

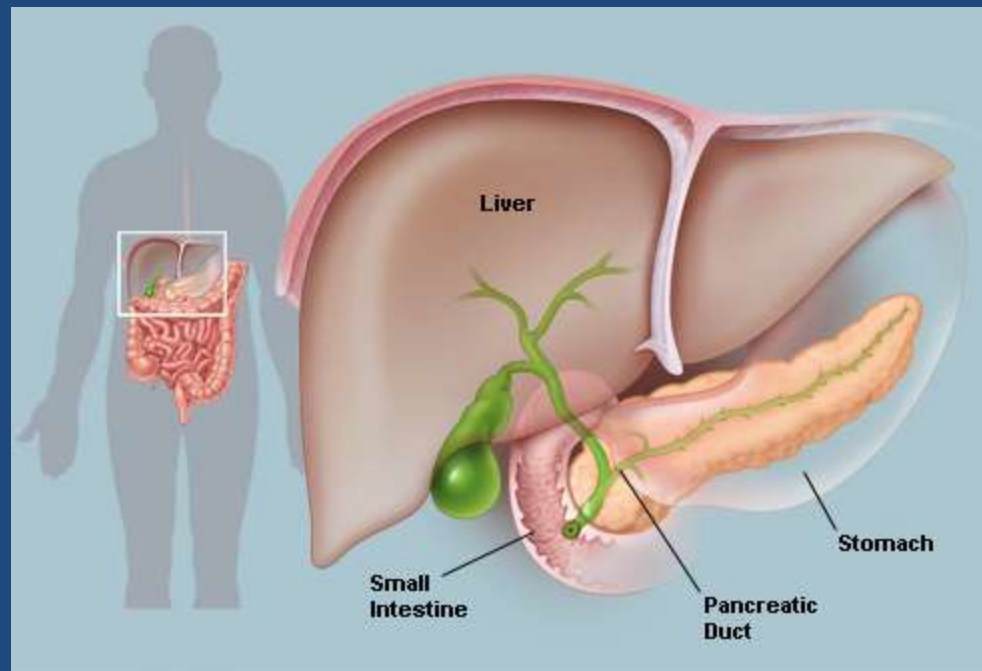
Nutrition in pancreatitis and pancreatic resection

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Objectives

- Overview
- Types of pancreatitis and surgeries
- Nutrition therapy for acute and necrotizing pancreas
- Optimizing nutrition after pancreaticoduodenectomy and total pancreatectomy

ANATOMY OF THE PANCREAS



FUNCTION OF THE PANCREAS

Exocrine cells

- Produce enzymes to help with the digestion of food.
- Pancreatic enzymes are released in the duodenum.

Endocrine cells

- Release hormones “insulin and glucagon” into the bloodstream.
- Controls blood sugar (glucose) levels.

Physiology

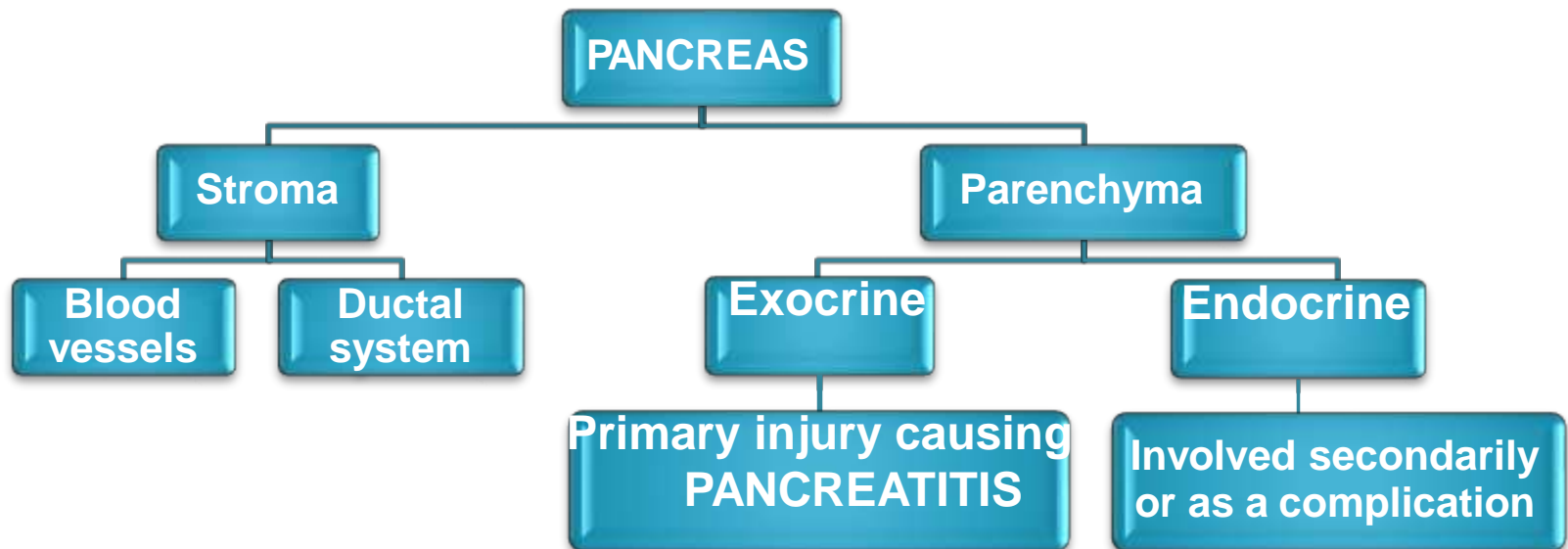
- Endocrine (20%)
 - Islets of Langerhans
 - Insulin (beta)
 - Glucagon (alpha)
 - Somatostatin (delta)
- Exocrine (80%)
 - Digestive enzymes (pro-enzymes)
 - Trypsinogen
 - Chymotrypsinogen
 - Controlled by
 - Gastrin
 - CCK
 - Secretin



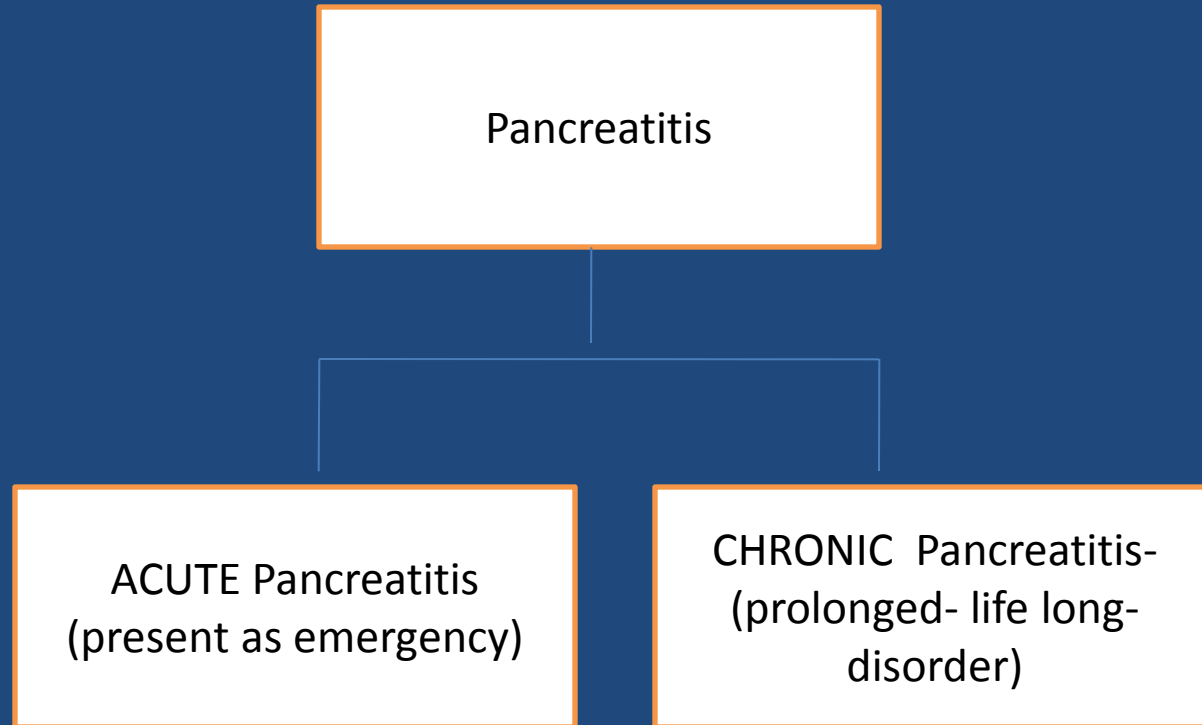
Exocrine function

- Normal fat digestion
 - Fat digestion begins in the mouth (very limited) and stomach (10-30% of all lipid breakdown)
 - Most fat digestion by pancreatic lipase
 - Approx 20,000-50,000 units of lipase are needed to digest a typical meal
- 2 key hormones
 - CCK – triggers the release of pancreatic enzymes from the pancreas
 - Secretin – stimulates bicarbonate secretion from the pancreas to ↑ pH (lipase inactivated in acidic environment)
- With pancreatic damage- lingual and gastric lipases cannot compensate 100% for loss of pancreatic function

PANCREATITIS



CLINICAL CLASSIFICATION



Acute Pancreatitis

**Two
phases**

Early

1st week

Late

After 1st week

Severity

Mild

No organ failure

Moderate

Organ failure
less than 48 h

Severe

Organ failure
longer than 48 h

**Two
types**

Oedematous

Complications

< 4 wk : acute peripancreatic collection
> 4 wk: pseudocyst

Necrotizing

< 4 wk : acute necrotic collection
> 4 wk : walled-off necrosis

Acute Pancreatitis - Fluid Collections

Interstitial Pancreatitis

< 4 weeks

Acute
Peripancreatic
Collection

> 4 weeks

Pseudocyst

Necrotizing Pancreatitis

< 4 weeks

Acute
Necrotic
Collection

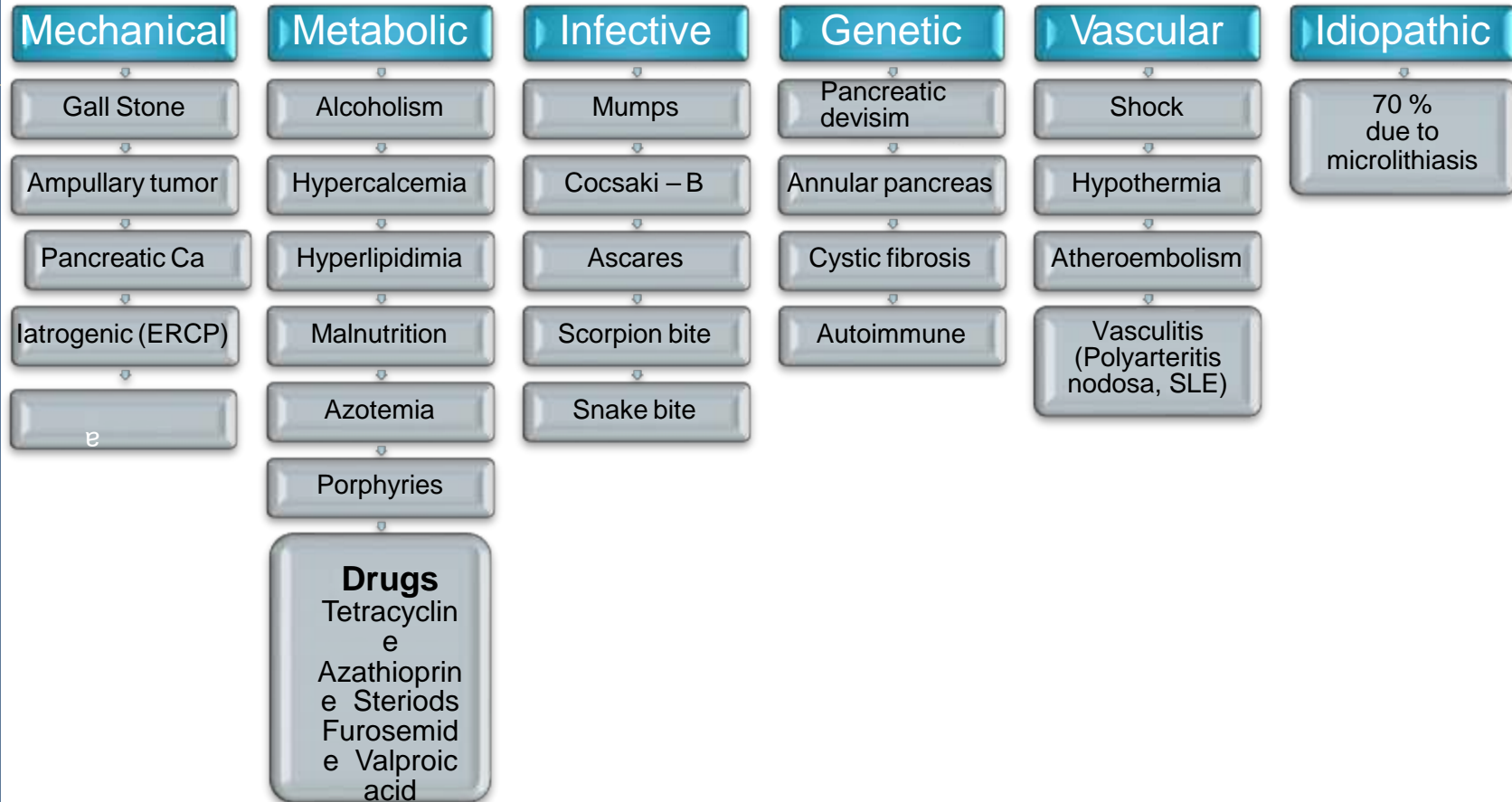
> 4 weeks

Walled off
Necrosis

PATHOPHYSIOLOGY

Autodigestion of pancreatic substance
by inappropriately activated pancreatic enzymes
(especially **trypsinogen**)

Etiology of Pancreatitis



Etiology of Acute pancreatitis

- Gallstones
 - Alcohol
 - Endoscopic retrograde cholangiopancreatography (ERCP) (overall 5% -20%)
- 80% of cases.
- Medications
 - Trauma
 - Neoplasms
 - Anatomic variants
 - Metabolic problems
 - Hypercalcemia
 - Hypertriglyceridemia
- 10% of patients.

Hyperlipidemia induced AP

- In the absence of gallstones and/or history of significant history of alcohol use, a serum triglyceride should be obtained and **considered the etiology if >1,000 mg /dl**
- **Lipase without increasment of serum amylase**
- In the injured pancreas, capillary permeability is increased which facilitates leakage of activated pancreatic enzymes
 - in turn promote local hydrolysis of triglycerides from chylomicrons
 - Which exhibit local toxicity towards capillary membranes causing further damage to pancreas



Risk factors of Pancreatic Cancer

- tobacco use
- family history of pancreatic cancer
- obesity
- diabetes
- advanced age
- Some evidence has suggested an association of increased consumption of red meat, processed meat, and meat cooked at high temperatures with pancreatic cancer . Consumption of fresh fruits and vegetables, particularly those high in dietary folate and cruciferous vegetables, may have protective effects . American cancer society, 2013

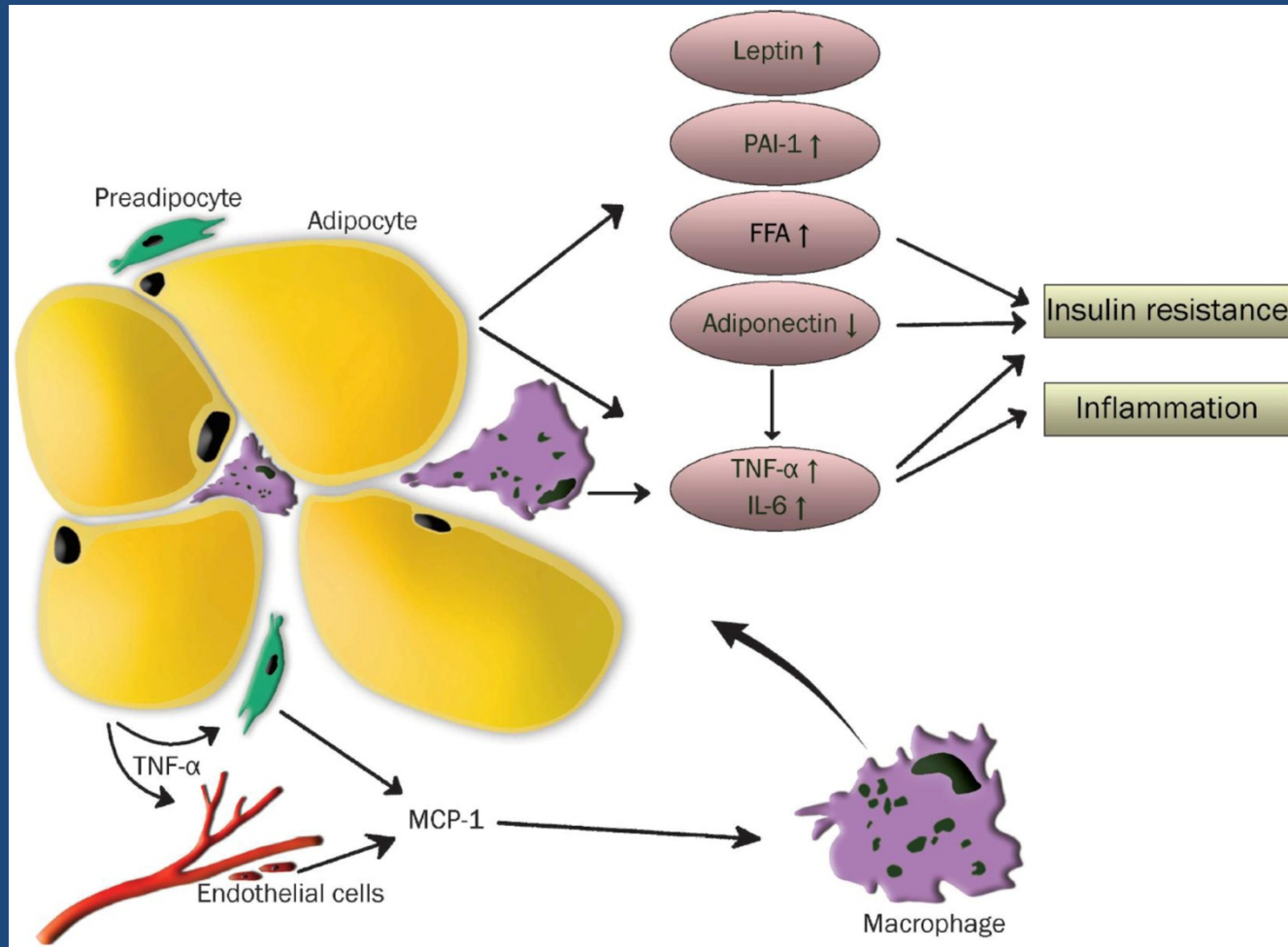
Pancreatic Cancer

Obesity and Diabetes as risk factors for
Pancreatic ductal adenocarcinoma (PDAC)

Central role of adipose tissue in mediating the
increased risk of PDAC by T2DM and obesity

Journal of the Academy of Nutrition and Dietetics
the Academy of Nutrition and Dietetics 118, Pages 555-567 (April 2018),

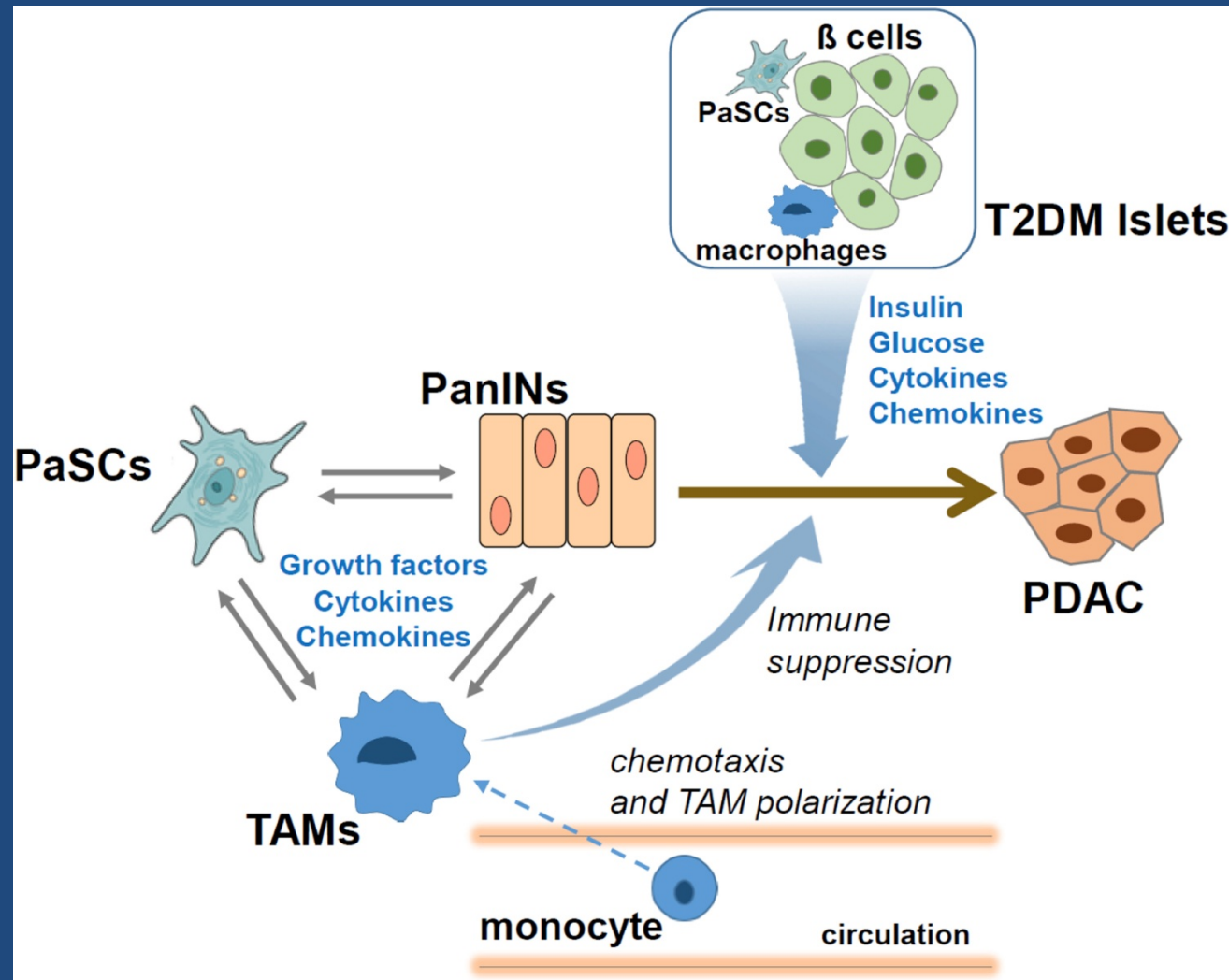
Obesity induced



Adipose tissue dysfunction during obesity.

Diabetes induced-crosstalk between Pre-cancer and PDAC cells ,immune cells and islets,

pancreatic pre-cancer (PanIN),
pancreatic stellate cells (PaSCs),
immune cells (eg, tumor-associated
macrophages [TAMs]),
and islets.



Promotion of pancreatic ductal adenocarcinoma (PDAC) by type 2 DM

Journal of the Academy of Nutrition and Dietetics 2018 118, 555-567)

Simplifying the process

- Insulin resistance and associated hyperglycemia, hyperinsulinemia, and inflammation have been suggested to be the underlying mechanisms contributing to development of diabetes-associated pancreatic cancer
- Signaling pathways that regulate the metabolic process also play important roles in cell production and tumor growth.

PANCREATIC ADENOCARCINOMA

Pancreatic Adenocarcinoma (exocrine tumor)
begins in the tissues of the pancreas, specifically
the cells that line the ducts of the pancreas.

Common symptoms:

upper abdominal pain, jaundice, loss of appetite,
nausea, vomiting, & weight loss


- Patients who develop cancer within the head of pancreas may undergo the Whipple procedure

Treatment

- Surgery
- Chemoterapy
- Radiation

Surgery options

- Distal pancreatectomy (PD)-for patents early P-CA and free of metastatic diseases
- Total pancreatectomy

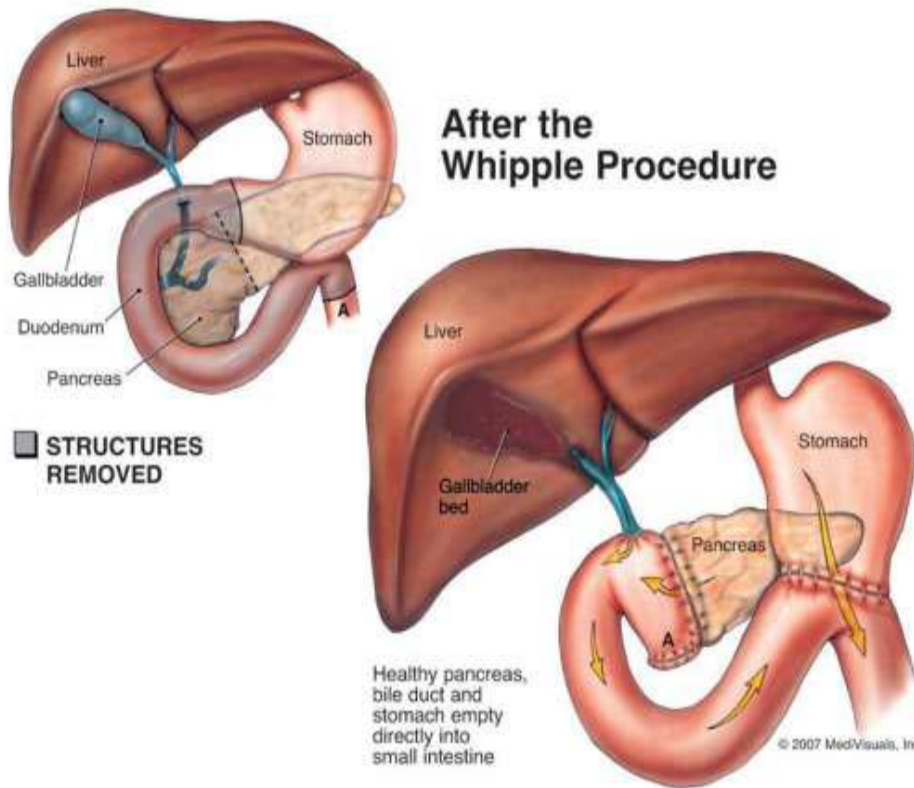


Optimizing nutrition after WHIPPLE PROCEDURE ALSO KNOW AS “ PANCREATODUODENECTOMY”

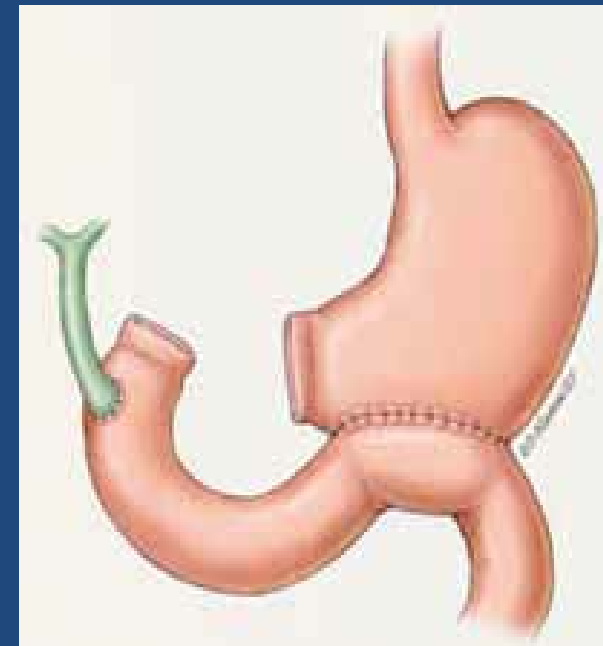
A procedure that involves removing the head of the pancreas, part of the small intestine (duodenum), gallbladder and a part of the bile duct.

- Remaining parts of the pancreas, stomach and intestines are reconnected to allow the body to digest food.
- Whipple procedure is used to treat tumors and other disorders of the pancreas, intestine and bile ducts
- WP and Total pancreatectomy are similar

BEFORE SURGERY VERSUS AFTER SURGERY



Total pancreatectomy.



After whipple

Symptoms of P-CA are vary

- depending on disease location and stage, although most pancreatic cancers occur at the head of pancreas

Symptoms may include

- anorexia, weight loss, jaundice, nausea/vomiting, abdominal discomfort, or mid-back pain .
- Liver function abnormalities, hyperglycemia, anemia,
- increased abdominal girth, and gastric outlet obstruction may also be present .
- Physical examination and review of systems may reveal jaundice, temporal wasting, hepatomegaly, ascites, dark urine, absence of bile pigments in stool, and pruritus

Total Pancreatectomy

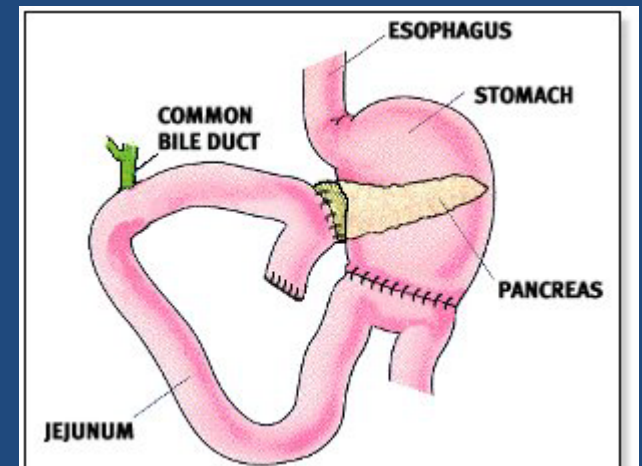
Total pancreatectomy (TP) is a treatment option for patients with

- neuroendocrine tumors,
- intraductal neoplasia,
- pancreatic adenocarcinoma,
- familial pancreatic cancer,
- and chronic pancreatitis (CP)

Support line 36 (5), 2014

Impaired digestion in pancreatic cancer

- Cancer in the head of the pancreas may obstruct the pancreatic duct, impairing secretion
- Surgery (e.g. Whipple) changes the mechanical and secretory process
- Damage to the intestinal mucosa (radiation therapy, surgery), may reduce CCK release
- Motility disturbances may affect secretory and motor functions of the GI tract



Signs and symptoms of malabsorption

- Steatorrhoea (foul smelling, fatty stools)
- Oily stools, undigested food in stools
- Diarrhoea
- Weight loss
- Bloating/ flatulence
- Abdominal pain/ cramping
- Dehydration
- Electrolyte disturbances



Adding pancreatic enzyme

- An alternative to formula change is the addition of pancreatic enzymes.
- Some patients may be able to take enzymes by mouth with physician approval. Otherwise, enzymes are administered via enteral access to achieve Nutrition

Clin Pract. 2014;29:312–321

NUTRITIONAL CONSIDERATIONS AFTER PD

Alteration of GI tract post Whipple procedure can result in multiple long term nutritional complications:

- Gastroparesis- delayed gastric emptying
- Dumping Syndrome
- Exocrine pancreatic insufficiency: Fat maldigestion
- Poor appetite/weight loss
- Endocrine insufficiency- Diabetes
- Nutrient deficiencies

Delayed gastric emptying

- DGE occurs in 19% to 57% of patients after pancreatic surgery
- An objective and universal definition for DGE has been suggested by the International Study Group of Pancreatic Surgery and includes nasogastric tube intubation lasting >3 postoperative days and the inability to tolerate a solid diet by postoperative day 7

DGE can be classified further into

- Mild
- moderate
- and severe

Ann Surg. 2009;249(6):986–994.

Delayed Gastric Emptying Consensus Definition

DGE Grade	Nasogastric Tube	Unable to tolerate Oral solids by POD	Vomiting Gastric Distention	Use of prokinetics
A	4-7 days or reinsertion >POD 3	7	May or may not be present	May or may not be needed
B	8 to 14 days or reinsertion >POD 7	14	Both present	Needed
C	>14 days or reinsertion >POD 14	21	Both present	Needed

DGE=delayed gastric emptying, POD=postoperative day

Surgery. 2007;142(5):761–768,

Symptoms of DGE

- nausea, vomiting, bloating, early satiety, and abdominal pain
- Nasogastric decompression and enteral nutrition
- (EN) beyond the gastrojejunostomy may be
- needed in some patients .
- Use of prokinetic agents such as metoclopramide or erythromycin may assist gastric motility
- Gastric emptying generally improves to preoperative levels within 6 months after surgery

After Surgery (ERASR) Society recommendations. *Clin Nutr.* 2012;31(6):817–830.

Endocrine Insufficiency

- Endocrine function is often compromised after PD and is related to the amount of pancreas removed
- Postoperative blood glucose control has been recommended in the range of 140 to 180 mg/dL
- Persistent hyperglycemia may require oral hypoglycemic agents or insulin
- Patients should be educated about signs and symptoms of hyper and hypoglycemia

After Surgery (ERASR) Society recommendations.
Clin Nutr. 2012;31(6):817–830

Dumping Syndrome

- **Dumping syndrome may result from gastric resection.**
- It is characterized by diarrhea or abdominal cramping and pain 15 to 30 minutes after meal consumption
- Late dumping syndrome, which occurs 1 to 3 hours after meals, is associated with feelings of weakness, flushing, and dizziness

Nutrition management of dumping syndrome

- may include consumption of five or six smaller meals, separating solids from liquids
- avoiding concentrated sources of sugar, and increasing protein consumption
- Supine positioning after meals has also been advised

Pancreatic Exocrine Insufficiency

- Decreased production of pancreatic enzymes used to digest food.
- Adequate enzyme replacement should be provided to all patients postoperatively.
- because dietary restriction of fat is not recommended

Pancreatic Insufficiency

- Anatomic and physiologic changes resulting from PD frequently lead to maldigestion
- Up to 82% of patients show evidence of fat malabsorption after PPPD
Matsumoto and Traverso documented reduced elastase concentrations reflective of pancreatic insufficiency at 6 months, 1 year, and 2 years after surgery in 48%, 73%, and 50% of cases, respectively. They concluded that exocrine supplementation should be provided to all patients postoperatively.

Matsumoto J, Traverso LW. Exocrine function following the Whipple procedure as assessed by stool elastase
J Gastrointest Surg. 2006;10(9):1225–1229.

Signs of pancreatic insufficiency

- Steatorrhea
- foul-smelling stool
- weight loss
- failure to thrive
- fat-soluble vitamin and mineral deficiencies

Weight Loss

- Weight loss has been estimated to occur in as many as 85% of pancreatic cancer patients, optimizing nutrition status in patients undergoing surgery for pancreatic cancer may support improved outcomes.
- Unintentional weight loss of more than 10% of body weight is associated with increased operative morbidity.
- pancreatic cancer patients without cachexia undergoing tumor resection has significantly better survival than those with cachexia, with weight loss identified as a prognostic factor for survival weight-stable patients undergoing

Symptoms of Malabsorption

Abdominal Symptoms	<ul style="list-style-type: none">•Bloating, distension and flatulence•Steatorrhoea (pale, oily, floating stool)•Diarrhoea (loose, large volume stool)•Faecal urgency•Reflux•Cramping abdominal pain and abdominal gurgling
Endocrine Function	<ul style="list-style-type: none">•Hypoglycaemia•Reduced insulin requirements in those already on insulin therapy

Symptoms of Malabsorption

Systemic / Biochemical	<ul style="list-style-type: none">• Vitamin A deficiency night blindness• Vitamin D deficiency• Low serum selenium, zinc, magnesium, calcium, phosphate, potassium, Vitamin E• Elevated parathyroid hormone (secondary to vitamin D deficiency)• Osteopenia / Osteoporosis
Nutritional	<p>Unexplained weight loss</p> <p>Sarcopenia</p> <p>Weakness / fatigue</p> <p>Food avoidance (due to concern over link with bowel symptoms)</p>

Micronutrient deficiencies Pre/post TP

Micronutrient	Potential cause of deficiency
Vitamin D3	<ul style="list-style-type: none">- Loss of exocrine pancreas- Inadequate PERT
Vitamin E	<ul style="list-style-type: none">- Loss of exocrine pancreas- Inadequate PERT
Vitamin A	<ul style="list-style-type: none">- Loss of exocrine pancreas- Inadequate PERT
Vitamin B-12	<ul style="list-style-type: none">-SIBO-Gastric acid suppression--Decreased proteolytic enzyme production
Folate	<ul style="list-style-type: none">-PH changes
Iron	<ul style="list-style-type: none">-Gastric acid suppression-Resection of duodenum and jejunum

Micronutrient deficiencies Pre/Post TP

Micronutrient	Potential cause of deficiency
Calcium	<ul style="list-style-type: none">- Resection of the duodenum- Vitamin D deficiency- Malabsorption
Copper	<ul style="list-style-type: none">-Gastric acid suppression- Resection of the duodenum-Previous whipple surgery
Zinc	<ul style="list-style-type: none">-Resection of the duodenum and jejunum- Gastric acid suppression- Pancreatic insufficiency- Previous whipple surgery
Selenium	<ul style="list-style-type: none">-Resection of the duodenum and jejunum-Oxidative stress



Type 3c or pancreatogenic Diabetes

different from both of type 1 or 2 DM

- There are specific hormonal differences between type 1, 2 and 3c diabetes, including low pancreatic polypeptide, insulin and glucagon levels in type 3c diabetes
- T3c more brittle than type 1 or type 2 diabetes; patients often require insulin therapy and are prone to significant episodes of hypoglycaemia due to enhanced peripheral insulin sensitivity and a decrease in glucagon production. . Hyperglycemia is frequent due to unsuppressed hepatic glucose production
- Typically, those with type 3 diabetes are older than those with type 1, but not type 2 diabetes.
- They have lower BMI than in type 2 diabetes.
- The risk of hypoglycemia is similar to that of type 1 diabetes, exacerbated in pancreatogenic diabetes by poor diet, malabsorption and, for some patients, persistent alcoholism

Goals of nutritional management

- Prevent malnutrition and nutritional deficiencies
- Maintain normal blood sugar level
- Prevent or optimally manage diabetes, kidney problems, and other conditions associated with chronic pancreatitis
- Avoid causing an acute episode of pancreatitis

Nutritional support in mild-to-moderate pancreatitis

1. Step — fasting
(2–5 day) — treat the cause of pancreatitis
— analgesics
— i.v. fluid and electrolyte replacement
↓
2. Step — refeeding
(3–7 day) — diet — rich in carbohydrates
no pain — moderate in protein
enzymes regredient — moderate in fat
↓
3. Step — normal diet



Nutrition in Acute Pancreatitis

- Targeted nutritional interventions
- Enteral or Parenteral nutrition
- Nutritional supplements, including
 - antiinflammatory imunonutrients ; glutamine. Omega 3 fatty acids
 - supplements with antioxidant; glutamine, vitamin C.
 - prebiotics and synbiotics



Glutamin and Omega 3 fatty acids

- Glutamin supplementation with TPN has promising clinical outcome
- Enteral glutamin supplementation needs to be investigated
- Omega 3 fatty acids beneficial with EN/PN. It suppress SIRS, modulate the balance of pro/anti- inflammatory cytokines and improve AP associated conditions

Vitamins

Oxidative stress is involved in the onset of AP and development of SIRS responses

Plasma concentration of Vitamin A and C is lower in AP pts. Supplementation with A,E,C is promising results

Vitamin D mainly from milk is inversely related with gall-stone related AP

Needs evaluation for dosing and timing

Generally

- Oral soft or regular diet can be beneficial if tolerated
- When oral feeding not tolerated NG/NJ feeding should start in 72 hrs
- Then PN can be considered



Nutrition support during Necrotizing pancreatitis

American collage of gastroentology

ACG 2013 Recommendations

EARLY AGGRESSIVE IV hydration is the only intervention in treating AP.

Followed by antibiotic coverage of the infected necrotic area, thereby eliminating the need for surgery

Reason for early hydration

- Frequent hypovolemia due to
 - vomiting,
 - reduced oral intake,
 - third spacing of fluids (increased vascular permeability)
 - increased respiratory losses, and
 - Sweating

provides micro- and macro circulatory support
to prevent serious complications such as
pancreatic necrosis

WHAT , WHEN, HOW MUCH

- Lactated Ringer 's solution may be the preferred isotonic crystalloid replacement fluid
 - Ringer lactate is better electrolyte balance and more pH-balanced
 - Normal saline given in large volumes may lead to the development of a non-anion gap, hyperchloremic metabolic acidosis and increased chances of SIRS
 - Low pH activates the trypsinogen, makes the cells more susceptible to injury and increases the severity of established AP
- Early aggressive IV hydration is most beneficial during the first 12 – 24 h, and may have little benefit beyond this time period
- Aggressive hydration, defined as 250 – 500 ml per hour of isotonic crystalloid solution should be provided to all patients, unless cardiovascular, renal, or other related comorbid factors exist.
- In a patient with severe volume depletion, manifest as hypotension and tachycardia, more rapid repletion (bolus) may be needed
- Fluid requirements should be reassessed at frequent intervals within 6 h of admission and for the next 24 – 48 h.

Nutrition

☐ In mild AP

- oral feedings can be started immediately if there is no nausea/vomiting, and the abdominal pain/tenderness/Ileus has resolved (amylase return to normal, patient feel hunger)
- Initiation of feeding with a small and slowly increasing low-fat (low-protein) soft diet appears as safe as a clear liquid diet, providing more calories

☐ Stepwise manner increase from clear liquids to soft diet NOT necessary

☐ In severe AP

- Enteral route is recommended to prevent infectious complications
- Parenteral nutrition should be avoided, unless enteral route is not available, not tolerated, or not meeting caloric requirement

Enteral feeding

- presence of complications (pancreatic ascites, fistula formation or fluid collection) is not a contraindication to enteral feeding

ACG 2013

In mild AP

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RATIONALE OF EARLY ENTERAL NUTRITION

- The need to place pancreas at rest until complete resolution of AP no longer seem essential
 - Bowel rest associated with intestinal mucosal atrophy and bacterial translocation from gut and increased infectious complications
- Early enteral feeding maintains the gut mucosal barrier,
prevents disruption, and prevents translocation of bacteria that seed pancreatic necrosis
 - Decrease in infectious complications, organ failure and mortality

RATIONALE	MANAGEMENT
PREVENTION OF STERILE NECROSIS	Early aggressive IV hydration
TREATMENT OF INFECTED NECROSIS	Early enteral feeding(NOT antibiotics)
PREVENTION OF INFECTED NECROSIS	Antibiotics, drainage, necrosectomy

Rather than using antibiotics to prevent infected necrosis.....start early enteral feeding to prevent translocation of bacteria

Route of enteral Nutrition

- jejunal administration induces less pancreatic secretory responses than gastric or duodenal perfusion of enteral diets.
- This provides a theoretical rationale for jejunal administration of nutrients in patients with acute pancreatitis receiving enteral nutrition.

Route of enteral Nutrition

- Traditionally nasojejunal route has been preferred to avoid the gastric phase of stimulation BUT
 - Nasogastric route appears comparable in efficacy and Safety

MERITS OF NASOGASTRIC ROUTE	DEMERITS OF NASOGASTRIC ROUTE
NG tube placement is far easier than nasojejunal tube placement (requiring interventional radiology or endoscopy, thus expensive) especially in HDU/ICU setting	Slight increased risk of aspiration (Can be overcome by placing patient in upright position and be placed on aspiration precautions)

- In chronic pancreatitis more than 80% of patients can be treated adequately with normal food supplemented by pancreatic enzymes.
- 10–15% of all patients require nutritional supplements, and in approximately 5% tube feeding is indicated

About lipids

- IV lipid does not increase exocrine pancreatic secretion
- Stimulatory effect of lipid administered into the small intestine depends on the anatomic site of administration.
 - Lipid perfused into the duodenum is a powerful stimulus for exocrine pancreatic secretion.
 - If, however, the same amount of lipid is perfused into the jejunum, then only a minimal stimulation of exocrine pancreatic secretion occurs

proteins

- Amino acids, when given parenterally, do not stimulate the exocrine pancreas directly
- stimulate gastric acid secretion which may stimulate itself in the duodenum, pancreatic secretion(INDIRECTLY)

Carbohydrates

- Preferred energy supply in AP
 - can be easily supplied
 - Protein conserving : prevents gluconeogenesis from protein degradation
 - reduce the potential risk of hyperlipidemia
- physiological maximum to the rate of glucose oxidation (4 mg/kg/min)
 - provision of glucose in excess of this is wasteful both in terms of lipogenesis and glucose recycling,
 - Results in hyperglycemia and hypercapnia IV high doses of glucose
 - Doesn't stimulate pancreatic exocrine secretions
 - carries the risk of hyperglycemia as the insulin response is often impaired.

Energy supply

- In severely ill patients, neither hypercaloric nor isocaloric nutritional support can prevent protein catabolism.
 - both enhance the metabolic burden as measured by energy expenditure, thermogenesis, urea production rate, glucose and lactate levels
- • Hypocaloric energy supply of 15–20 kcal/kg/day is more suitable during the early catabolic stage of non-surgical patients with MOF

Energy Supply

- Goal of 1.2–1.5 g/kg/day of protein intake is optimal for most patients with AP
- Attempt to deliver the caloric need by enteral route
 - determined by patient tolerance.
 - If the enteral supply is inadequate, then the rest should be given parenterally.
 - When enteral nutrition is impossible total parenteral nutrition should be started.
- impaired glucose oxidation rate cannot be normalized by insulin administration or by increasing glucose administration.
 - Normally, the blood glucose levels should not exceed 10 mmol/l.
 - Insulin doses higher than 4–6 units/h should be avoided
- Aggressive nutritional support (enteral or parenteral) is not required for mild-to moderate forms of acute pancreatitis (the majority of patients).

TPN in AP

Standard treatment for providing nutrients to patients with severe AP if enteral route not tolerated or not fulfilling the requirements

- avoid stimulation of exocrine pancreatic secretory responses

(‘to put the pancreas at rest’-*NO LONGER RECOMMENDED if enteral tolerated*) and

- secondly, to improve the nutritional status

Merits	Demerits
useful as an adjunct in the nutritional maintenance of the patients.	<ul style="list-style-type: none">• catheter-related sepsis and metabolic disturbances such as hyperglycemia (due to overfeeding)• prolonged TPN may suppress the immune system, promote gastrointestinal leakage by a loss of intestinal mucosal barrier with the potential risk of subsequent bacterial translocation



Nutrient requirements:

- – energy 25–35 kcal/kg BW/day;
- – protein 1.2–1.5 g/kg BW/day;
- – Carbohydrates 3–6 g/kg BW/day
- (BSR <10 mmol/l)
- – Lipids up to 2 g/kg BW/day
- (TAG <12 mmol/l)

patients with necrotizing pancreatitis should be treated as other critically ill patients.

Treatment of pancreatic insufficiency

- provision of pancreatic enzymes with all intake containing fat, including EN and oral intake.
- Dosing recommendation are 1,000 to 2,000 IU/kg lipase per meal or 25,000 to 50,000 IU for a main meal and 10,000 to 25,000 IU for snacks, without exceeding 10,000 IU/kg lipase per day
- Enzyme delivery during enteral feeding should occur every few hours.
- Adequate enzyme replacement should be provided because dietary restriction of fat is not recommended

Questions on enzyme delivery from USA DNS group

- For patients on continuous TF's requiring pancreatic enzyme replacement, I'm wondering whether practitioners are adding the enzyme "recipe" to the TF formula or flushing it through the tube every so many hours?
Thanks.
- Our practice is to give it as an intermittent bolus administration, which I don't think is very effective.
- Have you heard of Relizorb? They make a pancreatic enzyme cartridge that connects to the tube setup and mixes enzymes with the enteral formula before exiting the tube.
- We use only closed systems in house so we flush the enzyme solution every 4 hours for continuous enteral feeds. We also use Relizorb and have seen great success. The nurses love the ease of its use and don't have to wake the patient up in the middle of the night to infuse enzymes. On the outpatient side our CF patients are seeing great improvements in weight.

Preoperative Nutrition Considerations

- Guidelines for perioperative care for PD provided by the Enhanced Recovery After Surgery (ERAS) suggest that significantly malnourished patients scheduled for PD be “maximized” with oral or enteral nutrition support preoperatively
- A.S.P.E.N. guidelines support preoperative nutrition for moderately or severely malnourished patients if provided for 7 to 14 days before the procedure
- The European Society for Parenteral and Enteral Nutrition (ESPEN) guidelines encourage oral nutrition supplements or EN preoperatively for patients who cannot meet their energy needs orally

Postoperative Nutrition Considerations

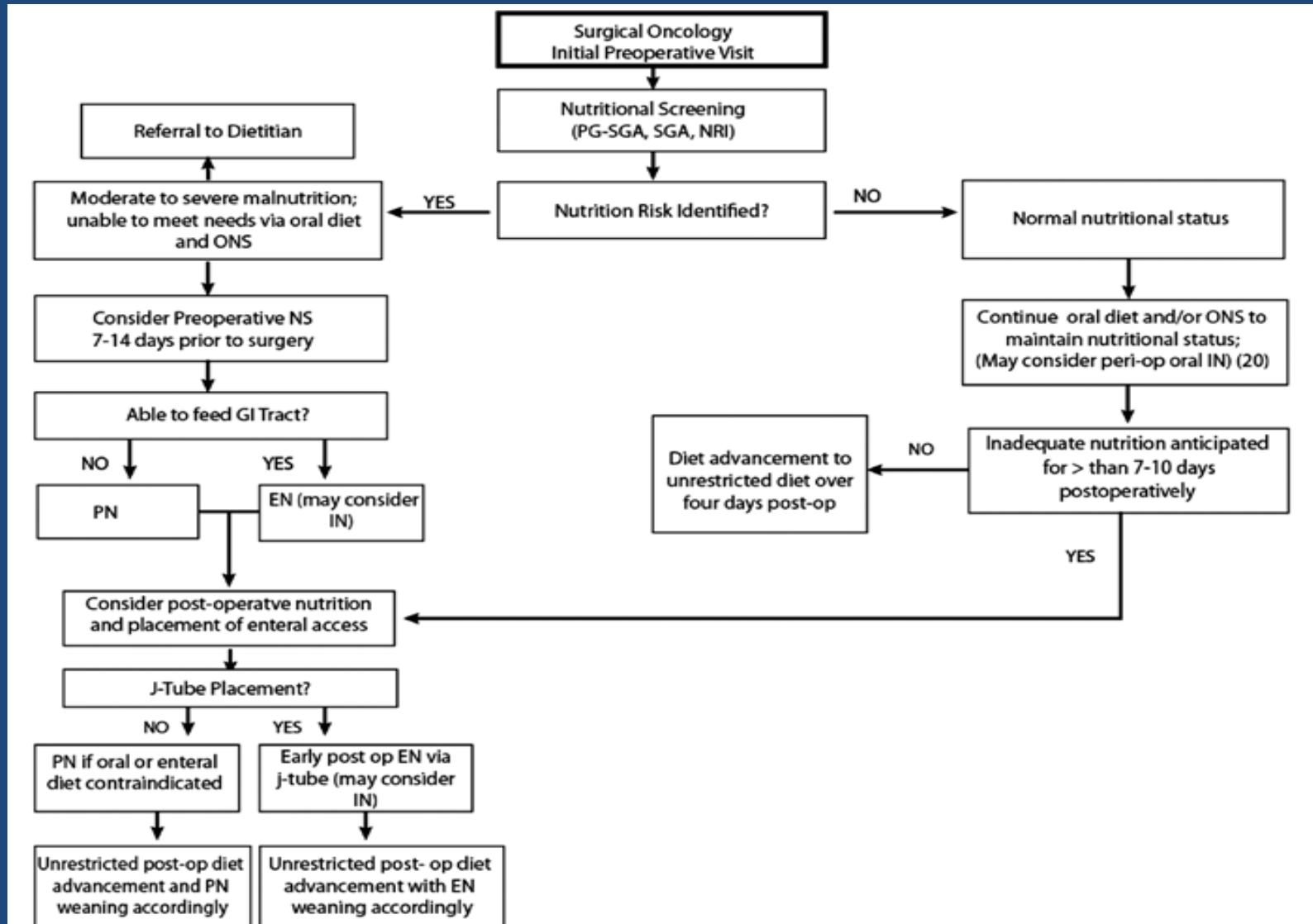
- ERAS guidelines suggest an early normal, unrestricted diet after PD, with cautious, patient-controlled intake over the first 4 days after surgery EN or PN may be needed if complications persist, with PN indicated only if EN and oral diet cannot be tolerated
- ESPEN guidelines suggest administration of EN postoperatively in patients with malnutrition and when intake is anticipated to be inadequate (<60% needs) for 10 days
- Guidelines for PN include postoperative administration in undernourished patients unable to tolerate EN, in those unable to absorb or consume adequate oral or enteral nutrients postoperatively for at least 7 days, or when prolonged gastrointestinal failure exists . EN or combined EN and PN are the first option



Formula Selection and Immunonutrition

- Immunonutrition (IN) employs specific nutrients in feedings, such as arginine, glutamine, omega-3 fatty acids, and nucleotides, in an effort to improve patient outcomes
- Perioperative IN provided for 5 to 7 days is supported by moderate-to-weak evidence in the ERAS guidelines for PD . While not specific to PD patients
- 2016 A.S.P.E.N. guidelines consider the administration of immuneenhancing formulas specifically containing arginine, nucleic acids, and essential fatty acids for 5 to 7 days preoperatively in malnourished surgical cancer patients
- ESPEN guidelines suggest EN with immune-modulating substrates perioperatively independent of nutritional risk in cancer patients undergoing PD, esophagectomy, or gastrectomy for 5 to 7 days before and after surgery

Clinical pathway for pancreaticoduodenectomy nutrition support



Management of TP

Total pancreatectomy (TP) is associated with both macro-nutrient and micro-nutrient malabsorption and metabolic abnormalities

Drawbacks of TP manageable

- **pancreatic enzyme supplementation**
- **control of diabetes mellitus**

Is there an indication for supplementing oral glutamine?

- Currently, no clear recommendation can be given regarding the supplementation of oral glutamine
- oral glutamine supplementation as a single substance are limited. In pancreatic surgery oral preconditioning with glutamine, antioxidants, and green tea extract versus placebo
- elevated plasma vitamin C concentrations significantly and improved total endogenous antioxidant capacity without reducing oxidative stress and inflammatory response

Is there an indication for supplementing immunonutrients

- Peri- or at least postoperative administration of specific formula enriched with immunonutrients (arginine, omega-3-fatty acids, ribo-nucleotides) should be given in malnourished patients undergoing major cancer surgery
- There is currently no clear evidence for the use of these formula enriched with immunonutrients vs. standard oral nutritional supplements exclusively in the preoperative period

Synbiotics- “ecoimmunonutrition”

- refers to formulae containing synbiotics with fibre and Lactobacillus.
- A significant reduction of the rate of postoperative incidence of infections after pancreatic and hepatobiliary resections

Ecoimmunonutrition with pre- pro- and synbiotics offer to be suitable tools

ESPEN guideline: Clinical nutrition in surgery, 2017

KEY POINTS

- RD's should provide an Individualized nutrition therapy based on each patient's preferences and ability to handle certain foods.
- RD's play a crucial role in counseling this patient population to:
 - avoid unnecessary dietary restriction
 - to increase variety in the diet
- - to improve the patient's quality of life through close monitoring and attention to signs and symptoms
- to help optimize nutritional status and help prevent