
  i) I/c Pseudocyst - see before
  ii) I/c Infected Necrosis - Necrosectomy. (But has high mortality rate).

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2) If we have gall stone induced Pancreatitis :-

> If Jaundice ⊕ —> ERCP to remove the stone.

> should undergo Cholecystectomy before Hospital discharge.

CHRONIC PANCREATITIS.

* Patho physiology = usually starts ̄c a Central acute Pancreatitis event

↓ b/c of this

It stimulates the Stellate cells + Myofibroblasts

↓

As there are stimulated even when the Acute episode is over, Inflammation persists in these stellate cells & myofibroblasts

↓ this stimulates

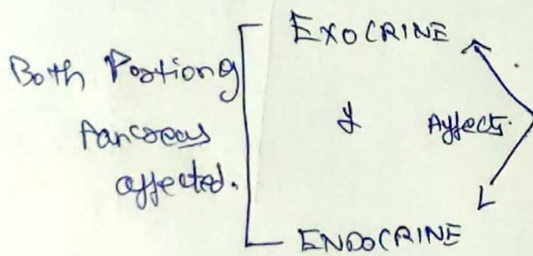
TGF B

↓ Causes

Fibrosis

↓

Loss of func.



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* TIGAR-O Class => Tells about Risk of CHRONIC PANCREATITIS.

Causes

T - TOXINS = • Alcohol (mc cause of 1st)

• Hypercalcemia (> 12.5 value seen in Hyperparathyroidism).

•

I - Idiopathic

G - Genetic factors

P.T.O.

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* PRSS1 & PRSS2 genes have been implicated in,

"HEREDITARY PANCREATITIS."

* SPINK 1 gene — Tropical " . (asso. c̄ Cassava diet).

A - Autoimmune.

R - Recurrent Pancreatitis.

O - Obstructive causes (Gall stones, strictures)

---

3 MAIN PROBLEMS IN Chronic Pancreatitis

EXOCRINE DYSFUNC.

means Pancreatic Enzymes are Reduced.

↓

- Malabsorption.
- Steatorrhoea. (↑ed fat content in (also foul smelling feces)
- Wt. loss.

ENDOCRINE Dysfunc.

Due to ↓ed insulin

↓ Pt. can have

- BRITTLE Diabetes (ie) Diabetes cannot get controlled.

* PAIN

→ occurs bcz of ineffective drainage (or) strictures/pseudocyst formation.

↓

Pancreatic Duct bcms, Dilated

↓ &

There can be stones inside duct.

↓ bcz of this

If we doan ERCP we get a "CHAIN OF LAKES APPEARANCE"

↓

Bcz of this stones there is ineffective drainage of secretion

↓

Pain.

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B.) Ans :- 1.) Best investigation = EUS (Endoscopic USG)

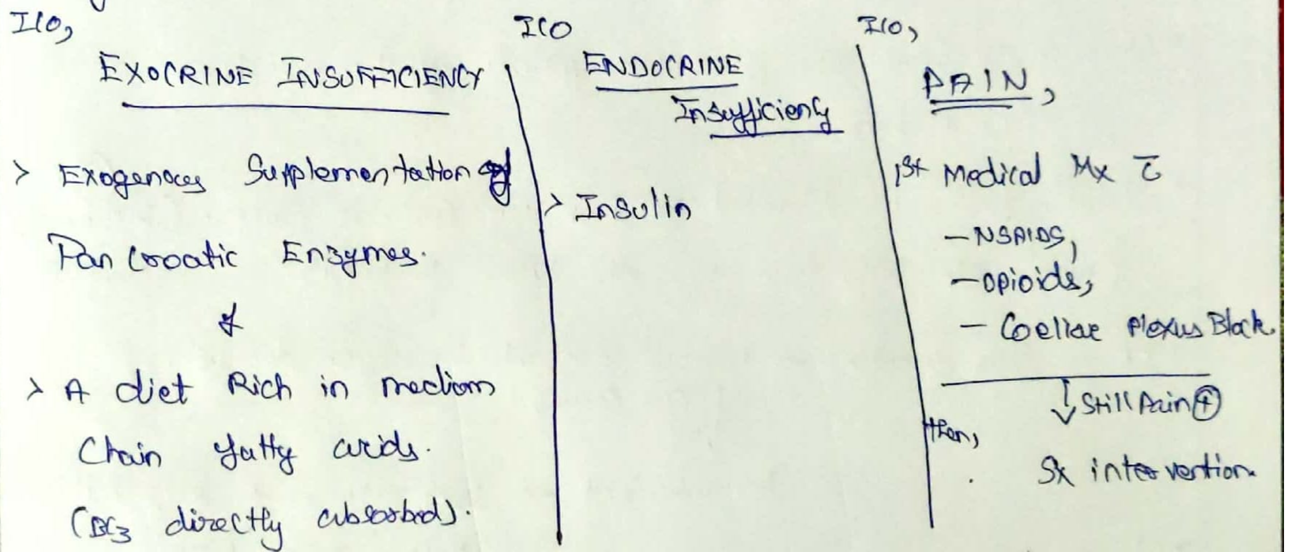
2.) IOC = MRCP c̄ Secretin Stimulation.

3.) ERCP = Chain of Lakes appearance.

4.) Biochemical Tests = 1.) Faecal Fat assay
2.) " Elastase Levels
3.) Pancreo Lauryl Test

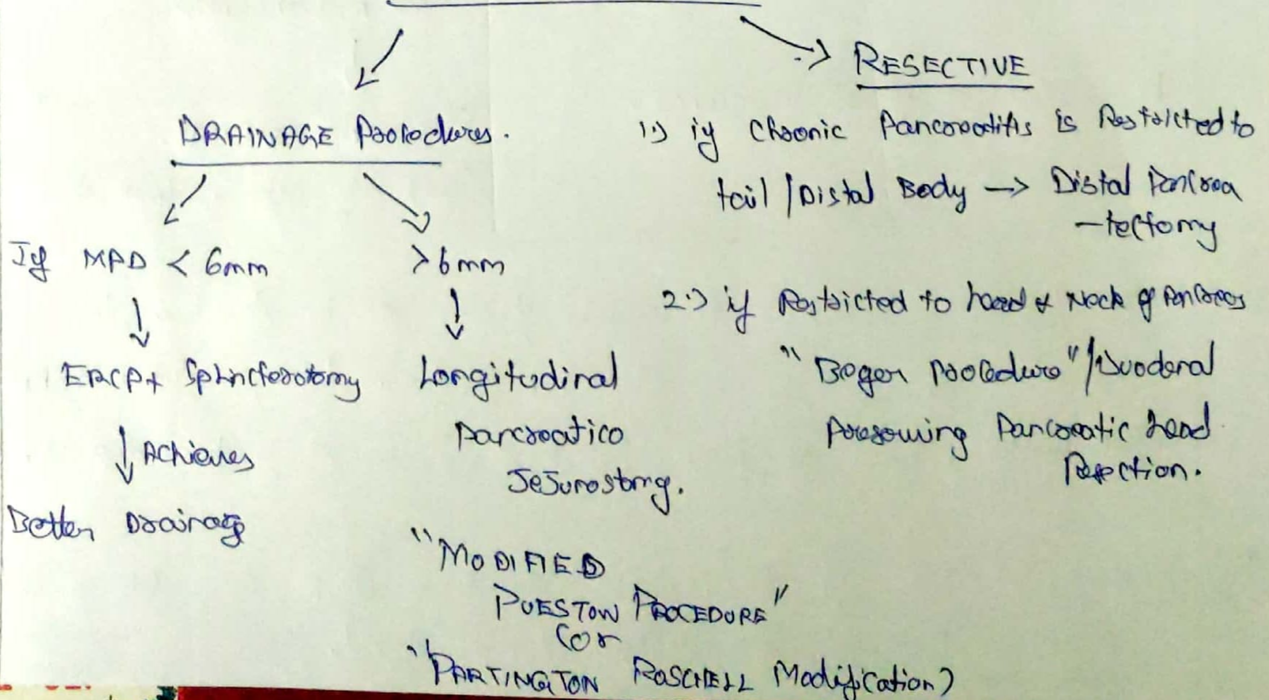
These will Tell us about malabsorption.

* Mx of Chronic Pancreatitis :-



MPD = Main Pancreatic duct diameter.

Sx INTERVENTIONS



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* If we do PESTOW's + BEGER => FREY's Procedure.
↓
we will expose the entire pancreatic duct & then anastomose it c̄ Jejunum.

OTHER Cong. Abnormalities in Pancreas:-

1.) Cystic fibrosis (CF) :- * Inherited Autosomal Recessive condition, (mc) among Caucasians.

* mutation in CFTR gene (Cystic fibrosis transmem conductance regulator) on Chromosome 7.

* CFTR gene -> creates a cell mem. protein -> c̄ controls Cl^- movement across C.m.

* CF -> multisystem D/o of exocrine glands affecting -> Lungs, intestines, Pancreas & Liver

* Charac. feature -> Elevated Na^+ & Cl^- ion conc. in SWEAT. (>90 mmol/L) (Dx) Confirmatory
(mother c/o salty taste when she kisses the Baby).

* CF -> thick secretions -> Passage Blockade -> organ damage.
↓ (mc cause)
of
Pancreatic Duct -> Duct ectasia & fatty Replacement of exocrine Acinar tissue.
↓ Pancreatic exocrine insufficiency
Fat malabsorption.
↓
Steatorrhea @ Birth.
(bulk, oily, offensive smell stools).

* C/F -> Poor growth, Poor apetite,
Rancid greasy stools, abd. distention,
Clubbing, Chronic Resp. D/E.

Rx -> Control of 2° consequences.

2.) Ectopic Pancreas :- ectopic Pancreatic tissue can be found in the submucosa of parts of -> stomach, Duodenum / S.I (including Meckel's), gall Bladder, hilum of Spleen, c̄ in Liver, alimentary tract duplication cyst.

3.) Cong. Cystic D/E of Pancreas:- Sometimes ⊕ c̄ Cong. D/E of Kidneys, Liver & Part » Von-Hippel-Lindau Synd.

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