CONSTIPATION IN PALLIATIVE CARE

MANDY TIMMS
ADVANCED NURSE PRACTITIONER
PILGRIMS HOSPICE

Aims of the session

- Fact or fiction
- Normal physiology
- Prevalence
- Definition
- Measuring Constipation
- Causes
- Assessment
- Treatments and the evidence

Faecal Composition

- Water (75%)

- Remainder: 1/3 dead bacteria, 1/3 residue (fibre), balance: sloughed cells from intestine, bilirubin, fats, salts

Constipation Prevalence

-General population **10%**

-Cancer population **30 – 50%**

-Palliative Care population **Up to 90%**

-Patients on opioids **50 – 100%**

Droney J etal. 2008. Supp Care Cancer
Potter J et al. 2003 Palliative Medicine
Riechelmann RP et al. 2007 Supp Care Cancer
Kurz A et al. 2003 Drugs

A lot of palliative care patients think they are constipated ???

- In a survey of 93 hospice cancer patients 46 (**49%**) said they were currently constipated, but 25 of the 46 (**54%**) had had a bowel movement either on the day of questioning or the day before

(Sykes, 1998)

DEFINING CONSTIPATION

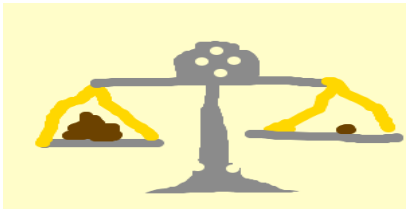
- **Infrequent**, **difficult** or **Incomplete** bowel evacuation that may lead to pain and discomfort

- Stools that can range from **small**, **hard** 'rocks', to a **large bulky mass**

- A **sensation** of incomplete evacuation

Compared with symptoms such as pain, constipation has a raft of **measurable features**

- How these features are combined and weighted to constitute a sense of constipation is very individual



- The range of normality is very wide – e.g. Bowel frequency in a healthy population varies from **3 to 21** per week

CONSTIPATION CRITERIA

The **Rome (III)** criteria for constipation is often cited, but relate to functional constipation:

• The presence of **two or more** of the following symptoms for at least **three months**, with symptom onset at least six months ago: – **<3 bowel movements per week** – Straining at least **25%** of the time – Hard stools at least **25%** of the time – Incomplete evacuation at least **25%** of the time – Manual manoeuvres needed at least **25%** of the time

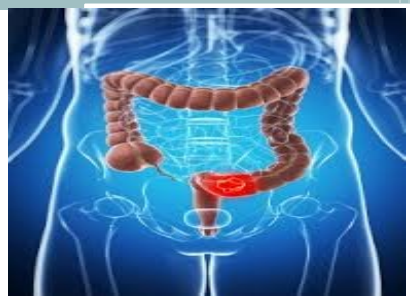
(Longstreth et al. 2006 Gastroenterology)

Bristol stool chart

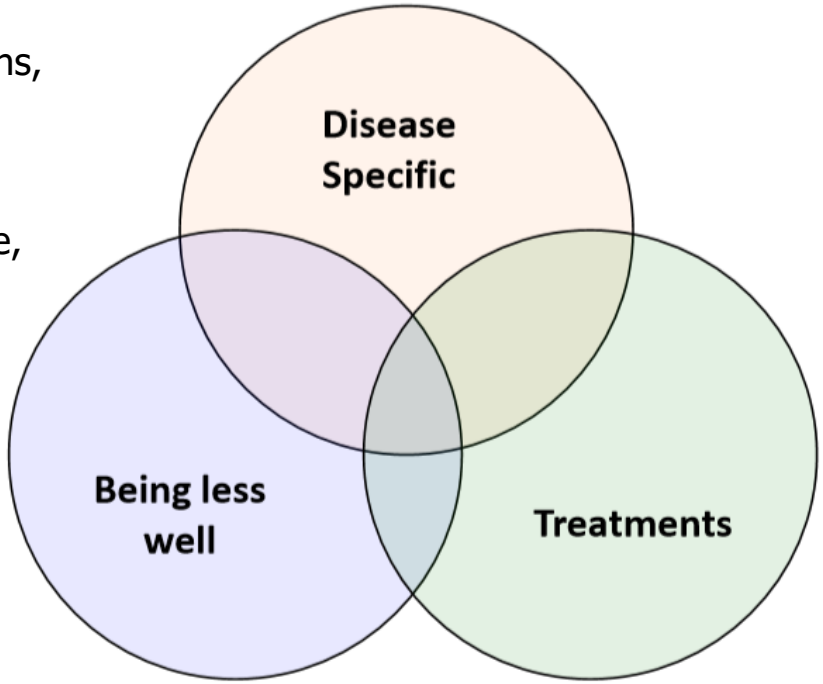
	Type 1 Separate hard lumps, like nuts (hard to pass)
	Type 2 Sausage-shaped, but lumpy
	Type 3 Sausage-shaped, but with cracks on surface
	Type 4 Sausage or snake like, smooth and soft
	Type 5 Soft blobs with clear-cut edges (easy to pass)
	Type 6 Fluffy pieces with ragged edges, mushy
	Type 7 Watery, no solid pieces (entirely liquid)


Why?

Causes



Age: Loss of enteric neurons, affected rectal sensitivity, impaired colonic mass movement, reduced fluid intake, reduced fibre intake, can't raise intra-abdominal pressure due to weakness



Invasion of bowel musculature, invasion of nerves, spinal lesions
Biochemical - Calcium / potassium - Neurotransmitters / cytokines / Hormones (T3/T4)
Disease related symptoms: - Pain / Vomiting

- Opioids
- Anti-cholinergics -TCA's -Anti-histamines -Neuroleptics
- Chemotherapy -Platinum based -Vinca Alkaloids e.g. vincristine
- Anti-emetics … 5HT3 antagonists

Assessment

History

- **When**: did they last have their B.O. ? Flatus?
- **What**: are their stools like? Hard or Soft? PR?
- **How**: often do they usually go? i.e. what is their 'normal' bowel habit
- **Why**: are they constipated? i.e. Treat reversible causes (e.g. ? Drugs/ biochemistry/ environment)

Managing Constipation

Non-Pharmacological / prophylaxis:

- Stay well hydrated

- Increase mobility **but** no consistent link demonstrated (Fallon and Hanks 1999, Bennett and Cresswell 2003)

- Increase fibre content **but** impossible for most palliative care patients to take enough (Mumford, 1986)

- Rationalise medications – stop/ reduce constipating meds

Evidence that prophylaxis is effective against constipation in palliative care is lacking

Do laxatives work?

- **Yes!** PHEW !!

- Laxatives are the most enduring of drugs – about half of the drugs that appear in the current BNF are laxatives and date back over 100 years

- Many things can act as laxatives if they either: – Stimulate intestinal contraction

- By distension

- By myenteric neural irritation – Soften the consistency of gut contents – (or act as a placebo)

Systematic reviews of laxative trials

Tramonte et al. 1997:

- **36** trials including 1,815 adult participants
- **All** classes of laxative included
- **Any** type of laxative increased bowel frequency by 1.5 (1.1-1.8) per week
- **No** agent clearly superior either in improving bowel frequency or stool consistency

Types of laxatives

- Stimulant

- Faecal softeners (and lubricants)

- Osmotic laxatives

- Bulk forming

Softening versus Stimulant laxatives

Inaccurate distinction because:

- Any softening of the stool entails an increase in volume that will stretch the bowel wall and stimulate reflex contraction

- Any stimulation of motility that accelerates transit will reduce time for absorption of water and lead to the stool being softer

Classified as per their primary action but many have a secondary action …

-**Lactulose**: = (i) osmotic (ii) also fermented into organic acids which irritate i.e. stimulate the large bowel

-**Docusate**: (i) low dose = softener (ii) higher doses stimulant

Stimulants

Acting on large bowel only

• Senna (inactive until hydrolysed by bacterial glycosidases into **irritant** active metabolites)

• Sodium picosulphate (hydrolysed by colonic bacteria to an active **stimulant** compound)

Acting on small and large bowel

• Bisacodyl (hydrolysed by intestinal enzymes to an active **stimulant** compound)

• Dantron (in co-danthramer and co-danthusate)
Direct Contact with the submucosal (Meissner's) and deeper myenteric (Auerbachs's) plexus = increases **propulsion**

Faecal softeners (and lubricants)

Lowers faecal surface tension allowing water and fats to allow penetration of dry hard faeces

Surface wetting agents

- Na Docusate

- Poloxamer 188 (in co-danthramer)

Lubricants

- Paraffin

- Arachis oil

Stimulant & softener Combo

- **Co-danthramer** (Danthron + Poloxamer 188)
- **Co-danthrusate**(Danthron + Docusate)

- Both potentially **CARCINOGENIC** (rodent models –Mori et al 1985-86. Committee on safety of medicine and Medicines control agency 2000)

- Reserved for **terminal care**

- Colours urine **red**

- Severe Danthron **burns** -> severe excoriation esp if any chance of faecal incontinence

Osmotic laxatives

Encourage water into GI tract increasing faecal volume and softening

Polyethylene Glycols

•**Macrogols (Movicol, Laxido) –**Unchanged in GI tract

(i) Need to be swallowed with enough water to make osmotically active. (ii) No point using if dehydrated

Hyperosmolar laxatives

•**Lactulose –**disaccharide**–**draws water into small intestine. Fermented in large bowel to acetic/ lactic/ formic acids= stimulant

(sweet, crampy, gas forming)

•Magnesium salts

Bulk Forming (fibre)

Increases faecal mass, some fermentation stimulant effect

- Isaghula (Fybogel)

- Sterculia (Normacol)

Laxatives for the management of constipation in people receiving palliative care (Review)

Candy B, Jones L, Larkin PJ, Vickerstaff V, Tookman A, Stone P

THE COCHRANE COLLABORATION®

The key to effectiveness is good assessment and using what we have fully

- Even in palliative care or cancer units **A third of patients reporting bowel problems at admission were not given laxatives** (Goodman, Wilkinson and Fellowes, 2001)

- **89% of constipated patients were on inadequate doses of laxatives** (Droney et al., 2008)

- We know that **enforcing dose titration** of laxatives can reduce enema and suppository use by 20% (Sykes, 1991)

- There is a **palatability** problem (Morrison and Pirello, 2011)

- Lack of clear differences in efficacy imply that ***acceptability*** and ***cost*** should guide laxative choice (NHS Centre for Reviews and Dissemination, 2001)

BOTTOMS UP

A third of patients will need rectal measures having failed on oral laxatives (Twycross et al. 1991, 1997)

Anal Retention

PR – empty/ soft / hard

• Suppositories – direct contact with rectal mucosa - Bisacodyl (stimulates propulsion locally) - Glycerol (Lubricant / osmotic)

• Micro-enemas - Docusate (softens stool) – Osmotic

• Enemas – 120ml volume (Hard impaction) - Docusate /Arachis [NB **Nuts**] (softens faeces – retain overnight) followed by stimulant suppositories morning after or osmotic enema - Phosphate enema

Conclusions

- Constipation exists in palliative care and we can **assess** it – but it's a symptom, not a disease

- **Prophylaxis** is OK in theory but there is no evidence of effectiveness in practice

- No laxative works if you don't **give enough** of it

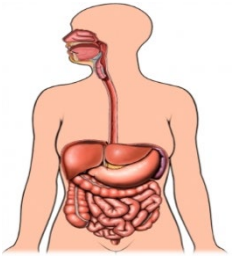
- All laxatives work if you give enough **but** doses, adverse effects and palatability vary both by agent and by patient

- Pragmatically best results may come from both **stimulating** motility and **softening** gut contents (No clear evidence)

THANK YOU

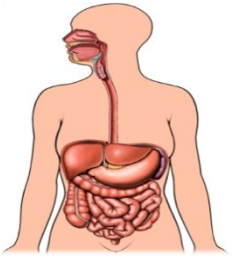
THE MANAGEMENT OF MALIGNANT BOWEL OBSTRUCTION

MANDY TIMMS
ADVANCED NURSE PRACTITIONER
PILGRIMS HOSPICE



AIMS AND OBJECTIVES

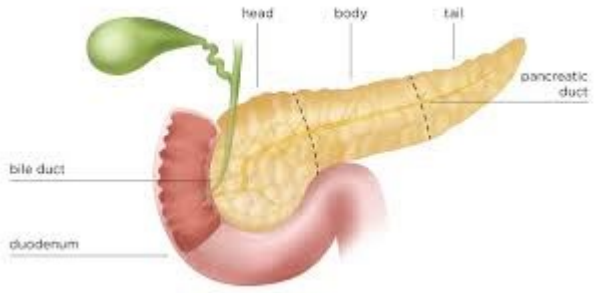
- **IDENTIFYING THOSE AT RISK**
- **RECOGNIZE SIGNS AND SYMPTOMS**
- **NON PHARMALOGICAL MANAGEMENT**
- **PHARMACOLOGICAL MANAGEMENT**
- **PSYCHO-SOCIAL CONSEQUENCIES**



INCIDENCE

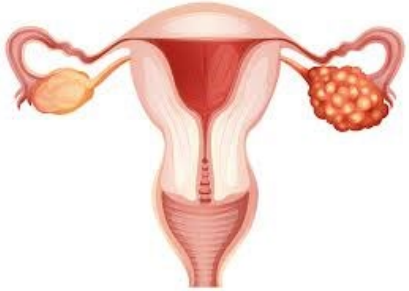
- Malignant bowel obstruction (MBO) is a frequent complication in advanced cancer patients, especially in those with **abdominal** tumours.
- Bowel obstruction occurs when there is **blockage** of the forward flow of gastric and intestinal contents through the gastrointestinal tract (Letizia and Norton 2003).
- The global prevalence of MBO is estimated to be **3% to 15%** of cancer patients (Tuca et al 2012).
- MBO occurs in 5% to 51% of women with **ovarian cancer** and in 10% to 28% of patients with **gastrointestinal** cancer, predominantly in the advanced stages (Mercadante 2009).
- Median survival after its onset ranges from **30 to 90 days** (Laval et al 2006).
- Lung and breast malignancies and melanoma can cause obstruction due to **metastatic spread** (Ripamonti and Mercadante 2004).

- Cholangiocarcinoma and pancreatic cancer are the most common tumours causing duodenal obstruction (Soriano and Davis 2010)



- Those with primary abdominal and pelvic malignancies can become obstructed because of post-radiation fibrosis, growth of the primary tumour, and metastatic disease (Letizia and Norton 2003).

- With ovarian cancer, tumour cells spread by peritoneal seeding and can result in multiple sites of obstruction (Rawlinson 2001).

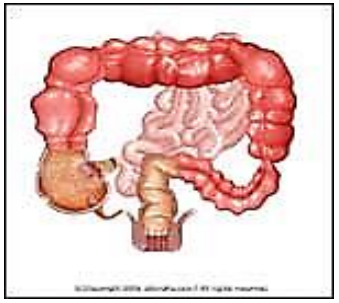


- At the end of life, many patients with MBO are not candidates for surgical intervention.

- **Patients require thorough assessment and the goal of care is to providing comfort and managing symptoms.**

CAUSES

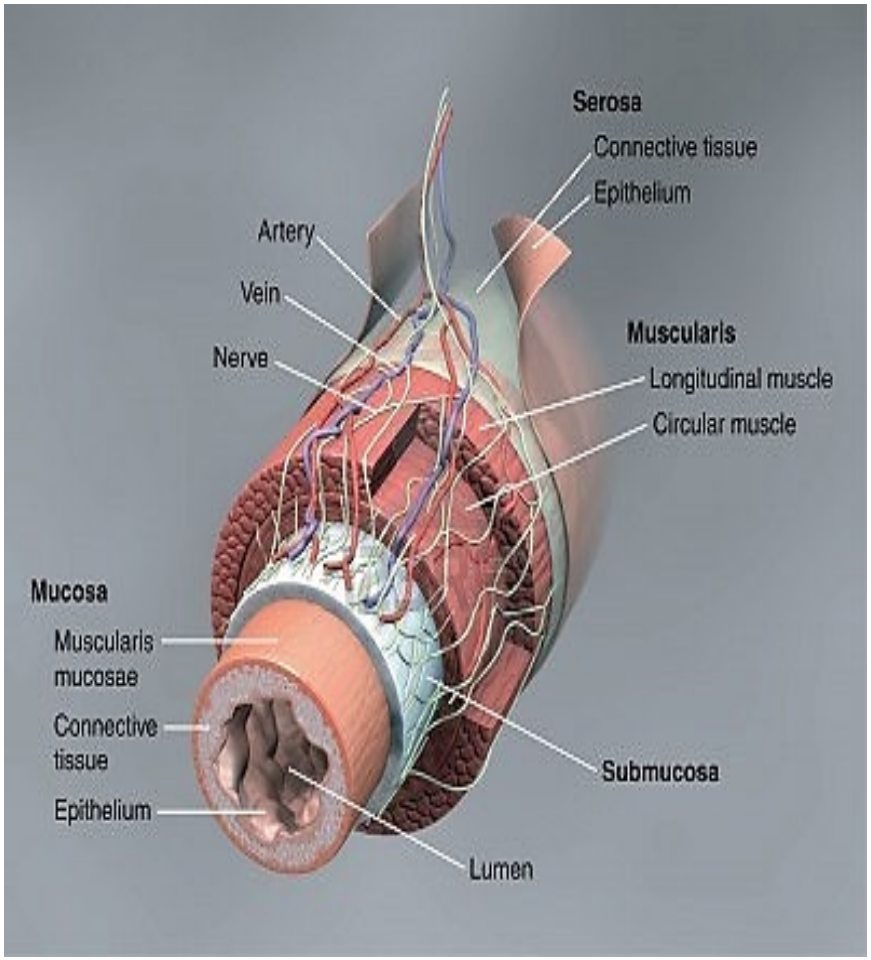
- Primary/secondary tumours large enough to occlude the lumen.
- Tumour spread within the muscle of the bowel wall, narrowing the lumen.
- Extrinsic compression of the bowel by mesenteric and omental masses, and adhesions.
- Intestinal motility disorders.
- Radiotherapy
- Constipation and faecal impaction.



TYPES OF OBSTRUCTION

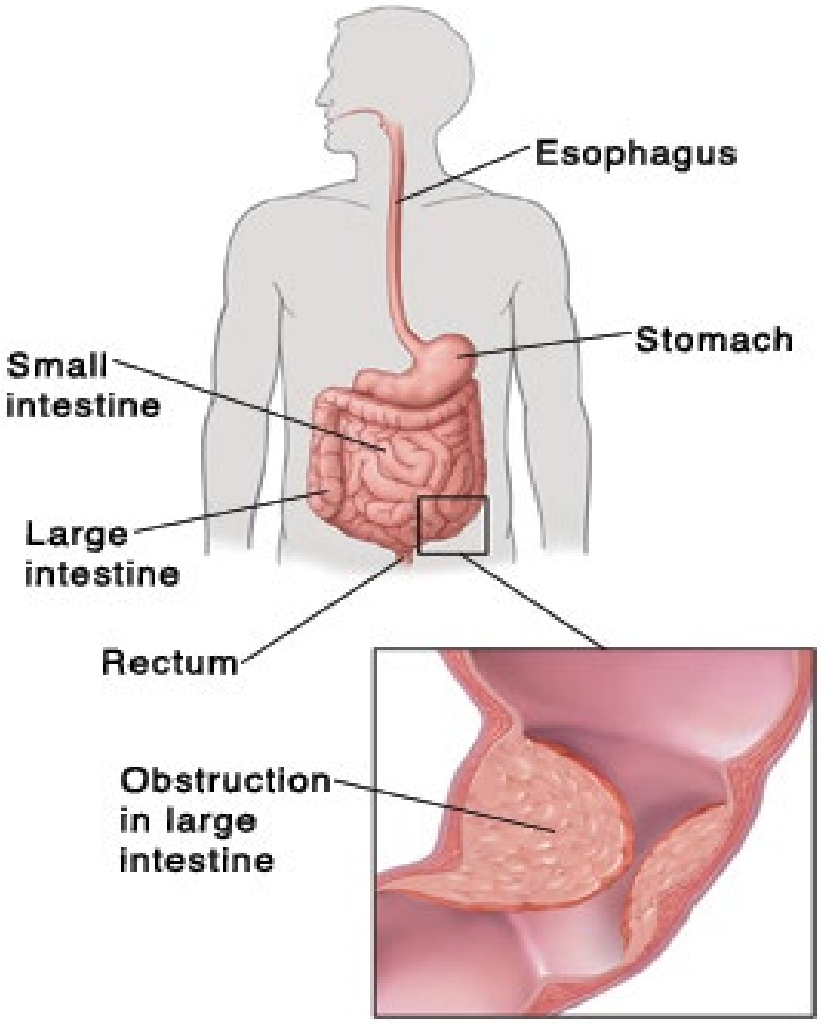
- Bowel obstruction can be mechanical or functional, partial or complete, and may occur at one site or at multiple levels (Roeland and Von Gunten 2009) .
- Intraluminal tumours can occlude the lumen or act as a point of intussusception.
- Intramural tumours extend to the mucosa and obstruct the lumen or impair peristalsis.
- Mesenteric and omental masses or malignant adhesions can kink the bowel, creating an extramural obstruction.
- Tumours that infiltrate into the mesentery, bowel muscle, or the celiac plexus can cause dysmotility.

(Soriano and Davis 2010)



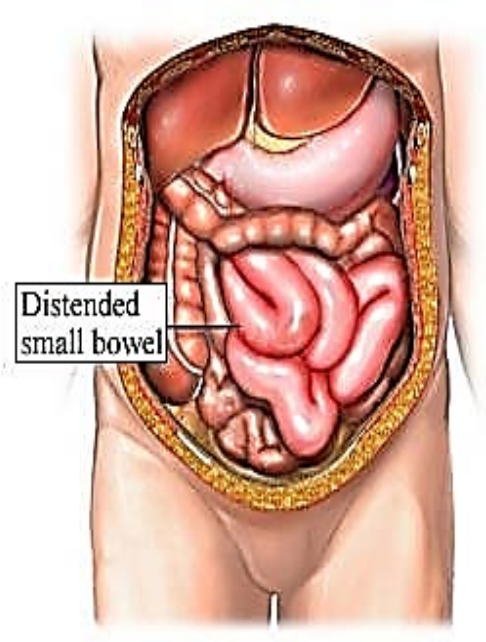
LARGE BOWEL OBSTRUCTION (LBO)

- Large bowel contents are semi solid to solid so moderate luminal narrowing may cause symptomatic obstruction (Casola and Sirlin 2005).
- LBO is usually caused by intrinsic factors (Szucs et al 2000).
- LBO tends to present more insidiously
- The major risk in LBO is perforation. This tends to occur at or adjacent to the cancer site (Casola and Sirlin 2005)



SMALL BOWEL OBSTRUCTION

- SBO at least 3 times more common than LBO (Brant 1999).
- SBO luminal obstruction is usually severe as the contents are liquid, mild or moderate narrowing may not cause symptoms (Kottler et al 2005).
- SBO is usually caused by extrinsic factors (Herlinger et al 2000).



SIGNS AND SYMPTOMS

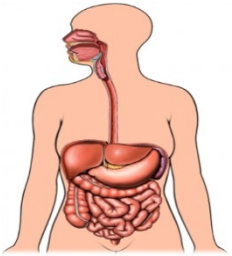
- Nausea and vomiting (68-100%)

- Increasing abdominal distension

- Abdominal pain (90%)

- Colic (75%)

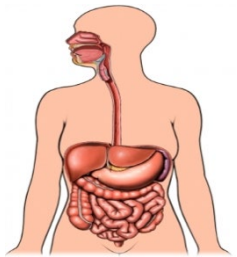
- Constipation



SYMPTOMS OF SMALL AND LARGE BOWEL OBSTRUCTION

- **SMALL BOWEL OBSTRUCTION**
- FLATUS AND FAECAL MATTER MAY CONTINUE TO BE PASSED, DIARRHOEA
- ABSENT OR DIMINISHED BOWEL SOUNDS
- INTRACTABLE/SUDDEN VOMITS OF ACIDIC BILE (NOT ALWAYS PRECEEDED BY NAUSEA)
- LARGE ODOURLESS VOMITS DEVELOP AT AN EARLY STAGE
- ABDOMINAL FULLNESS AND EXCESSIVE BELCHING
- PAIN AND CRAMPS IN EPIGASTRIC AND UMBILICAL AREAS

- **LARGE BOWEL OBSTRUCTION**
- ABSOLUTE CONSTIPATION
- HYPERACTIVE BOWEL SOUNDS/ HIGH PITCHED PROXIMAL TO LEVEL OF OBSTRUCTION. ABSENT OR DIMINISHED SOUNDS DISTAL TO OBSTRUCTION
- VOMIT CAN BE FOUL SMELLING AND FAECULANT OCCURING SEVERAL HOURS AFTER THE ONSET OF PAIN
- DIFFUSE ABDOMINAL PAIN TYPICALLY IN THE MIDDLE TO LOWER ABDOMEN
- LARGE ABDOMINAL DISTENSION
- OFTEN CONTINUOUS NAUSEA



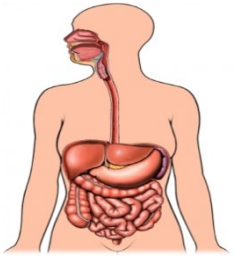
NON PHARMALOGICAL TREATMENT

Palliative surgery – de-bulking, resection, bypass and stoma formation

Stenting

Venting gastroscopy

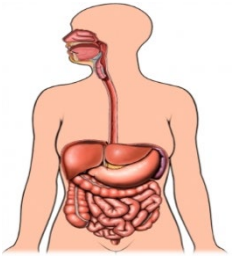
NG tubes



PHARMACOLOGICAL TREATMENT

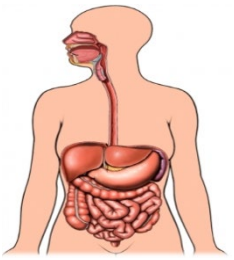
In MBO the body enters a vicious cycle of distension, secretion and increased contractility. This then results in nausea, vomiting and severe colicky pain.

Combinations of analgesics, anti-secretory drugs and anti-emetics alongside adjuvant treatments are used to provide symptom relief.



LAXATIVES

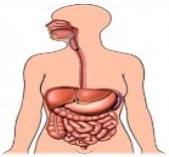
- Stop osmotic and stimulant laxatives i.e Lactulose, Senna, Danthron and Bisacodyl.
- Sodium Docusate is minimally stimulative and should be titrated to produce a comfortable stool without colic.
- Movicol (if volume of fluid is tolerated) is effective.
- Stop all oral laxatives in complete obstruction.



PAIN MANAGEMENT

- Patients with MBO can experience two types of abdominal pain: continuous pain and intermittent **colic**. Each type of pain requires different treatment approaches and medication.

- Potent opioids such as morphine, oxycodone and fentanyl are used to relieve continuous abdominal pain (Ripamonti et al 2008), the dose titrated for adequate relief



- However, opioids can aggravate colic by stimulating circular smooth muscle, leading to segmental contractions (Soriano and Davis 2010). Opioid-sparing adjuvant drugs such as ketorolac may improve colic and continuous pain and prevent a partial obstruction from becoming a complete obstruction by sparing opioid doses (Davis and Walsh 2000).

- Colic may persist or worsen with the use of opioids. Drugs that reduce colic include hyoscine butylbromide (buscopan), hyoscine hydrobromide and glycopyrronium bromide (Bicanovsky et al 2006).

ANTI EMETICS

- **Anticholinergics** reduce gastrointestinal secretions, fluid accumulation, and vomiting. Dosages: Hyoscine butylbromide 40 to 120 mg/day and Hyoscine hydrobromide 0.2 to 0.9 mg/day (Ripamonti et al 2008). Glycopyrronium Bromide has minimal central nervous system stimulation and is less likely to cause delirium, the recommended dose is up to 0.8mg (800mcg) subcutaneously daily (Davis and Furste 1999).

- **Metoclopramide** a dopaminergic antagonist which blocks D2 receptors in the central chemoreceptor trigger zone. Metoclopramide should not be used with anticholinergics (as they are competitively blocked by the latter) or in patients with colic or complete obstruction (Davis and Walsh 2000, Mercadante et al 2004). Metoclopramide should continue to be given if the patient continues to pass flatus and does not have colic. Metclopramide should be stopped immediately if colic develops. In some centres it is the first-line drug for partial bowel obstruction (Ripamonte et al 2008). Dosages range from 40 to 240 mg/day.

- **Cyclizine** is an Anticholinergic antiemetic and reduces peristalsis. Cyclizine blocks ACh and histamine H1 receptors in the vomiting centre that are triggered by the mechanoreceptors in the abdominal and pelvic viscera. Cyclizine is advocated as first line treatment for patients **with colic (**Mercadante 2007). It blocks the stimulation of the vomiting centre via the vagal afferents, which happens in complete obstruction. If this fails, change to levomepromazine. Doses range from 50-150mg s/c daily.

- **Levomepromazine** works by blocking a variety of receptors in the brain, particularly dopamine receptors. Doses from 5-25mg have been advocated (Glare 2007). Higher doses of levomepromazine can cause significant adverse effects (postural hypertension, dry mouth, sedation).

- **Haloperidol** a selective dopamine D2-receptor antagonist, is another option for the management of persistent vomiting or nausea in the **absence** of colic. At low doses it produces less sedation than phenothiazines (i.e Prochlorperazine) and is an ideal agent for patients with nausea and delirium (Davis and Walsh 2000). Doses range from 5 to 15 mg/day s/c, given in divided doses or as a continuous intravenous infusion. However Prommer (2012) reports that small doses – e.g. 2.5 mg once at bedtime by subcutaneous injection - are normally effective.

CORTICOSTEROIDS

- Dexamethasone has added value as an anti-inflammatory by decreasing gut wall oedema, thereby relieving some of the stenosis and decreasing the excretion of water into the lumen. It also has a central antiemetic effect (Glare 2004)

- Evidence from a meta-analysis found that 6 to 16 mg of parenteral dexamethasone per day reduced symptoms and improved bowel function in 60% of patients (Feuer and Broadley 2000).

- A trial of 4 or 5 days is adequate to determine response. If there is no response, the corticosteroid should be rapidly tapered (Soriano and Davis 2010).

OCTREOTIDE

- **Octreotide** reduces the excretion of water, sodium, and chloride into the bowel lumen and increases the absorption of electrolytes and water (Soriano A and Davis 2011). The result effect is reduced luminal content, reduced motility and reduced ascites (Ripamonti and Mercadante 2004).

- In small randomized trials, octreotide was more successful than anticholinergics at improving nausea, vomiting, and colic in patients requiring a nasogastric tube and in those whose symptoms were refractory to standard medical treatment (Laval et al 2006, Ripamonti et al 2000, Shima et al 2008). A recent case report found octreotide helpful in resolving symptoms of partial bowel obstruction that were unresponsive to standard measures (Myers et al 2010).

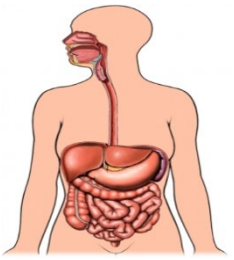
- Octreotide is well tolerated and reduces the time patients require a nasogastric tube (Ripamonte et al 2000) High cost limits its use as a first line treatment despite evidence of its efficacy.

- Octreotide doses of 300mcg – 900mcg/24 hours s/c have been advocated in MBO (Soriano A and Davis 2011).

COMBINATION THERAPY

Symptom control often involves the use of a combination of drugs. Antiemetics, analgesics, corticosteroids, antisecretory anticholinergics, and octreotide. These are often required in combination to achieve acceptable symptom relief (Pameijer et al 2005).

In a small prospective case series, the combination of metoclopramide 60 mg/day, octreotide 0.3 mg/day, and dexamethasone 12 mg/day improved intestinal transit within 1 to 5 days and resolved vomiting within 24 hours (Mercadante et al 2004).



AND WHAT ABOUT THE PSYCHO-SOCIAL ISSUES ?

REFERENCES

Bicanovsky LK, Lagman RL, Davis MP and Walsh D (2006) Managing non-malignant chronic abdominal pain and malignant bowel obstruction Gastroenteroly Clinics of North America (35)131–142.

Brand WE (1999) Abdomen and pelvis. In Brant WE, Helms CA, ed. Fundamentals of Diagnostic Radiology. 2nd ed. Baltimore, MD: Williams and Wilkins; 651-667.

Casola G and Sirlin CB (2005) Imaging the obstructed bowel and other intestinal emergencies Applied Radiology 34(4)20-34.

Davis MP and Furste A (1999) Glycopyrrolate: a useful drug in the palliation of mechanical bowel obstruction Journal of Pain and Symptom Management (18)153–154.

Davis MP and Walsh D (2000) Treatment of nausea and vomiting in advanced cancer Support Care Cancer (8) 444-452.

Feuer DJ and Broadley KE (2000) Corticosteroids for the resolution of malignant bowel obstruction in advanced gynaecological and gastrointestinal cancer Cochrane Database Syst Rev 2000; CD001219.

Glare P, Pereira G, Kristjanson LJ, Stockler M and Tattersall M (2004) Systematic review of the efficacy of anti-emetics in the treatment of nausea in patients with far-advanced cancer Supportive Care in Cancer (12)432–440.

Gwilliam B and Bailey C (2001) The nature of terminal malignant bowel obstruction and its impact on patients with advanced cancer International Journal of Palliative Nursing 7(10):474-81.

Kottler NE, Aguirre DA, Casola G and Sirlin CB (2005) Imaging the obstructed bowel and other intestinal emergencies Applied Radiology 34 20-34.

Laval G, Arvieux C, Stefani L, Villard ML, Mestrallet JP,Cardin N (2006) Protocol for the treatment of malignant inoperable bowel obstruction: a prospective study of 80 cases at Grenoble University Hospital Center Journal of Pain and Symptom Management 31:502–512.

Letizia M and Norton E (2003) Successful Management of Malignant Bowel Obstruction
Disclosures Journal of Hospice and Palliative Nursing 5(3).

Mecadante S, Ferrera P, Villari P and Marrazzo A (2004) Aggressive pharmacological treatment for reversing malignant bowel obstruction Journal of Pain and Symptom Management (28)412–416.

Mercadante S (2007) Medical treatment for inoperable malignant bowel obstruction: a qualitative systematic review Journal of Pain and Symptom Management 33(2):217-223.

Mercadante S (2009) Intestinal dysfunction and obstruction. In:Walsh D, editor. Palliative Medicine. Philadelphia, PA:Saunders/Elsevier 1267–1275.

Myers J, Tamber A and Farhadian M (2010) Management of treatment-related intermittent partial small bowel obstruction: the use of octreotide Journal of Pain and Symptom Management (39)e1–e3.

Pameijer CR, Mahvi DM, Stewart JA and Weber SM (2005) Bowel obstruction in patients with metastatic cancer: does intervention influence outcome? International Journal of Gastrointestinal Cancer 2005 (35)127–133.

Prommer E (2012) Role of haloperidol in palliative medicine: an update American Journal of Hospice and Palliative Care 29(4):295-301.

Rawlinson F (2001) Malignant bowel obstruction European Journal of Palliative Care 8(4) 137-140.

Ripamonti CI and Mercadante S (2004) Pathophysiology and Management of Malignant bowel obstruction In: Doyle D, Hanks G, Cherny N, Calman K (eds) Oxford Textbook of Palliative Medicine 3rd edn. Oxford University Press:496-507.

Ripamonti CI, Easson AM and Gerdes H (2008) Management of malignant bowel obstruction European Journal of Cancer (44) 1105-1115.

Rawlinson F (2001) Malignant bowel obstruction European Journal of Palliative Care 8(4) 137-140.

Ripamonte CI, Mercadante S, Groff L, Zecca E, De Conno F and Casuccio A (2000) Role of octreotide, scopolamine butylbromide, and hydration in symptom control of patients with inoperable bowel obstruction and nasogastric tubes: a prospective randomized trial Journal of Pain and Symptom Management (19)23–34.

Ripamonti CI and Mercadante S (2004) How to use octreotide for malignant bowel obstruction Journal of Supportive Oncology (2)357–364.

Ripamonti CI, Easson AM and Gerdes H (2008) Management of malignant bowel obstruction European Journal of Cancer (44) 1105-1115.

Roeland E and Von Gunten CF (2009) Current concepts in malignant bowel obstruction management Current Oncology Reports (11)298–303.

Shima Y, Ohtsu A, Shirao K and Sasaki Y (2008) Clinical efficacy and safety of octreotide (SMS201-995) in terminally ill Japanese cancer patients with malignant bowel obstruction Japanese Journal of Clinical Oncology (38)354–359.

Soriano A and Davis MP (2011) Malignant Bowel Obstruction: Individualised treatment near the end of life Cleveland Clinic Journal of Medicine (78)3 197-206.

Szucs RA, Wolf EL, Gramm HF et al (2000) Miscellaneous abnormalities of the colon In Gore RM, Levine MS, ed. Textbook of Gastrointestinal Radiology. 2nd ed. Philadelphia, PA:W.B. Saunders: 1089-1122.

Tuca A, Guell E, Martinez-Losada E and Codorniu N (2012) Malignant bowel obstruction in advanced cancer patients: epidemiology, management, and factors influencing spontaneous resolution Cancer Management and Research 4: 159–169.