Principles of gut nutrition

- common sense!
- find out which part of the gut is affected and recall the function of that part
- predict likely consequences (nutrient losses, requirements)

Principles of gut nutrition
- early enteral nutrition, with complex nutrients
- fibre and ?probiotics
- activity, fluid, psychosocial support, QOL
- “The gut is as individual as the face”

Geography of the gut

<table>
<thead>
<tr>
<th>Duodenum (25-30cm)</th>
<th>PROXIMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>• receives pancreatic secretions and bile</td>
<td></td>
</tr>
<tr>
<td>• some digestion</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Jejunum (200-300cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• digestion</td>
</tr>
<tr>
<td>• absorption</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ileum (300-400cm)</th>
<th>DISTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>• absorption</td>
<td></td>
</tr>
<tr>
<td>• Peyer’s patches (gut-associated lymph tissue)</td>
<td></td>
</tr>
<tr>
<td>• “ileal brake” (ileocaecal region feeds back to control motility)</td>
<td></td>
</tr>
<tr>
<td>• ileo-caecal valve prevents colonic bacteria coming up to small bowel</td>
<td></td>
</tr>
</tbody>
</table>

Absorptive sites in the gut

<table>
<thead>
<tr>
<th>duodenum (DIGESTION)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• minerals (calcium, magnesium, zinc, iron)</td>
</tr>
<tr>
<td>jejunum (DIGESTION/ABSORPTION)</td>
</tr>
<tr>
<td>• products of carbohydrate digestion (saccharides)</td>
</tr>
<tr>
<td>• watersoluble vitamins</td>
</tr>
<tr>
<td>• starts absorbing nutrients listed under “ileum”</td>
</tr>
<tr>
<td>ileum (ABSORPTION / RECYCLING)</td>
</tr>
<tr>
<td>• products of protein digestion (peptides and aa)</td>
</tr>
<tr>
<td>• fat-soluble vitamins</td>
</tr>
<tr>
<td>• products of fat digestion (monoglycerides, fa, cholesterol)</td>
</tr>
<tr>
<td>• bile salts, vitamin B12</td>
</tr>
<tr>
<td>colon (ABSORPTION)</td>
</tr>
<tr>
<td>• water, sodium and potassium</td>
</tr>
</tbody>
</table>

Motility in the gut

<table>
<thead>
<tr>
<th>oesophagus</th>
</tr>
</thead>
<tbody>
<tr>
<td>• upper: voluntary control</td>
</tr>
<tr>
<td>• lower: lower oesophageal sphincter (vagus nerve)</td>
</tr>
<tr>
<td>stomach</td>
</tr>
<tr>
<td>• pacemaker cells in fundus</td>
</tr>
<tr>
<td>• pyloric sphincter (vagus nerve, mechanical pressure (angle), particle size + composition)</td>
</tr>
<tr>
<td>small bowel</td>
</tr>
<tr>
<td>• pacemaker cells in duodenum control small bowel peristalsis</td>
</tr>
<tr>
<td>• ileo-caecal valve (vagus nerve), ileal brake</td>
</tr>
<tr>
<td>colon</td>
</tr>
<tr>
<td>• pacemaker cells in ascending colon</td>
</tr>
<tr>
<td>• right side: thin stool (slow transit / absorption)</td>
</tr>
<tr>
<td>• left side: thick stool, strong muscle control</td>
</tr>
</tbody>
</table>
Outline
1. oesophageal
2. gastric
3. pancreatic / biliary
   - an afterthought. . . . . . . chyle leaks
4. immune function and the gut
5. small bowel
6. large bowel

Please Note: the journal references provided here are for when you are on placement or working, and need further information. They are not required reading for this course.

Anatomy of the oesophagus
- What is the role of the oesophagus? – it is a muscular tube about 24cm long, 1.5-2.5cm in width and responsible for peristalsis. The Lower Oesophageal Sphincter prevents food re-entering the oesophagus once in the stomach
- What can go wrong?
  - gastro-oesophageal reflux
  - oesophageal obstruction
  - oesophageal strictures
  - achalasia
- What are the nutritional consequences?

Anatomy of the stomach
- Location
- Muscular wall
- Layers

Gastro-Oesophageal Reflux (GORD)
- Definition: the recurrent forcing of gastric contents back up the oesophagus causing a burning sensation and inflammation of the oesophageal mucosa (oesophagitis)
- Refluxed stomach contents may contain partly digested foods, acid, pepsin, bile and pancreatic enzymes
- Diagnosis: endoscopy, barium swallow

Causes of oesophageal reflux
- lower oesophageal sphincter pressure (eg in pregnancy, obesity)
- increased abdominal pressure (eg coughing)
- recurrent vomiting
- restrictions on gastric emptying (eg hiatus hernia: protrusion of part of the stomach above the diaphragm muscle ↓ stomach volume and ↑ gastric pressure)
- smoking, alcohol and some drugs also related

Symptoms of oesophageal reflux
- substernal burning (heartburn), pain, pressure (may be mistaken for a heart attack)
- excessive salivation
- coughing, choking, pulmonary aspiration
If prolonged:
- ulceration of oesophagus (bleeding/perforation)
- stricture, Barrett’s Oesophagitis
- ↑ risk of oesophageal cancer
- dysphagia and odynophagia

Goals of treatment
- eliminate or minimise oesophageal reflux
- neutralise / reduce gastric acid
- achieve and maintain a healthy weight
- stop smoking and reduce alcohol intake
  - medication
  - surgery
  - nutrition
Medical and surgical treatment

**Medications**
- antacids
- histamine H\(_2\) receptor antagonists
- prokinetic drugs
- Proton Pump Inhibitors (PPIs)

**Surgical fundoplication** (if severe reflux)

**What nutritional advice could you suggest?**
- Small frequent meals (keep fluids in between meals) to reduce abdominal distension and intra-gastric pressure
- Strategies for weight reduction if overweight
- Avoid foods that slow stomach emptying (eg alcohol, fatty foods)
- Avoid foods that can irritate the oesophageal mucosa (eg bitter foods, spicy foods, acidic foods)

**Other tips**
- Elevate the bed head and avoid eating 2-3 hours before bed
- Avoid tight or constricting clothes
- Remain upright for 2 hours after meals

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**Achalasia**

- motor disorder of the oesophagus resulting in:
  ⇒ lower oesophageal sphincter (LOS) fails to relax during swallow → food unable to pass → food builds up in oesophagus which progressively dilates ⇒ aperistalsis (absence of normal rhythmic contractions post swallow)

**Management of achalasia**

- treatment is often to control symptoms only
- medications – relaxation of the LOS
- balloon dilatation
- chemical paralysis (botulinum toxin A)
- surgical
- nutritional management:
  - minimal evidence base
  - modify texture (liquid or semi-solid)
  - avoid irritants eg caffeine, alcohol, spices
  - nutrition support if necessary

---

**Oesophageal obstructions**

**Definition:** the inability of food and fluid to pass through the oesophagus due to a partial or complete obstruction

**Common causes:**
- benign or malignant tumours
- inflammation or stricture
  - as a result of reflux, trauma, radiotherapy etc

**Symptoms of obstruction**

- dysphagia (difficulty swallowing)
  - coughing / regurgitation on intake
  - sensation of food sticking in oesophagus
  - risk of aspiration
  - progressive deterioration (solids - liquids)
- odynophagia (pain on swallowing)
- weight loss and anorexia
- iron deficiency anaemia
Medical management of obstruction

Carcinoma:
- surgery (if operable): oesophageal resection with reconstruction +/- proximal gastrectomy (Ivor-Lewis procedure)
- adjunctive treatment: chemotherapy +/- radiotherapy
- palliative: endoscopic intubation (oesophageal stent)

Stricture: dilatation

Nutrition management of obstruction
(pre-op and palliative)
- provide small, frequent meals
- ensure adequate hydration
- provide high protein, high energy diet
- modify texture of meals as needed
- if stent, avoid ‘sticky’ foods, and consume plenty of fluids with meals to flush stent
- palliative nutrition care

Nutrition management of obstruction
(surgical)

Oral Nutrition
- Initially NBM
- Ideally start fluids ASAP (Surgeon advice varies)
- Continue until oral intake is fully established (may be needed until adjunct therapy is ceased)
- Progress to soft, moist foods
- May be needed long term where swallow is impaired

Nutrition Post Oesophageal Resection

Ivor-Lewis Oesophagectomy

Anatomy of the stomach

- What is the role of the stomach? – it holds a large volume of food and starts meal breakdown.
- extensive blood and nerve supply (of particular note is the vagal nerve)
- produces ~2L secretions/day (including intrinsic factor, HCl, gastrin & pepsinogen)
- pyloric sphincter regulates the release of a meal into the duodenum
- What can go wrong:
  - gastric ulcer
  - gastroparesis
  - gastric carcinoma
- What are the nutritional consequences?

Long term aims & advice post-oesophagectomy

Aims:
- Maintain nutritional status & avoid deficiencies
- Manage symptoms (mainly reflux, vomiting, nausea)
- Palliative care if necessary (only 25% survival rate after 5 years)

Nutritional Advice
- HEHP
- Take time with meals
- Small frequent meals
- Avoid fluids for ½ hour before and after meals
- Avoid lying down straight after meals
**Symptoms**
- may be completely asymptomatic
- heartburn
- abdominal pain and discomfort
- nausea and/or vomiting
- loss of appetite; weight loss
- malaena (old dark blood in faeces)
- complications - haemorrhage, perforation, gastric outlet obstruction, ↑↑↑ risk of cancer

**4. Gastric and duodenal ulcers**
- **Definition:** erosion of the mucosal tissue of the distal oesophagus, stomach or proximal duodenum
- **Caused by:**
  - hypersecretion of gastric HCl and pepsin
  - prolonged or excessive use of aspirin products
  - Helicobacter pylori infection
  - Stressors (surgery, trauma, RTx, severe burns)

**Medical management of ulcers**
- reduce gastric acid secretion - histamine H₂ receptor antagonists (ranitidine)
- reduce direct irritation of gastric mucosa - antacid (mylanta)
- treat H. pylori infection (if present) - antibiotics (amoxycillin)
- surgery: partial gastrectomy / duodenal ulcer oversew
- vagotomy: resection or removal of part of vagus nerve to reduce acid secretion, promote gastric emptying and minimise pain

**Nutritional management of ulcers**
- no special “Ulcer Diet”
- optimise nutrition and minimise food avoidances (eliminate foods only if problematic eg spicy foods, windy vegies, alcohol, caffeine)
- supplement any nutritional deficiencies ie Vit C, Fe
- small frequent meals (reduces effect of acid on empty stomach)

**Gastroparesis**
- a gastric motility disorder resulting in delayed gastric emptying. Common in diabetic patients
- symptoms include nausea, vomiting, early satiety, bloating and discomfort.
- medical management similar to that of achalasia
- diet management
  - small frequent meals, HEHP, Liquids, Lower fat, Lower fibre (prevent bezoar formation)
  - nutrition support

**Further reading:**

**Gastric carcinoma**
- **Diagnosis:** CT, endoscopy, laparotomy
- **Symptoms:**
  - often asymptomatic until cancer is advanced
  - weight loss (due to decreased intake and increased nutritional needs)
  - fatigue
Medical management of gastric cancers

- 50% of gastric cancers are incurable (may have palliative chemo/radiotherapy)
- surgery is the only cure
  - partial gastrectomy - removal of cancerous segment (preserve stomach capacity)
  - total gastrectomy - resection of entire stomach +/- reconstruction
- 5 year survival after surgery is 10-70%

Total gastrectomy with roux-en-y
(Note patients can also undergo an oesopho-gastrectomy)

Partial gastrectomy (Bilroth 1 & Bilroth 2)

- significant and rapid weight loss due to reduced intake, because of:
  - ↓↓ ↓↓ stomach size, early satiety, feeling of fullness
  - ↓↓ ↓↓ appetite and nausea, vomiting
  - altered gastric emptying (dumping)
- malabsorption due to:
  - rapid gastric emptying
  - decreased secretion of intrinsic factor, pancreatic enzymes
  - poor mixing of food with bile and enzymes
  - decreased absorption of fat, Fe, Ca, fat soluble vitamins and especially protein
- anaemia & oesteomalacia
- bezoar formation due to abnormal stomach motility

Immediate post-op nutritional management

- jejunal feeding if needed
  (as for oesophageal resection)
- reintroduction of oral diet – soft foods
- small frequent meals to prevent dumping syndrome and early satiety
- avoidance large amounts of hyperosmolar food and fluid

Gastric resection - consequences

- significant and rapid weight loss due to reduced intake, because of:
  - ↓↓↓ stomach size, early satiety, feeling of fullness
  - ↓↓↓ appetite and nausea, vomiting
  - altered gastric emptying (dumping)
- malabsorption due to:
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  - decreased secretion of intrinsic factor, pancreatic enzymes
  - poor mixing of food with bile and enzymes
  - decreased absorption of fat, Fe, Ca, fat soluble vitamins and especially protein
- anaemia & oesteomalacia
- bezoar formation due to abnormal stomach motility

Long term nutritional management

- HPHE diet, with supplement drinks if needed
- small frequent meals
- manage malabsorption (& dumping)
  (what type of malabsorption?)
  (slow eating; ?MCT oil; ?low lactose diet)
- screen for and correct specific nutritional deficiencies (eg B12 (no intrinsic factor post total resection), folate, vitamin C, vitamin D, iron, calcium)
Medical management

Medications to control symptoms:
- delay gastric emptying
- antidiarrhoeals if indicated (codeine phosphate, loperamide)
- antibiotics (if bacterial overgrowth)

What is dumping syndrome?
- occurs mostly with total gastrectomy
- a complex physiologic response to rapid emptying of hypertonic contents into the duodenum (when lacking pyloric sphincter)
- involves 2 stages

Anatomy of the pancreas

What is the role of the pancreas?
- it is a large gland that attaches to the duodenum via 2 ducts - the pancreatic duct & the common bile duct. Produces pancreatic digestive enzymes (exocrine) and insulin and other hormones (endocrine). A highly metabolic organ

What can go wrong?
- pancreatitis (acute & chronic)
- pancreatic resection

Stage One - early stage dumping
- 10-30 minutes from start of meal
- fluid shifts in the small bowel in response to hyperosmolar content of meal
- leads to:
  - vasomotor symptoms – palpitations, fatigue, faintness
  - abdominal symptoms - bowel distension, nausea, cramps, and explosive diarrhoea

Stage Two - late dumping
- 1-3 hours after meal
- reactive hypoglycaemia (rebound reaction to large glucose load)
- perspiration, weakness, shakiness, anxiety and poor concentration level

Medical management

PANCREATITIS = inflammation of the pancreas

Two types (essentially different diseases):

ACUTE
- sudden inflammatory disease (often caused by pancreatic duct obstruction)
- enzymes activated in pancreas instead of duodenum
- autodigestion of pancreas

CHRONIC
- gradual damage to pancreas resulting in destruction of cells and reduction in function
- decreased enzymes (maldigestion)
- decreased hormones (impaired glucose tolerance)


What is dumping syndrome?

- involves 2 stages

Stage One
- early stage dumping
- 10-30 minutes from start of meal
- fluid shifts in the small bowel in response to hyperosmolar content of meal
- leads to:
  - vasomotor symptoms – palpitations, fatigue, faintness
  - abdominal symptoms - bowel distension, nausea, cramps, and explosive diarrhoea

Stage Two
- late dumping
- 1-3 hours after meal
- reactive hypoglycaemia (rebound reaction to large glucose load)
- perspiration, weakness, shakiness, anxiety and poor concentration level

What are the nutritional consequences?
### CHRONIC Pancreatitis

**Causes**
- primarily alcohol (usual onset approx 8-10 years after commencement of drinking)

Less common causes:
- familial
- CF
- obstructive cause

**Symptoms**
- persistent intense abdo / back pain
- nausea/ vomiting
- + steatorrhoea
- + impaired glucose tolerance

**Diagnosis:**
- presence of steatorrhoea
- ultrasound

### Chronic pancreatitis: nutritional management

- main strategy is conservative, symptom-related treatment (surgery is only used if there is obstruction, fistula or pseudocyst).
- small, frequent meals with high energy and protein, supplements if needed, correct any deficiencies
- avoiding alcohol is essential
- fat as tolerated:
  - may need enzyme replacement to prevent malabsorption
  - may need low fat diet +/- MCT oil
- diabetic diet if appropriate

### ACUTE Pancreatitis

**Causes = “get smashed”**
- Gallstones
- Ethanol
- Trauma
- Steroids
- Mumps
- Auto-immune cause
- Scorpion bite
- Hyperlipidaemia
- ERCP
- Drugs

**Symptoms**
- vomiting, nausea
- severe abdo / back pain
- fever
- jaundice
- abdo distension

**Diagnosis:**
- serum amylase & clinical symptoms.
- severity via CRP and Glasgow/APACHE score

### Acute Pancreatitis: nutritional consequences

**What would you expect some of the nutritional effects to be?**
- poor intake due to nausea
- increased requirements due to inflammatory process and hypermetabolism
- maldigestion and consequent malabsorption
- nutritional deficiencies due to malabsorption of vitamins/minerals
- raised BSLs
- dehydration

### Acute Pancreatitis: medical management

- pancreatic enzyme replacement therapy (“PERT”).
  - dose relates to fat content of meal but does not correlate with usual CF guidelines
  - dose is taken at start of / during meal
- oral hypoglycaemic agents or insulin
- antibiotics (?)
- surgery: pancreatic resection

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Pancreatic resection

• removal of necrotic or malignant section of pancreas (for chronic pancreatitis or cancer)
• total pancreatectomy: become Type 1 Diabetic + require pancreatic enzyme replacement
• partial pancreatectomy: variable effect but normal function can be maintained with just 10% of the pancreas remaining.
• very poor prognosis post-op for all (prognosis for pancreatic cancer at five years is 4%, which increases to 10-20% with surgery)

Whipple’s Procedure (Pancreatoduodenectomy)

Pancreatitic resection: nutritional management

• ideally a jejunal feeding tube is placed during surgery (use a standard feeding formula unless malabsorbing)
• progress to small, frequent meals
• risk of dumping syndrome
• may lose substantial pancreatic function and require a diabetic diet (+/- pancreatic enzyme replacement) with high protein and energy as substantial weight loss is common

Tricky Questions….

• TIPS
  – reassure yourself that patients are used to answering these questions, as doctors ask them all the time.
  – the conversation will only be awkward if you feel awkward. These questions should be asked like any other questions.
  – try to avoid nervously rushing over the topic – there is no use asking the question if you do not get the useful information you need.
  – don’t push the issue if the patient does not want to discuss it – you can always raise the topic again at another visit when rapport is better.
  – dietitians are not drug & alcohol counsellors
  – be sensitive and understanding

Anatomy of the gall bladder

• What is the role of the gall bladder? It stores bile made by the liver. Also concentrates the bile by removing water.
• What can go wrong?
  – cholelithiasis
  – cholecystitis
• What are the nutritional consequences?

GALL BLADDER DISORDERS

most common in patients with “the five Fs” (female, fat, fertile, fair/caucasian, 40s+)

CHOLELITHIASIS
= gallstones in gall bladder
Stones consist of cholesterol, bilirubin, calcium salts
Caused by:
• chronic inflammation
• change in bile causing decrease in cholesterol solubility
May be asymptomatic.

CHOLECYSTITIS
= inflammation of gall bladder
Caused by:
• gall stones
• infection
• drugs
Diagnosis
• ultrasound
Cholecystitis: medical management

CHOLECYSTITIS
- antibiotics
- lithotripsy (shock-wave blasting of stones)
- surgery:
  - ERCP (keyhole removal of stones)
  - cholecystectomy (removal of gall bladder)

Cholecystitis: nutritional management
- symptom control (before and after surgery) with reduced fat diet and attention to type of fat, spread through day (e.g., fried foods often a problem)
- soluble fibre helps remove cholesterol
- may need nutrition support
- may need weight loss advice
- general healthy diet

Chyle leaks
- Long Chain fats are absorbed from the gut and transported in the lymphatic system as chyle. Chyle contains approx 200cal/l; 30g/l protein; 4-40g/l lipid.
- accidental damage to the lymph system is most common in surgery to head/neck and thorax (e.g., upper GI or liver surgery, vascular surgery, lymphoma)
- can result in chylothorax, chylous ascites, chylopericardium or chyle coming out an external drain.
- many ‘inappropriate’ diets historically used in the treatment of chyle leaks

Nutritional management of chyle leaks
- No clear treatment guidelines but patients have high protein and energy requirements:
  - very low fat diet (<20 gm/day)
  - with fat free oral supplements
  - very low fat enteral feed/MCT feed
  - TPN (no fat restriction necessary)
  - MCT supplementation
- a chyle leak can take days to months to heal. Surgery is an option if conservative management fails to heal the leak.

Further reading:

The lower GI tract
- What is the role of the lower GI tract?
  - it is a tube about 4m long, responsible for digestion and absorption of nutrients. It also has a major role in the whole body’s immune function.
- What can go wrong?
  - radiation enteritis
  - inflammatory bowel disease (IBD)
  - irritable bowel syndrome (IBS)
  - diverticular disease
  - bowel obstruction
  - short bowel syndrome
- What are the nutritional consequences?

The gut is the centre of the immune system
- the gut surface area is ~300m²
  - a large area to protect!
The gut is the centre of the immune system

- the gut must coordinate complex traffic:
  - allow nutrients IN
  - keep harmful substances (such as toxins and bacteria) OUT
  - allow adhesion by “good” bacteria
- this demands an effective immune capability

Lymphocytes can be:
- B-lymphocytes, which mature to become plasma cells that produce antibodies (humoral immunity)
  - or
- T-lymphocytes, which produce cytokines and are responsible for cell-mediated immunity
  (T-cells destroy infected cells and also recruit and activate macrophages)

Peyer’s Patches are areas of lymphoid tissue in the ileocaecal region of the gut. This area contains similar bacteria (but in smaller numbers) to those in the colon.

- in the Peyer’s Patches, antigen is presented to lymphocytes (B and T) underneath the microvillus cells

- the majority (~80%) of our antibodies are produced in the gut (mostly IgA)
- this is the main type of antibody found in external body secretions (eg saliva, mucus, tears, milk etc)

the gut generates the entire body’s mucosal immune system
("Common Mucosal Immune Hypothesis")

This mucosal immune system can deteriorate if:
- bacteria overgrow the gut (eg after antibiotics)
- no nutrition is received in the gut (eg NBM, TPN – traditional management for gut patients!)
- complex nutrition is not received in the gut (eg no fibre; elemental formula or clear fluids for a long period)

Evidence-based approach emphasises early enteral nutrition, even if it is limited to a small amount and supplemented with TPN to meet the patient’s needs.
Disorders of the lower GI tract

Problems with inflammation:

- radiation enteritis
- inflammatory bowel disease

What is inflammation?

---

Radiation enteritis

What is it?
Radiation damage to the small bowel. Also called radiation enteropathy. Inflammation and swelling of the bowel lining, interferes with absorption. Usually temporary, but sometimes damage is permanent.

How is it diagnosed?
Any diarrhoea experienced during or after radiation therapy to the abdomen (such as for gut or cervical cancer). Sometimes the radiation therapy can be adjusted or postponed to allow gut to recover.

How is it managed?
- monitor absorption, with food as tolerated (may need low lactose, reduced fibre?elemental formula may be better absorbed)
- consider bile salt malabsorption (may need bile salt binder +/- low fat diet)
- TPN may be required to supplement enteral intake or to provide full needs if extensive damage / malabsorption
- mild damage may recover more quickly if there is some enteral nutrition

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Inflammatory bowel disease

What is it?
Chronic inflammation of the bowel

How is it diagnosed?
Abdo x-ray. Then may use endoscopy if IV steroids do not produce a reasonable improvement within 48 hours. Faecal cultures are performed to exclude an infective cause of the symptoms.

---

Two types of IBD

ULCERATIVE COLITIS
- affects large bowel and/or rectum only
- inner lining only (mucosa and submucosa)

CROHN'S DISEASE
- can affect any part of gut, from mouth to anus
- full thickness of gut wall (so deep fissures and ulceration can occur, which may develop into fistulas and strictures)

---

Ulcerative colitis

![Diagram of ulcerated colon](image_url)
Inflammatory bowel disease

What happens?

**Pattern of remissions and flare-ups**

During active disease:
- abdominal pain
- fever, nausea, vomiting
- diarrhoea (blood & mucus)
- tiredness & anaemia
- weight loss & malnutrition

Factors affecting nutritional impact:
- duration of flare-up
- severity of disease activity
("severe"= diarrhoea > 6/day plus one of: fever, bloody stool, anaemia, tachycardia)

Severe IBD formerly had a mortality rate of 30-60% (now <2% due to steroids and early colectomy for UC)

Causes of malnutrition:
- inadequate oral intake (due to pain, anxiety, food avoidances)
- malabsorption (due to gut malfunction, bacterial overgrowth, some medications)
- increased nutritional requirements (for healing, and due to inflammation)
- increased nutrient losses in tissue (protein, iron, calcium, zinc)
Inflammatory bowel disease

What happens?

Other common nutritional issues:

- psychosocial food issues (disordered eating, food avoidances, fear of eating)
- anaemia (due to deficiencies in iron, folate, B12)
- osteoporosis – very common (due to calcium and Vit D malabsorption, and longterm use of corticosteroids)
- impaired glucose tolerance and diabetes (often steroid-induced)

Effects of malnutrition:

- increased mortality and morbidity
- decreased immune function
- impaired healing
- ongoing poor gut function (even in parts of the gut not directly affected by disease)
- increased risk of high homocysteine levels
- increased risk of gut cancers

How is it managed?

maintenance

- normal balanced diet, maintaining healthy weight:
  - discourage unnecessarily restricted diet
  - restore any deficiencies (eg calcium, VitD, iron, zinc)
  - supplement protein and energy as needed
during a flare-up

- oral/enteral as tolerated, maintain hydration
- no evidence that “bowel rest” helps the gut heal
- tolerance to certain foods may be diminished (eg fat, lactose, fibre)
- may need tube feeding to maintain nutritional status

enteral nutrition

- controversial whether can be used instead of steroids in a flare-up

omega-3 fatty acids

- unclear extent of benefit, or dose needed (↑↑↑?)

glutamine

- amino acid used by gut, unclear benefit

probiotics

- improvement in gut flora, immune function

worms

Irritable bowel syndrome

What is it?

A group of intestinal dysmotility symptoms: often diarrhoea alternating with constipation, abdominal pain, bloating, belching, heartburn, nausea. May be associated with headache, fatigue, low blood pressure.

How is it diagnosed?

With difficulty! Physical examination is usually normal. It is often a “last resort” diagnosis after other conditions are excluded, or if stress or psych issues are present.
**Irritable bowel syndrome**

What causes it?

**Complex and multifactorial aetiology:**
- abnormal (constant) vagus stimulation? Often a significant psychological component. (often associated with anxiety/depression/stress)
- after-effect of a gut infection (gut flora problem?) (= “postinfective IBS”, may be up to 25% of cases)
- genetic factors (twice as common in women; familial tendency)
- gut sensitivity / interpretation of gut signals?

How is it managed?

- exclude other possible diagnoses
- define the problem clearly (eg use stool chart)
- control symptoms
- provide individualised dietary advice (reduce abnormal eating practices)
- provide emotional support (counselling and relaxation may help)
- address lifestyle factors (exercise improves blood flow to gut, assists normal gut motility and improves mental health)

**Identify dietary triggers:**
- alcohol
- caffeine-containing beverages
- lactose
- high-fibre foods
- high-fat foods
- sorbitol

**Diverticular disease**

What is it?

**DIVERTICULOSIS (convalescent state)**
= disease where small pouches (diverticula) form at weak areas in the muscles of colon wall

**DIVERTICULITIS (inflamed state)**
= inflammation of the diverticula
  - when bacteria & colonic contents become trapped in pouches

How is it diagnosed?

Colonoscopy
### Symptoms

<table>
<thead>
<tr>
<th>DIVERTICULOSIS (convalescent state):</th>
</tr>
</thead>
<tbody>
<tr>
<td>• chronic constipation or asymptomatic</td>
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<table>
<thead>
<tr>
<th>DIVERTICULITIS (inflamed state):</th>
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<tbody>
<tr>
<td>• spasm &amp; crampy stomach pain</td>
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<tr>
<td>• distension in lower abdomen</td>
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<tr>
<td>• fever</td>
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<tr>
<td>• diarrhoea with blood &amp; mucous</td>
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<tr>
<td>• ± nausea &amp; vomiting</td>
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### Diverticular disease

**What causes it?**
- More common in countries with lower fibre intake; insoluble fibre may protect against diverticulosis
- Theory is that colonic wall strength decreases with age
- Higher fibre intake creates a softer more bulky stool with shorter transit time, that places less pressure on colon wall

### How is it managed?

**DIVERTICULOSIS (convalescent state):**
- high fibre diet prevents further diverticula formation and reduces symptoms
- need adequate fluid and activity

**DIVERTICULITIS (inflamed state):**
- clear fluids diet until symptoms reduce
- if this is for extended period, need nutritionally complete supplements
- gradually increase fibre back to a high-fibre diet

### Bowel obstruction

**What is it?**
Partial or complete blockage of the gut. May be due to adhesions or tumour.

**How is it diagnosed?**
Usually by xray or CT scan after symptoms are noted (distended abdomen, abdominal pain, no bowel output, or thin watery “overflow” diarrhoea; sometimes faecal vomiting)

**How is it managed?**
“Conservative” management (eg for adhesions or ileus): wait for blockage to dislodge / normal motility to resume. May be NBM, or on low residue diet, or just avoid fibrous foods.

Surgical management (for tumour, strangulating adhesions, necrotic gut): resection of the blockage part.

### Pseudo-obstruction or ileus

**What is it?**
Loss of peristalsis in part of the gut (so that it behaves as if blocked). May be due to necrotic (dead) bowel or just inflammation interfering with normal motility.

**How is it diagnosed?**
Usually by xray - symptoms are same as for obstruction, but xray shows a buildup of gut contents without any blockage
**Bowel resection**

**What is it?**
Removal of a damaged or non-functioning part of the bowel. May be:
- bowel obstruction (from cancer, adhesions, congenital defect)
- severe IBD (Crohn’s Disease or Ulcerative Colitis)
- bowel perforation, abscess or fistula
- bowel necrosis / irreversible trauma

**Stoma formation**

**What is it?**
Intestine is brought up to abdo wall and a hole is formed to the outside, through the skin. May be:
- colostomy (hole from colon to outside)
- ileostomy (hole from ileum to outside)

**Stoma formation**

**What is it?**
Intestine is brought up to abdo wall and a hole is formed to the outside, through the skin. May be:
- temporary (with plan to rejoin the gut ends later), or
- permanent

**Stoma formation**

**How is it managed?**
Patient wears a bag to collect stoma output.
- colostomy: output like a normal stool (runny if large amount of colon removed); normal smell
- ileostomy: output is pasty (looks like watery peanut butter); less smell

**Stoma formation**

**How is it managed?**
- a variety of stoma devices is available
- inner face of bag adheres firmly to skin, with a hole fitting around stoma
- bag may be a sealed or openable (drainable) type
- sizes vary
- gas venting system
Stoma formation
How is it managed?
Nutrition care aims to:
• help patient return to a normal diet
• ensure adequate fluid and electrolyte replacement
• avoid stoma blockages
• manage output texture
• minimise gas and odours
• provide psychosocial support as needed

Stoma management
Helping patient return to a normal diet:
• Early postoperative nutrition improves outcomes
  (high risk of malnutrition postoperatively, nutrition support may be needed initially post-op)
• Initially ileostomy output is often watery and volumes are large (stabilises with eating)
• Rapid progression to normal diet as tolerated
• In first 6-8 weeks the area is oedematous and more prone to blocking. Diet should avoid stringy/membranous foods but still be varied and well-balanced.

Stoma management
Ensuring adequate fluid / electrolytes:
• ileostomy and high colostomy
  • large fluid losses (due to absence of colon) increases risk of dehydration
  • normal output 600-800mL/day (expect much more initially postop)
  • need additional fluids to replace losses (2-2.5L/day) avoiding diuretic drinks
  • extra fluids in hot weather
  • individualised electrolyte supplementation depending on intake and absorption

Stoma management
Avoiding stoma blockages:
• blockages occur when undigested food remnants are trapped in the narrow abdominal opening
• initially avoid foods high in insoluble fibre
• after 6-8 weeks, diet can be liberalised and higher-fibre foods reintroduced
• chew all foods well

Stoma management
Managing output texture:
• output >800mL is usually watery and causes fluid and electrolyte loss
• thickening may reduce volume
• the following may help:
  - probiotics
  - soluble fibre, pectin, guar
  - resistant starch
  • do not restrict fluid

Stoma management
Managing gas output:
• sound may be embarrassing
• stoma bag can become distended
• gas increased by swallowing air
  (avoid fizzy drinks, smoking, chewing gum etc)
• gas produced by fermentation of dietary fibre, starches and sugars in the large intestine
• limiting certain foods may help minimise gas
  (eg lactose, legumes, green veg, cucumber etc) but should only be avoided on an individual basis
Stoma management

Managing odour:
• may be embarrassing
• odour exacerbated by steatorrhoea or bacterial activity as well as odorous food components
• limiting certain foods may help minimise odour
  (eg legumes, spices, fish, cheese, egg, odorous veg, antibiotics, vitamin supplements etc) but
  should only be avoided on an individual basis
• lactobacillus probiotics may help, also parsley, chlorophyll tablets, cranberry juice

Short bowel syndrome

What is it?
A group of symptoms that may follow extensive resection of the gut (particularly when small bowel is involved). Can occur with anastomosis or stoma formation.

• fluid and electrolyte losses
• nutrient malabsorption(s)
• difficulty maintaining weight
• occurs due to loss of absorptive gut surface

Short bowel syndrome

What is it?
A group of symptoms that may follow extensive resection of the gut.

Severity of condition depends on:
• site of resection
• length of remaining bowel
• if large bowel involved
• if ileocecal valve present
• time lapsed since resection done
• if any remaining disease

Colon functions: absorbs fluid and sodium, harbours flora, stores faeces (slowing intestinal transit)

Effect of colonic resection:
• depleted fluid and sodium, risk of dehydration (due to reduced resorption; more watery stool)
• ileocaecal valve and surrounding tissue is an important means of slowing faecal speed and preventing colon bacteria entering small bowel (if removed, increased risk of malabsorption due to faster faecal speed, risk of bacterial overgrowth)

Jejunal functions: absorb monosaccharides, amino acids, monoglycerides and fatty acids, fat-soluble vitamins, iron, calcium, water

Effect of jejunal resection:
• usually well-tolerated if remaining bowel function is normal
• jejunum functions can be taken over by ileum (but not vice versa)

Ileum functions: absorbs fat, fat-soluble vitamins, bile salts (distal 100cm is the most critical section), vitamin B12, sodium, water

Effect of ileal resection:
• loss of bile salts
• loss of fat (if >100cm resected) and fat-soluble vitamins
• loss of positive ions such as calcium, magnesium, zinc, copper, selenium (due to soap formation with bile salts)
• if still have colon, oxalates are reabsorbed and build up in kidneys as stones
Short bowel syndrome

Duodenum functions: absorb minerals, site for digestion

Effect of duodenum resection:
• dumping syndrome with sugars
• malabsorption of lactose, calcium, iron, folate

How is it diagnosed?
May be identified by:
• assumption based on length of bowel resected
• malabsorption symptoms postoperatively
• difficulty in maintaining fluid and electrolyte status (may take longer to show up)

How is it managed?
Immediately post-op:
• TPN +/- small oral intake
• IV fluids +/- electrolytes as needed to replace losses
• transition to enteral/oral feeds with supplements as needed (may also need IV fluid/electrolytes and/or supplemental TPN). Polymeric diet (vs elemental) encourages gut adaptation.
• monitor gut adaptation

Malabsorption:
• fluid and electrolytes (esp if colon)
• increased intake and/or IV supplementation
• fat (esp if distal ileum)
• may need low fat diet, fat-sol vit supps, +/- MCT oils
• CHO (esp if jejunum)
• may need low lactose diet, avoiding concentrated sugars or hyperosmolar foods/fluids
• fibre
• low-insoluble fibre diet, with more soluble fibre, may be better tolerated

Long-term adaptation and recovery:
The bowel can adapt to resection.
• absorptive capacity of the bowel continues to improve for up to 1-2 years post-resection
• may be enhanced by presence of short chain fatty acids (from fibre) and other nutrients
• unclear whether glutamine or growth hormone can help
• bowel transplantation is another area of research
• ongoing nutritional monitoring is needed
• if <30-60cm small bowel remaining, may need lifelong TPN