**DISORDERS OF THE MOUTH**

**CANDIDIASIS (MONILIASIS/THRUSH)**

This is fungal infection of the mouth.it is caused by a group of yeasts called candida.

**Causes**

Low numbers of the fungus candida are naturally found in the mouth and digestive system of most people. They don’t usually cause any problems, but can lead to oral thrush if they multiply. There are a number of reasons why this may happen, including:

* Taking antibiotics particularly over a long period of time.(they kill bacteria which live in the mouth but do not kill candida which can multiply faster if there is no bacteria around)
* Taking inhaled corticosteroid medication for asthma (reduces the immune response against microorganisms and promotes fungal growth).
* Wearing dentures (false teeth) particularly if they don’t fit properly. Encourages accumulation of bacteria and candida on areas where they tooth cannot be cleaned with saliva promoting fungal growth.
* Having poor oral hygiene.
* Having a dry mouth either because of a medical condition or medication being taken.less saliva thereby promoting fungal growth.
* Smoking
* Chemotherapy or radiotherapy for cancer treatments.
* Babies, the elderly and those with certain underlying medical conditions like DM, vitamin B12 deficiency, hypothyroidism and HIV.
* As most people already have candida fungi living in their mouths, oral thrush is not contagious, meaning it cannot be passed to others.

**Signs and symptoms**

* Cheesy white (patches) plaque that looks like milk curds; when rubbed off, leaving behind red areas that may bleed slightly.
* Loss of taste or unpleasant taste in the mouth.
* Redness inside the mouth and throat.
* Cracks at the corners of the mouth.
* A painful burning sensation in the mouth.

**Management**

* Treat with antifungals which come in the form of gels or liquid which you can apply directly inside the mouth several times a day for 7-14 days, although tablets or capsules are sometimes used.
* If antibiotics or corticosteroids are thought to be the cause, change or reduce the dosage.

**Prevention**

* Rinse the mouth after meals.
* Brush the teeth at least twice a day with tooth paste containing fluoride.
* Regular dental check-up.
* Removing dentures every night, cleaning them with paste or soap and water before soaking the in a solution of water and denture cleaning tablets.
* Stop smoking.
* Rinsing the mouth with water and spitting it out after using a corticosteroid inhaler.
* Ensuring underlying condition such as DM is well controlled.

**DISORDERS OF THE GUMS**

**GINGIVITIS**

* This is inflammation of the gums.

**Causes**

* Poor oral hygiene:
* Food debris,
* Bacterial plaque, (the soft, sticky, colourless film of bacteria that forms constantly on the teeth and gums) if the plaque is not removed daily by brushing, it produces toxins (poisons) that can irritate the gum tissue, causing gingivitis.

**Signs and symptoms**

* Red swollen tender gums that may bleed when you brush.
* The gums may also recede or pull away from the teeth, giving the teeth an elongated appearance.
* Pockets may form between the teeth and gums where plaque and food debris may collect.
* Sometimes recurring bad breath or a bad taste in their mouth.

**Nursing management**

* Teach patient proper oral hygiene; see Preventive Oral Hygiene chart
* Proper brushing to remove plaque and debris and control tartar build up.
* Eating right to ensure proper nutrition for the jaw bone and teeth.
* Avoiding cigarettes and other forms of tobacco.
* Regular dental check-ups.

**PERIODONTITIS**

* This is inflammation of the gums and the supporting structures of the teeth.This occurs if gingivitis is left untreated.

**Causes**

* Bacterial plaque.
* May result from untreated gingivitis
* Poor or inadequate dental hygiene
* Inadequate diet contributes to development.
* Smoking, uncontrolled diabetes, obesity, poor oral hygiene.

**Signs and symptoms**

* Like gingivitis
* Red swollen tender gums that may bleed when you brush
* The gums may also recede or pull away from the teeth, giving the teeth an elongated appearance.
* Loosening of teeth
* Pockets may form between the teeth and gums which are defined by an opening around the gum tissue of the teeth where plaque and food debris may collect leading to the formation of tartar to get deeper under the gum line.
* This deepening of the pocket is associated with bone loss and can lead to tooth loss if left untreated
* Sometimes recurring bad breath or a bad taste in their mouth.

**Complications**

* Tooth loss
* Increased risk of cardiovascular disease

**Management**

* Proper oral hygiene to reduce bacteria in the mouth and inflammation.
* Professional cleaning by a dentist and application of fluoride paste.
* Antibiotic therapy for persistent gum infection.
* Sometimes surgical treatment to clean away plaque, bacteria and deposits that are under the gums.
* If a smoker, quit.

**STOMATITIS**

* A condition that causes painful swelling and sores inside the mouth. Can occur anywhere in the mouth including inside of the cheeks, gums, tongue, lips and palate.

**Types**

**Canker sore/apthous ulcer:** This is a single pale or yellow ulcer with a red outer ring or a cluster of such ulcers in the mouth usually on the cheeks, tongue or inside the lip.

**Causes**

* Certain medications which causes mucosal reactions
* Trauma to the mouth
* Poor nutrition(malnutrition as lack of iron, vitamin B12 which is necessary for cell replication and repair lack of it leads to ineffective repair and regeneration of epithelial cells especially in the mouth and lips)
* Bacteria or viruses which causes inflammation
* Certain foods such as citrus foods.
* Temporarily reduced immune system because of a flu or cold.
* Hormonal changes.
* Biting inside of the cheek
* A sharp piece of food.

**Signs and symptoms**

* Can be painful.
* Usually lasts 5-10 days.
* Tend to recur.
* Are generally not associated with fever.

**Management**

The main aim of treatment is to relieve discomfort and protect against infection by the following ways:

* Drinking more water.
* Rinse the mouth with salty water.
* Proper dental hygiene.
* Soft bland diet.
* Apply topical corticosteroid (prednisone) as they reduce swelling and pain.

**Cold sores/fever blisters:** These are fluid filled sores that occur on or around the lips.

**Causes**

* Herpes simplex type I which leads to inflammation of the mucosal lining.
* They are contagious from the time the blister ruptures to the time it has completely healed.
* The initial infection often occurs before adulthood and may be confused with a cold or the flu.
* Once the person is infected with the virus, it stays in the body becoming dormant and reactivated by such conditions as stress, fever, trauma.
* When the sores reappear, they tend to form in the same location. In addition to spreading to other people, the virus can also spread to another body part of the affected person, such as the eyes or genitals.

**Signs and symptoms Cold sores**

* Are usually painful.
* Are usually gone in 7-10 days.
* Are sometimes associated with cold or flu like symptoms.

**Management**

* Taking acyclovir 400mg four times a day for 3 days.
* Applying acyclovir cream to the lesion 4 times a day.
* Applying ice to the lesion.
* Soft, bland (mildly spiced) diet.

**DENTAL CARIES/CAVITIES**

This is the breakdown of teeth due to activities of bacteria.

The bacteria produce acid that destroys the tooth enamel and the layer under it, the dentin.

**How they occur**

Many different types of bacteria normally live in the human mouth. They build up on the teeth in a sticky film called plaque.

This plaque also contains saliva, bits of food and natural substances. It forms most easily in certain places which include:

Cracks, grooves in the back of the teeth.

Between teeth.

Around dental fillings.

Near the gum line.

The bacteria turn sugar and carbohydrates in the food we eat into acids. The acid dissolves minerals in the hard enamel that covers the tooth crown (the part you can see). The enamel erodes or develops pits. They are too small to see at first but they get larger over time.

Acid can seep through the pores in the enamel. This how decay begins in the softer dentin layer, the main body of the tooth. As the dentin and enamel break down, a cavity is created.

If the decay is not removed, bacteria will continue to grow and produce acid that eventually will get into the tooth’s inner layer. This contains the soft pulp and sensitive nerve fibres.

Tooth roots exposed by receding gums also can develop decay. The root’s outer layer, cementum, is not as thick as enamel. Acids from plaque bacteria can dissolve it rapidly.

When the blood, lymph vessels, and nerves are exposed, they become infected and an abscess may form, either within the tooth or at the tip of the root.

**Symptoms**

* Early caries may not have symptoms.
* Later when the decay has eaten through the enamel, the teeth may be sensitive to sweet, hot or cold foods or drinks.
* Soreness and pain usually occur with an abscess.
* As the infection continues, the patient’s face may swell, and there may be pulsating pain.

The dentist can determine by x-ray studies the extent of damage and the type of treatment needed.

Treatment for dental caries includes fillings, dental implants, and extractions.

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**TREATMENT**

* In the early stages, tooth decay can be stopped.it can even be reversed. Fluoride help a tooth in the early stages of decay to repair itself (remineralize)
* Once the carries get worse and there is a break in the enamel, only the dentist can repair the tooth. The standard treatment for a cavity is to fill the tooth. The decayed material in the cavity is removed and the cavity is filled.
* If a cavity is large, the remaining tooth may not be able to support enough filling material to repair it. In this case, the dentist will remove the decay and cover the tooth with a ceramic inlay or onlay or artificial crown. Which can be made in an office or lab.
* Sometimes bacteria infect the pulp inside the tooth even if the part of the tooth you can see remains intact. In this case, the tooth will need root canal treatment. The pulp will be removed and replaced with an inert material. In most cases, the tooth will need a crown.
* If treatment is not successful, the tooth may need to be extracted

**Prevention**

Measures used to prevent and control dental caries include:

* Practicing effective mouth care.
* Reducing the intake of starches and sugars (refined carbohydrates).
* Applying fluoride to the teeth or drinking fluoridated water,
* Refraining from smoking.
* Controlling diabetes.
* Using pit and fissure sealants.

**MOUTH CARE**

Healthy teeth must be effectively cleaned on a daily basis.

Brushing and flossing are particularly effective in mechanically breaking up the bacterial plaque that collects around teeth.

Normal mastication (chewing) and the normal flow of saliva also aid greatly in keeping the teeth clean.

Because many ill patients do not eat adequate amounts of food, they produce less saliva, which in turn reduces this natural tooth cleaning process.

The nurse may need to assume the responsibility for brushing the patient’s teeth. In any case, merely wiping the patient’s mouth and teeth with a swab is ineffective. The most effective method is mechanical cleansing (brushing). If brushing is impossible, it is better to wipe the teeth with a gauze pad, then have the patient swish an antiseptic mouthwash several times before expectorating into an emesis basin.

A soft-bristled toothbrush is more effective than a sponge or foam stick.

The lips may be coated with a water soluble gel to prevent drying.

**DIET**

Dental caries may be prevented by decreasing the amount of sugar and starch in the diet. Patients who snack should be encouraged to choose less cariogenic alternatives, such as fruits, vegetables, nuts, cheeses, or plain yogurt.

**FLUORIDATION**

Fluoride provides a protective coating that neutralizes acid attack caused by plaque.

Fluoridation of public water supplies has been found to decrease dental caries.

Fluoridation may be achieved also by having a dentist apply a concentrated gel or solution to the teeth, adding fluoride to home water supplies, using fluoridated toothpaste or mouth rinse.

**PIT AND FISSURE SEALANTS**

The occlusal (the chewing surface) surfaces of the teeth have pits and fissures for the molar and premolar teeth, areas that are prone to caries. These areas can trap plaque, causing decay. In adults, molars can be protected with sealants. In children both baby molars and permanent molars can be sealed. Dentists also can use sealants on molars that have early signs of tooth decay as long as the decay has not broken through the enamel to fill and seal these areas from potential exposure to cariogenic processes. These sealants last up to 7 years.

**DENTAL ABSCESS**

A dental abscess is a collection of pus that can form inside the teeth, in the gums or in the bone that holds the teeth in place.

An abscess at the end of a tooth is called periapical abscess. An abscess in the gums is known periodontal abscess.

**Symptoms**

* An intense, throbbing pain in the affected tooth or gum that may come on suddenly and gets gradually worse.
* Pain that spreads to the ear, jaw and neck on the same side as the affected tooth or gum.
* Pain that’s worse when lying down, which disrupts sleep.
* Redness and swelling in the face.
* Tender, discoloured and or loose tooth.
* Shiny red swollen gums.
* Sensitivity to hot or cold food and drink.
* Bad breath and or unpleasant taste in the mouth.
* Fever if the infection has spread.
* Hard to fully open the mouth (trismus).
* Difficulty in swallowing or breathing.
* General malaise (the patient generally feels unwell)

**Causes**

* The abscess typically originates from a bacterial infection, often one that has accumulated in the soft pulp of the tooth.
* Poor oral hygiene which leads to building up of plague.
* Consuming lots of sugary or starchy foods and drinks which can encourage the growth of bacteria in plaque and may lead to decay that can result in an abscess.
* An injury or previous surgery to the teeth and gums: bacteria can get into any damaged parts of the teeth or gums.
* Having a weakened immune system: this includes people with certain underlying conditions such as DM, and those having treatments including steroid medication or chemotherapy.

**Relieving the symptoms**

* Take pain killers to relieve the pain.
* Avoid hot or cold food and drink if it makes the pain worse.
* Try eating cool, soft foods if possible, using the opposite side of the mouth.
* Use a soft brush on the affected side.

**Management**

Aim of treatment is to remove the source of infection and draining away the pus.

* Depending on the location of the abscess and how severe the infection is, possible treatments include:
* Removing the affected tooth (extraction) – this may be necessary if root canal treatment is not possible. Root canal treatment is a procedure to remove the abscess from the root of the affected tooth before filling and sealing it.
* Incision and drainage-involves a small incision is made in the gums to drain the abscess (this is temporary solution and further treatment may be needed)

**Prevention**

* Brush the teeth with fluoride tooth paste twice a day for at-least two minutes each time.
* Avoid rinsing the mouth with water after brushing because this washes the protective tooth paste away – just spit out any excess tooth paste.
* Cut down on sugary and starchy food and drinks particularly between meals or shortly before going to bed.
* Regular dental check-up.

**Nursing Management**

* The nurse assesses the patient for bleeding after treatment and instructs the patient to use a warm saline or warm water mouth rinse to keep the area clean.
* The patient is also instructed to take antibiotics and analgesics as prescribed, to advance from a liquid diet to a soft diet as tolerated, and to keep follow-up appointments.

**DISORDERS OF THE ESOPHAGUS**

The esophagus is a mucus-lined, muscular tube that carries food from the mouth to the stomach. Its ability to transport food and fluid is facilitated by two sphincters.

The upper esophageal sphincter, also called the hypopharyngeal sphincter, is located at the junction of the pharynx and the esophagus.

The lower esophageal sphincter, also called the gastroesophageal sphincter, is located at the

junction of the esophagus and the stomach.

**Dysphagia** (difficulty swallowing) is the most common symptom of esophageal disease.

This symptom may vary from an uncomfortable feeling that a bolus of food is caught in the upper esophagus (before it eventually passes into the stomach) to acute pain on swallowing (**odynophagia**).

Obstruction of food (solid and soft) and even liquids may occur anywhere along the esophagus.

Often the patient can indicate that the problem is located in the upper, middle, or lower third of the esophagus.

**ACHALASIA**

* Thisis absent or ineffective peristalsis of the distal (lower) esophagus, accompanied by failure of the esophageal sphincter to relax in response to swallowing.
* Narrowing of the esophagus just above the stomach results in a gradually increasing dilation of the esophagus in the upper chest. Achalasia may progress slowly and occurs most often in people 40 years of age or older.
* This is a disease of the muscle of the lower esophageal sphincter that prevents relaxation of the sphincter and an absence of contractions or peristalsis of the esophagus.

**CAUSES**

* Degeneration of the esophageal muscles and nerves that control the muscles.

**SYMPTOMS**

* Difficulty in swallowing both solids and liquids.
* The patient has a sensation of food sticking in the lower portion of the esophagus
* As the condition progresses, food is commonly regurgitated, either spontaneously or intentionally by the patient to relieve the discomfort produced by prolonged distention of the esophagus by food that will not pass into the stomach.
* The patient may also complain of chest pain and heartburn (**pyrosis**).

**DIAGNOSIS**

* X-ray studies show esophageal dilation above the narrowing at the gastroesophageal junction. Barium swallow, computed tomography(CT) of the esophagus, and endoscopy may be used for diagnosis.

**MANAGEMENT**

The goal of therapy for achalasia is to relieve symptoms by eliminating the outflow resistance caused by the hypertensive and non relaxing LES.

Pharmacologic and other non surgical treatments include the following:

Administration of calcium channel blockers and nitrates decrease LES pressure by preventing calcium entry into the cells and improve swallowing.

Endoscopic intrasphincter injection of botulin toxin to block acetylcholine release at the level of the LES. Injection of botulinum toxin (Botox) to quadrants of the esophagus via endoscopy has been helpful because it inhibits the contraction of smooth muscle.

If these methods are unsuccessful, pneumatic (forceful) dilation or surgical separation of the muscle fibers may be recommended.

COMPLICATIONS

Lung problems, weight loss.

**HIATAL HERNIA**

* Any time an internal body part pushes into an area where it doesn’t belong, it’s called a hernia. The hiatus is an opening in the diaphragm-the muscular wall separating the chest cavity from the abdomen. Normally the esophagus goes through the hiatus and attaches to the stomach.
* In a hiatal hernia the stomach bulges up into the chest through that opening.
* The esophagus enters the abdomen through an opening in the diaphragm and empties at its lower end into the upper part of the stomach.
* Normally, the opening in the diaphragm encircles the esophagus tightly, and the stomach lies completely within the abdomen.
* In a condition known as hiatus (or hiatal) **hernia**, the opening in the diaphragm through which the esophagus passes becomes enlarged, and part of the upper stomach tends to move up into the lower portion of the thorax.
* There are two types of hiatal hernias: sliding and paraesophageal (next to the esophagus).
* In a **sliding hiatal hernia**, the stomach and the section of the esophagus that joins the stomach slide up into the chest through the hiatus. Common type.
* Sliding, or type I, hiatal hernia occurs when the upper stomach and the gastroesophageal junction (GEJ) are displaced upward and slide in and out of the thorax
* The paraesophageal hernia, the esophagus and the stomach stay in their normal locations, but part of the stomach squeezes through the hiatus, landing it next to the esophagus.
* A paraesophageal hernia occurs when all or part of the stomach pushes through the diaphragm beside the esophagus.

**Causes**

* Most of the time the cause is unknown.
* A person may be born with a larger hiatal opening.
* Increased pressure in the abdomen such as from pregnancy, obesity, coughing or straining during bowel movements.

**Risk Factors**

* Overweight.
* People older than 50 years.

**Symptoms**

* The patient with a sliding hernia may have heartburn, regurgitation, and dysphagia, but at least 50% of patients are asymptomatic.
* Sliding hiatal hernia is often implicated in reflux. The patient with a paraesophageal hernia usually feels a sense of fullness after eating or may be asymptomatic.
* Reflux usually does not occur, because the gastroesophageal sphincter is intact.

**Diagnosis**

* Diagnosis is confirmed by x-ray studies, barium swallow, and fluoroscopy.

**Management**

* Most people do not experience any symptoms of their hiatal hernia so no treatment is necessary.
* However, the paraesophageal hernia can sometimes cause the stomach to be strangled, so surgery is recommended especially if the hiatal hernia is in danger of becoming constricted or strangulated(so that the blood supply is cut off) surgery is needed to reduce the hernia,meaning to put it back to where it belongs.
* The complications of hemorrhage, obstruction, and strangulation can occur with any type of hernia.

**GASTROESOPHAGEAL REFLUX DISEASE**

* This is a digestive disorder that affects the lower esophageal sphincter, the ring of muscle between the esophagus and the stomach.
* Gastroesophageal refers to the stomach and esophagus.reflux means to flow back or return. Therefore GERD is the return of the stomach’s content back up into the esophagus.

**How does it occur?**

* In normal digestion, the lower esophageal sphincter (LES) opens to allow food to pass into the stomach and closes to prevent food and acidic stomach juices from flowing back into the esophagus. GERD occurs when the LES is weak or relaxes inappropriately, allowing stomach and duodenal contents to flow up into the esophagus.

**Clinical Manifestations**

* Pyrosis (burning sensation in the esophagus) heartburn usually feels like a burning chest pain beginning behind the breastbone and moving upward to the neck and throat. Many people say it feels like food is coming back into the mouth leaving an acid or bitter taste,
* Dyspepsia (indigestion), regurgitation, dysphagia or odynophagia (difficulty swallowing, pain on swallowing),

**Assessment and Diagnostic Findings**

* Diagnostic testing may include an endoscopy or barium swallow to evaluate damage to the esophageal mucosa.

**Management**

Doctors recommend lifestyle and dietary changes for most people needing treatment for GERD. Treatment aims at decreasing the amount of reflux or reducing damage to the lining of the esophagus from refluxed materials.

Management begins with teaching the patient to avoid situations that decrease lower esophageal sphincter pressure or cause esophageal irritation. That can weaken the LES. Such as fatty foods, coffee and alcoholic beverages.

* Foods and beverages that can irritate a damaged esophageal lining, such as citrus fruits and juices, tomato products, pepper should be avoided if they cause symptoms.
* Decreasing the size of portions at mealtime also helps control symptoms.
* Eating meals at least 2-3 hours before bed time may lessen reflux by allowing the acid in the stomach to decrease and the stomach to empty partially.
* Being overweight often worsens symptoms, relief when one loses weight.
* Cigarette smoking weakens the LES, stopping reduces GERD symptoms.
* Elevating the head of the bed with blocks on 6 inch blocks reduces heartburn by allowing gravity to minimize reflux of stomach contents into the esophagus.
* If reflux persists, the patient may be given medications such as antacids which help neutralize acid in the esophagus and stomach. or histamine receptor blockers(cimetidine,ranitidine) which inhibit acid secretion in the stomach. Proton pump inhibitors (medications that decrease the release of gastric acid, such as lansoprazole [Prevacid] or rabeprazole may be used.

**CANCER OF THE ESOPHAGUS**

This is a type of cancer that affects the esophagus. It mainly affects people in their 60s and 70s.

**Symptoms**

In the early stages when the tumor is small, the cancer usually doesn’t cause any symptoms. Its only when it gets bigger symptoms tends to develop which include:

* Difficulty in swallowing initially with solid foods eventually with liquids.
* Persistent indigestion or heartburn.
* Bringing up food soon after eating.
* Loss of appetite and weight loss.
* A sensation of a mass in the throat.
* Pain or discomfort in the upper part of the abdomen, chest or back.
* Haemorrhage may take place.
* Progressive loss of weight and strength occurs from starvation.
* Later symptoms include substernal pain, persistent hiccup, respiratory difficulty, and foul breath.

**Causes**

* The exact cause is unknown, but the following things can increase the risk:
* Persistent GERD.
* Smoking.
* Drinking too much alcohol over a long period of time.
* Being overweight or obese.
* Having unhealthy diet that’s low in fruits and vegetables.

**Treatment**

* If diagnosed at an early stage, it may be possible to cure it with:
* Surgery to remove the affected section of the esophagus.
* Chemotherapy with or without radiotherapy to kill the cancerous cells and shrink the tumour.
* If diagnosed at a later stage, a cure may not be achievable. But in these cases, surgery, chemotherapy and radiotherapy can be used to help keep the cancer under control and relieve symptoms.

**CONDITIONS OF THE STOMACH**

**GASTRITIS**

* This is an inflammation, irritation, or erosion of the lining of the stomach. It can occur suddenly lasting several hours to a few days, (acute) or gradually resulting from repeated exposure to irritating agents or recurring episodes of acute gastritis.(chronic)

**Causes**

Acute gastritis is often caused by:

* The person eats food that is contaminated with disease-causing microorganisms
* That is irritating or too highly seasoned.
* Overuse of aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs),
* Excessive alcohol intake
* Bile reflux
* Radiation therapy.
* A more severe form of acute gastritis is caused by the ingestion of strong acid or alkali, which may cause the mucosa to become gangrenous or to perforate. Scarring can occur, resulting in pyloric obstruction.
* Gastritis also may be the first sign of an acute systemic infection.

Chronic gastritis and prolonged inflammation of the stomach may be caused by

Either benign or malignant ulcers of the stomach

* The bacteria *Helicobacter pylori.*
* Autoimmune diseases such as pernicious anemia.
* Dietary factors such as caffeine
* The use of medications, especially NSAIDs
* Alcohol
* Smoking;
* Reflux of intestinal contents into the stomach.
* Infections caused by bacteria and viruses.
* If gastritis is left untreated, it can lead to severe loss of blood and may increase the risk of developing stomach cancer.

**Pathophysiology**

* In gastritis, in response to inflammation, the gastric mucous membrane becomes edematous and hyperemic (congested with fluid and blood) and undergoes superficial erosion.
* It secretes a scanty amount of gastric juice, containing very little acid but much mucus. Superficial ulceration may occur and can lead to hemorrhage.

**Symptoms**

* Nausea or recurrent upset stomach.
* Abdominal bloating.
* Abdominal pain.
* Vomiting.
* Indigestion.
* Burning or gnawing feeling in the stomach between meals or at night.
* Hiccups
* Loss of appetite.
* Vomiting blood or coffee ground like material.
* Black tarry stools.

**Diagnosis**

* Upper endoscopy: an endoscope, a thin tube containing a tiny camera, is inserted through the mouth and down into the stomach to look at the stomach lining. Inflammation is checked and biopsy may be done.
* Blood tests: FHG to check the RBC count to determine the presence of anaemia.
* Screen for the presence of H.pylori
* Fecal occult blood tests (stool test): checks for the presence of blood in stool which is a possible sign for gastritis.

**Management**

* Taking antacids and other drugs such as proton pump inhibitors or H-2 blockers to reduce stomach acid.
* Avoiding hot and spicy foods.
* For gastritis caused by h.pylori infection,antibiotics will be given plus acid blocking drugs.
* Acute gastritis is also managed by instructing the patient to refrain from alcohol until symptoms subside. After the patient can take nourishment by mouth, a nonirritating diet is recommended.
* If the symptoms persist, fluids may need to be administered parenterally as nutritional supplement.
* If gastritis is caused by ingestion of strong acids or alkalis, treatment consists of diluting and neutralizing the offending agent. To neutralize acids, common antacids (eg, aluminum hydroxide) are used; to neutralize an alkali, diluted lemon juice or diluted vinegar is used.
* If corrosion is extensive or severe, emetics and lavage are avoided because of the danger of perforation and damage to the esophagus.

**PEPTIC ULCERS**

* A peptic ulcer is an excavation (hollowed-out area) that forms in the mucosal wall of the stomach, in the pylorus (opening between stomach and duodenum), in the duodenum (first part of small intestine), or in the esophagus.
* A peptic ulcer is frequently referred to as a gastric, duodenal, or esophageal ulcer, depending on its location, or as peptic ulcer disease.

**Causes**

* Erosion of a circumscribed area of mucous membrane is the cause. This erosion may extend as deeply as the muscle layers or through the muscle to the peritoneum.
* In the past, stress and anxiety were thought to be causes of ulcers.
* Infection with the gram-negative bacteria *H. pylori.*
* However, ulcers do seem to develop more commonly in people who are tense; whether this is a contributing factor to the condition is uncertain.
* Excessive secretion of HCl in the stomach may contribute to the formation of gastric ulcers, and

stress may be associated with its increased secretion.

* The ingestion of milk and caffeinated beverages, smoking,chronic use of NSAIDS and alcohol also may increase HCl secretion.

**How the disease occurs?**

* Peptic ulcers occur mainly in the gastroduodenal mucosa because this tissue cannot withstand the digestive action of gastric acid (HCl) and pepsin.
* The erosion is caused by the increased concentration or activity of acid-pepsin, or by decreased resistance of the mucosa.
* A damaged mucosa cannot secrete enough mucus to act as a barrier against HCl.
* The use of NSAIDs inhibits the secretion of mucus that protects the mucosa.

**Symptoms**

* As a rule, the patient with an ulcer complains of dull, continuous pain or a burning sensation in the middle or upper stomach or in the back.
* It is believed that the pain occurs when the increased acid content of the stomach and duodenum erodes the lesion and stimulates the exposed nerve endings.
* Pain is usually relieved by eating, because food neutralizes the acid, or by taking alkali; however, once the stomach has emptied or the alkali’s effect has decreased, the pain returns.
* Sharply localized tenderness can be elicited by applying gentle pressure to the epigastrium at or slightly to the right of the midline.
* Other symptoms include **pyrosis** (heartburn), vomiting blood that can look like coffee ground, constipation, dark or black stool due to bleeding, nausea or vomiting.

**Assessment and Diagnostic Findings**

* A physical examination may reveal pain, epigastric tenderness, or abdominal distention.
* A barium study of the upper GI tract may show an ulcer; however, endoscopy is the preferred diagnostic procedure because it allows direct visualization of inflammatory changes, ulcers, and lesions.
* Stools may be tested periodically until they are negative for occult blood.
* *H. pylori* infection may be determined by biopsy and histology with culture.

**Management**

Methods used include medications, lifestyle changes.

Pharmacology:

* If NSAIDS are the cause and there is no H.pylori infection,stop taking or reduce how much of it that is taken.
* Proton pump inhibitors: reduce acid levels,protect the lining of the stomach and allow the ulcer to heal. E.g omeprazole.
* Histamine receptor blockers: block histamine, a chemical in the body that signals the stomach to produce acid. E.g cimetidine,ranitidine
* Antacids: makes the pain from a peptic ulcer go away temporarily, but does not treat the infection
* Antibotitics:to eradicate H.pylori infections. Taken for one or two weeks. Consider triple therapy i.e clarithromycin,metronidazole,amoxicillin
* The patient is advised to adhere to the medication regimen to ensure complete healing of the ulcer. Because most patients become symptom-free within a week, it becomes a nursing responsibility to stress the importance of following the prescribed regimen so that the healing process can continue uninterrupted and the return of chronic ulcer symptoms can be prevented.
* Reducing environmental stress requires physical and psychological modifications on the patient’s part as well as the aid and cooperation of family members and significant others. The patient may need help in identifying situations that are stressful or exhausting.
* Studies have shown that smoking decreases the secretion of bicarbonate from the pancreas into the duodenum, resulting in increased acidity of the duodenum. Therefore, the patient is strongly encouraged to stop smoking.
* The intent of dietary modification for patients with peptic ulcers is to avoid over-secretion of acid and hypermotility in the GI tract.
* In addition, an effort is made to neutralize acid by eating three regular meals a day.

**GASTRIC/STOMACH CANCER**

* Begins when cancer cells form in the inner lining of the stomach. These cells can grow into a tumor.

**Causes**

* The exact cause is unknown.

**Risk factors**

* Infection with H.pylori
* Gastritis.
* long lasting anemia (pernicious anemia)
* Growths in the stomach (polyps)
* Smoking
* Being overweight or obese
* A diet high in smoked, preserved or salty foods.
* Certain genes.
* Working in coal, metal, timber or rubber industries.

**Symptoms**  
In the early stages of gastric cancer, symptoms may be absent or the following may be observed:

* Indigestion.
* Feeling bloated after eating.
* Heartburn.
* Slight nausea
* Loss of appetite.

As the stomach tumour grows, there may be serious symptoms as:

* Stomach pain
* Blood in stool
* Vomiting.
* Weight loss for no reason.
* Trouble swallowing.
* Yellowish eyes or skin.
* Swelling in the stomach.
* Constipation or diarrhoea.
* Weakness or feeling tired.
* Heartburn.

**Diagnosis**

* Taking history to determine if there is any family history.
* Upper endoscopy: a thin flexible tube with a small camera is put down the throat to look into the stomach.
* Barium swallow: Coats the stomach and makes it show up clearly on x-rays.
* Ct scan: Powerful x-ray that makes detailed pictures of inside the stomach.
* Biopsy: to look for signs of cancer.

**Management**

* Surgery: involves removing part of the stomach and nearby tissues that might have cancer cells.
* Chemotherapy: drugs kill cancer cells or keep them from growing.
* Radiotherapy: high energy waves or particles can kill cancer cells and shrink tumours.

**Prevention**

* Treat stomach infections.
* Eat healthy: get more fresh fruits and vegetables.
* Avoid very salty, preserved, cured or smoked foods like processed lunch meats and smoked cheeses. Maintain normal weight.
* Avoid smoking.
* Reduce use of NSAIDS.

**MANAGEMENT OF PATIENTS WITH INTESTINAL AND RECTAL DISORDERS**

**APPENDICITIS**

* The appendix is a small, finger-like appendage about 10 cm (4 in) long that is attached to the cecum just below the ileocecal valve.
* The appendix fills with food and empties regularly into the cecum.
* Because it empties inefficiently and its lumen is small, the appendix is prone to obstruction and is particularly vulnerable to infection (ie, appendicitis).
* A condition in which the appendix becomes inflamed and filled with pus, causing pain.

**Pathophysiology**

* The appendix becomes inflamed and edematous as a result of either becoming kinked or occluded by a fecalith (ie, hardened mass of stool), tumor, or foreign body. The inflammatory process increases intraluminal pressure, initiating a progressively severe, generalized or upper abdominal pain that becomes localized in the right lower quadrant of the abdomen within a few hours.
* Eventually, the inflamed appendix fills with pus.

**Symptoms**

* Dull pain near the navel or the upper abdomen that becomes sharp as it moves to the lower right abdomen. This is usually the first sign.
* Loss of appetite.
* Nausea and vomiting soon after abdominal pain begins.
* Abdominal swelling
* Fever
* Inability to pass gas.
* Local tenderness is elicited at McBurney’s point when pressure is applied.
* Rebound tenderness (ie, production or intensification of pain when pressure is released) may be present.

**Assessment and Diagnostic Findings**

* Diagnosis is based on results of a complete physical examination(abdominal exam to detect inflammation) and on laboratory and x-ray findings.
* The complete blood cell count demonstrates an elevated white blood cell count.
* Abdominal x-ray films, ultrasound studies, and CT scans may reveal a right lower quadrant density or localized distention of the bowel.

**Complications**

The major complication of appendicitis is perforation of the appendix, which can lead to peritonitis or an abscess.

**Medical Management**

* Surgery is indicated if appendicitis is diagnosed.
* To correct or prevent fluid and electrolyte imbalance and dehydration, antibiotics and intravenous fluids are administered until surgery is performed.
* Analgesics can be administered after the diagnosis is made.
* Appendectomy (ie, surgical removal of the appendix) is performed as soon as possible to decrease the risk of perforation.if this happens infectious material will be spill into the abdominal cavity which can lead to peritonitis.
* It may be performed under a general or spinal anesthetic with a low abdominal incision or by laparoscopy.

**Nursing Management**

* Goals include relieving pain, preventing fluid volume deficit, reducing anxiety, eliminating infection from the potential or actual disruption of the GI tract, maintaining skin integrity, and attaining optimal nutrition.
* The nurse prepares the patient for surgery, which includes an intravenous infusion to replace fluid loss and promote adequate renal function and antibiotic therapy to prevent infection.
* If there is evidence or likelihood of paralytic ileus, a nasogastric tube is inserted.
* An enema is not administered because it can lead to perforation.
* After surgery, the nurse places the patient in a semi-Fowler position. This position reduces the tension on the incision and abdominal organs, helping to reduce pain.
* An opioid, usually morphine sulfate, is prescribed to relieve pain.
* When tolerated, oral fluids are administered.
* Any patient who was dehydrated before surgery receives intravenous fluids. Food is provided as desired
* and tolerated on the day of surgery.
* When the patient is ready for discharge, the nurse teaches the patient and family to care for the incision and perform dressing changes and irrigations as prescribed.

**ULCERATIVE COLITIS**

* This is a disease that causes inflammation and sores or ulcers in the lining of the large intestine/colon. It usually affects the lower section (sigmoid colon) and the rectum. But it can affect the entire colon.
* It is thought to be an autoimmune disorder, meaning the body’s defence against infection goes wrong and attacks the healthy tissue. The immune system mistakes harmless bacteria inside the colon for a threat and attacks the tissues of the colon, causing it to become inflamed.

**Pathophysiology**

* Ulcerative colitis affects the superficial mucosa of the colon and is characterized by multiple ulcerations, diffuse inflammations, and desquamation or shedding of the colonic epithelium.
* Bleeding occurs as a result of the ulcerations.
* The mucosa becomes edematous and inflamed. The lesions are contiguous, occurring one after the other. Abscesses form, and infiltrate is seen in the mucosa and submucosa with clumps of neutrophils in the crypt
* lumens (ie, crypt abscesses).
* The disease process usually begins in the rectum and spreads proximally to involve the entire colon.
* Eventually, the bowel narrows, shortens, and thickens because of muscular hypertrophy and fat deposits.

**Causes**

???? the immune system overreacting to normal bacteria in the digestive tract.

**Symptoms**

* Abdominal pain or cramps
* Diarrhoea
* Bleeding from the rectum.
* The patient may have anorexia, weight loss, fever, vomiting, and dehydration, as well as cramping, the feeling of an urgent need to defecate,

**Diagnosis**

* Doctor asks about the symptoms,do a physical exam
* A colonoscopy in this test a doctor uses a thin lighted tool to look at the inside of the entire colon.
* Blood tests (FHG) which look for infection or inflammation.( laboratory test results reveal

a low hematocrit and hemoglobin concentration in addition to an elevated white blood cell count,)

* Stool sample testing to look for blood, infection, and white blood tests.

**Complications**

Complications of ulcerative colitis include toxic megacolon, perforation, and bleeding as a result of ulceration, vascular engorgement, and highly vascular granulation tissue.

In toxic megacolon, the inflammatory process extends into the muscularis, inhibiting its ability to contract and resulting in colonic distention. Symptoms include fever, abdominal pain and distention, vomiting,

and fatigue.

MANAGEMENT

aimed at reducing inflammation, suppressing inappropriate immune responses, providing rest for a diseased bowel so that healing may take place, improving quality of life, and preventing or minimizing complications.

NUTRITIONAL THERAPY

Oral fluids and a low-residue, high-protein, high-calorie diet (vegatables) with supplemental vitamin therapy and iron replacement are prescribed to meet nutritional needs, reduce inflammation, and control pain and diarrhea. Fluid and electrolyte imbalances from dehydration caused by diarrhoea are corrected by intravenous therapy as necessary

PHARMACOLOGIC THERAPY

Sedatives and antidiarrheal and antiperistaltic medications are used to minimize peristalsis to rest the inflamed bowel. They are continued until the patient’s stools approach normal frequency and consistency.

Aminosalicylate formulations such as sulfasalazine (Azulfidine) are often effective for mild or moderate inflammation and are used to prevent or reduce recurrences in long-term maintenance regimens.

Antibiotics are used for secondary infections, particularly for purulent complications such as abscesses, perforation, and peritonitis.

Corticosteroids are used to treat severe and fulminant disease.

These corticosteroids (eg, prednisone) can be administered orally in outpatient treatment or parenterally in hospitalized patients.

Topical (ie, rectal administration) corticosteroids are also widely used in the treatment of distal colon disease.

**INTESTINAL OBSTRUCTION**

An obstruction can occur when there is no open passageway for food or digested food waste to move through the bowel or intestine. It can occur anywhere in the small or large intestine, and there can be a partial or complete blockage.

The bowel is basically a hollow tube that transports food and digested food waste from the stomach to the back passage (anus)

There are two sections of the bowel: the small bowel/small intestine, which is where the nutrients in the food are digested and absorbed; and the colon and rectum from te large bowel/large intestine, which absorbs water from the digested food, forming it into stools that are passed out of the back passage.

When an obstruction occurs,undigested food,liquids and digestive secretions accumulate above the blockage,the bowel section involved in the blockage becomes distended and the segment can collapse.

Intestinal obstruction exists when blockage prevents the normal flow of intestinal contents through the intestinal tract.

**Causes**

Can be mechanical and non-mechanical.

*Functional obstruction/non mechanical:* situations in which the intestinal musculature cannot propel the contents along the bowel. Examples are amyloidosis, muscular dystrophy, endocrine disorders such as diabetes mellitus, or neurologic disorders such as Parkinson’s disease. The blockage also can be temporary and the result

of the manipulation of the bowel during surgery.

In paralytic ileus, the rhythmic muscle contractions of the intestines, known as peristalsis,stops. The bowel becomes dilated and can no longer move the contents to the anus.it occurs if there are medical conditions as above.

Ileus can sometimes occur after some types of surgery or during the post natal period.

*Mechanical obstruction:* An intraluminal obstruction or amural obstruction from pressure on the intestinal walls occurs. Examples are intussusception, polypoid tumors and neoplasms, stenosis, strictures, adhesions, hernias, and abscesses.

Adhesions/scar tissue,that can form after abdominal surgery and trap a section of the bowel, are some of the most commoncauses of mechanical bowel obstruction. Other common types of mechanical obstruction of the small bowel include a hernia (where part of the bowel pushes through a weak area in the abdominal wall) or volvulus (where the bowel becomes twisted)

A mechanical obstruction in the large bowel is most often caused by a malignant tumour. Volvulus can also occur in the large bowel ,most often in the sigmoid colon.

Other possible causes of bowel obstruction include:

Impacted stool from severe constipation.

Diseases that affect the intestinal wall such as crohn’s or diverticular disease.

Gall stones

A swallowed item

Intussusceptions, where part of the intestines folds on itself.

Congenital malformation of the bowel.

**SMALL BOWEL OBSTRUCTION**

**Pathophysiology**

Intestinal contents, fluid, and gas accumulate above the intestinal obstruction. The abdominal distention and retention of fluid reduce the absorption of fluids and stimulate more gastric secretion.

With increasing distention, pressure within the intestinal lumen increases, causing a decrease in venous and arteriolar capillary pressure.

This causes edema, congestion, necrosis, and eventual rupture or perforation of the intestinal wall, with resultant peritonitis.

Reflux vomiting may be caused by abdominal distention.

Vomiting results in a loss of hydrogen ions and potassium from the stomach, leading to a reduction of chlorides and potassium in the blood and to metabolic alkalosis.

Dehydration and acidosis develop from loss of water and sodium. With acute fluid losses, hypovolemic shock may occur.

**Clinical Manifestations**

The initial symptom is usually crampy pain that is wavelike and colicky.

The patient may pass blood and mucus, but no fecal matter and no flatus. Vomiting occurs.

If the obstruction is complete, the peristaltic waves initially become extremely vigorous and eventually assume a reverse direction, with the intestinal contents propelled toward the mouth instead of toward the rectum. If the obstruction is in the ileum, fecal vomiting takes place.

First, the patient vomits the stomach contents, then the bile-stained contents of the duodenum and the jejunum, and finally, with each paroxysm of pain, the darker, fecal-like contents of the ileum.

The unmistakable signs of dehydration become evident: intense thirst, drowsiness, generalized malaise, aching, and a parched tongue and mucous membranes. The abdomen becomes distended.

The lower the obstruction is in the GI tract, the more marked the abdominal distention.

If the obstruction continues uncorrected, hypovolemic shock occurs from dehydration and loss of plasma volume.

**Assessment and Diagnostic Findings**

Diagnosis is based on the symptoms described previously and on x-ray findings. Abdominal x-ray studies show abnormal quantities of gas, fluid, or both in the bowel. Laboratory studies (ie, electrolyte studies and a complete blood cell count) reveal a picture of dehydration, loss of plasma volume, and possible infection.

**Medical Management**

Decompression of the bowel through a nasogastric or small bowel tube is successful in most cases. E.g in a case of paralytic ileus, treatment involves inserting an NGT down the throat to drain fluids from the stomach. In most cases a partial blockage will not require surgery,but a complete blockage will.Before surgery, intravenous therapy is necessary to replace the depleted water, sodium, chloride, and potassium.

When the bowel is completely obstructed, the possibility of strangulation warrants surgical intervention.

The surgical treatment of intestinal obstruction depends largely on the cause of the obstruction. In the most common causes of obstruction, such as hernia and adhesions, the surgical procedure involves repairing the hernia or dividing the adhesion to which the intestine is attached. In some instances, the portion of affected bowel may be removed and an anastomosis performed.

**Nursing Management**

Nursing management of the nonsurgical patient with a small bowel obstruction includes maintaining the function of the nasogastric tube, assessing and measuring the nasogastric output, assessing for fluid and electrolyte imbalance, monitoring nutritional status, and assessing improvement (eg, return of normal

bowel sounds, decreased abdominal distention, subjective improvement in abdominal pain and tenderness, passage of flatus or stool).

The nurse reports discrepancies in intake and output, worsening of pain or abdominal distention, and increased nasogastric output.

If the patient’s condition does not improve, the nurse prepares him or her for surgery. The exact nature of the surgery depends on the cause of the obstruction.

Nursing care of the patient after surgical repair of a small bowel obstruction is similar to that for other abdominal surgeries (see Chap. 20).

**LARGE BOWEL OBSTRUCTION**

**Pathophysiology**

As in small bowel obstruction, large bowel obstruction results in an accumulation of intestinal contents, fluid, and gas proximal to the obstruction.

Obstruction in the large bowel can lead to severe distention and perforation unless some gas and fluid can flow back through the ileal valve.

Large bowel obstruction, even if complete, may be undramatic if the blood supply to the colon is not disturbed.

If the blood supply is cut off, however, intestinal strangulation and necrosis (ie, tissue death) occur; this condition is life threatening. In the large intestine, dehydration occurs more slowly than in the small

intestine because the colon can absorb its fluid contents and can distend to a size considerably beyond its normal full capacity.

**Clinical Manifestations**

Large bowel obstruction differs clinically from small bowel obstruction in that the symptoms develop and progress relatively slowly.

In patients with obstruction in the sigmoid colon or the rectum, constipation may be the only symptom for days.

Eventually, the abdomen becomes markedly distended, loops of large bowel become visibly outlined through the abdominal wall, and the patient has crampy lower abdominal pain.

Finally, fecal vomiting develops. Symptoms of shock may occur.

**Assessment and Diagnostic Findings**

Diagnosis is based on symptoms and on x-ray studies. Abdominal x-ray studies (flat and upright) show a distended colon. Barium studies are contraindicated.

A rectal tube may be used to decompress an area that is lower in the bowel. The usual treatment, however, is surgical resection to remove the obstructing lesion. A temporary or permanent colostomy may be necessary.

**Nursing Management**

The nurse’s role is to monitor the patient for symptoms that indicate that the intestinal obstruction is worsening and to provide emotional support and comfort. The nurse administers intravenous

fluids and electrolytes as prescribed. If the patient’s condition does not respond to nonsurgical treatment, the nurse prepares the patient for surgery.

This preparation includes preoperative teaching as the patient’s condition indicates.

After surgery, general abdominal wound care and routine postoperative nursing care are provided.

**COLORECTAL CANCER**

* This is the development of cancer from the colon or rectum. A cancer is the abnormal growth of cells that have the ability to invade or spread to other parts of the body.

**Risk factors**

The exact cause is unknown, but the following are risk factors:

* Increasing age
* Family history of colon cancer or polyps
* Previous colon cancer or adenomatous polyps
* History of inflammatory bowel disease
* High-fat, high-protein (with high intake of beef ), low-fiber diet
* Genital cancer or breast cancer (in women)
* Obesity, smoking, lack of physical exercise

**Clinical Manifestations**

The symptoms are greatly determined by the location of the cancer, the stage of the disease, and the function of the intestinal segment in which it is located.

* The most common presenting symptom is a change in bowel habits.
* The passage of blood in the stools is the second most common symptom.
* Symptoms may also include unexplained anemia, anorexia, weight loss, and fatigue.
* The symptoms most commonly associated with right-sided lesions are dull abdominal pain and melena (ie, black, tarry stools).
* The symptoms most commonly associated with left-sided lesions are those associated with obstruction (ie, abdominal pain and cramping, narrowing stools, constipation, and distention), as well as bright red blood in the stool.
* Symptoms associated with rectal lesions are tenesmus (ie, ineffective, painful straining at stool), rectal pain, the feeling of incomplete evacuation after a bowel movement, alternating constipation and diarrhea, and bloody stool.

**Assessment and Diagnostic Findings**

* Along with an abdominal and rectal examination, the most important diagnostic procedures for cancer of the colon are fecal occult blood testing, barium enema, proctosigmoidoscopy, and colonoscopy.

**Complications**

* Tumor growth may cause partial or complete bowel obstruction.
* Extension of the tumor and ulceration into the surrounding blood vessels results in hemorrhage.
* Perforation, abscess formation, peritonitis, sepsis, and shock may occur.

**Medical Management**

* The patient with symptoms of intestinal obstruction is treated with intravenous fluids and nasogastric suction.
* If there has been significant bleeding, blood component therapy may be required.
* Treatment for colorectal cancer depends on the stage of the disease (Chart 38-8) and consists of surgery to remove the tumor, supportive therapy, and adjuvant therapy (chemotherapy and radiotherapy).
* Adjuvant therapy
* Chemotherapy is used before surgery to shrink the tumour before attempting to remove it.
* Radiation therapy is used before, during, and after surgery to shrink the tumor, to achieve better results from surgery, and to reduce the risk of recurrence.

**Surgical Management**

* Surgery is the primary treatment for most colon and rectal cancers.
* It may be curative or palliative. if the cancer is found at an early stage it may be removed through surgery

**DISEASES OF THE RECTUM**

**HEMORRHOIDS**

These are swollen veins in the lowest part of the rectum and anus.

Hemorrhoids are classified as one of two types. Those above the internal sphincter are called internal hemorrhoids, and those appearing outside the external sphincter are called external haemorrhoids.

**Internal haemorrhoids** are far enough inside the rectum that you can’t usually see or feel them.

They don’t generally hurt because there are few pain sensing nerves there. Bleeding may be the only sign of them.

**External haemorrhoids** are under the skin around the anus, where there are many more pain sensing nerves, so they tend to hurt as well as bleed.

Sometimes the haemorrhoids prolapse, or get bigger and bulge outside the anal sphincter. Then you may be able to see them as moist bumps that are pinker than the surrounding area. And they are more likely to hurt, often when you poop.

Prolapsed haemorrhoids usually go back inside on their own. Even if they don’t, they can often be pushed back gently pushed back into place.

A blood clot can form in an external haemorrhoid, turning it purple or blue which is called thrombosis.

**Causes**

A build-up of pressure in the lower rectum can affect the blood flow and make the veins there swell. That may happen from extra weight, when you are obese or pregnant. Or it could come from:

* Pushing down during bowel movements.
* Straining when you do something that’s physically hard, like lifting something heavy.
* People who stand or sit for long stretches of time are at greater risk, too.
* You may get them when you have constipation or diarrhoea that doesn’t clear up. coughing vomiting or sneezing makes them worse.

**Management**

* Hemorrhoid symptoms and discomfort can be relieved by good personal hygiene and by avoiding excessive straining during defecation.
* A high-residue diet that contains fruit and bran along with an increased fluid intake may be all the treatment that is necessary to promote the passage of soft, bulky stools to prevent straining.
* If this treatment is not successful, the addition of hydrophilic bulk-forming agents such as psyllium and mucilloid may help.
* Warm compresses, sitz baths, analgesic ointments and suppositories, astringents (eg, witch hazel), and bed rest allow the engorgement to subside.
* Hemorrhoidectomy, or surgical excision, can be performed to remove all the redundant tissue involved in the process.
* During surgery, the rectal sphincter is usually dilated digitally and the hemorrhoids are removed with a
* clamp and cautery or are ligated and then excised.
* After the operative procedures are completed, a small tube may be inserted through the sphincter to permit the escape of flatus and blood; pieces of Gelfoam or Oxycel gauze may be placed over the anal wounds.

**LIVER DISEASES**

**JAUNDICE**

This is yellow discolouration of the skin and sclera caused by raised levels of bilirubin in the blood.

**Conjugation of bilirubin**

* Conjugation changes the end product of red cells breakdown so they can be excreted in faeces or urine. Ageing, immature or malformed red cells are removed from the circulation and broken down in the reticuloendothelial system (liver, spleen and macrophages)
* Haemoglobin from these cells is broken down to the by-products of haem, globin and iron.
* **Haem** is converted into biliverdin and then to unconjugated bilirubin.
* **Globin** is broken down to amino acids which are used by the body to make proteins.
* **Iron:** is stored in the body or used for formation of new red cells.

Two main forms of bilirubin are present in the body:

**Unconjugated bilirubin:** This is fat soluble and cannot be excreted easily in bile or urine.

**Conjugated bilirubin:** Which has been converted into water soluble in the liver and can be excreted in faeces and urine.

3 stages are involved in the process of bilirubin conjugation: transport, conjugation and excretion.

**Transport of bilirubin**

* Unconjugated or fat soluble bilirubin is transported to the liver bound to protein albumin. If not attached to albumin, this unbound or free bilirubin can be deposited in extravascular fatty and nerve tissues (skin and nerves)
* Skin deposits of unconjugated or fat soluble bilirubin cause jaundice while brain deposits can cause bilirubin toxicity or kernicterus.

**Conjugation**

* Once in the liver, unconjugated bilirubin is detached from albumin combined with glucose and glucuronic acid and conjugation occurs in the presence of oxygen and the enzyme uridinediphosphoglucuronyltransferase (UDP-GT)
* The conjugated bilirubin is now water soluble and available for excretion.

**Excretion**

* Conjugated bilirubin is excreted via the biliary system into the small intestines where normal bacteria change conjugated bilirubin into urobilinogen. This is then oxidized into orange coloured urobilin. Most is excreted in the feces, with a small amount excreted in urine.

**HEPATIC DYSFUNCTION**

* Hepatic dysfunction results from damage to the liver’s parenchymal cells, either directly from primary liver diseases or indirectly from obstruction of bile flow or derangements of hepatic circulation.
* Liver dysfunction may be acute or chronic; chronic dysfunction is far more common than acute.
* Disease processes that lead to hepatocellular dysfunction may be caused by infectious agents such as bacteria and viruses and by anoxia, metabolic disorders, toxins and medications, nutritional deficiencies, and hypersensitivity states.
* The most common cause of parenchymal damage is malnutrition, especially that related to alcoholism.
* The parenchymal cells respond to most noxious agents by replacing glycogen with lipids, producing fatty infiltration with or without cell death or necrosis.
* This is commonly associated with inflammatory cell infiltration and growth of fibrous tissue.
* Cell regeneration can occur if the disease process is not too toxic to the cells.
* The result of chronic parenchymal disease is the shrunken, fibrotic liver seen in cirrhosis.

**JAUNDICE**

* When the bilirubin concentration in the blood is abnormally elevated, all the body tissues, including the sclerae and the skin, become yellow-tinged or greenish-yellow, a condition called jaundice.
* Jaundice becomes clinically evident when the serum bilirubin level exceeds 2.5 mg/dL (43 mmol/L). Increased serum bilirubin levels and jaundice may result from impairment of hepatic uptake,

conjugation of bilirubin, or excretion of bilirubin into the biliary system.

* There are several types of jaundice: hemolytic, hepatocellular, obstructive, or jaundice due to hereditary hyperbilirubinemia.
* Hepatocellular and obstructive jaundice are the two types commonly associated with liver disease.

**Hemolytic /pre-hepatic Jaundice**

* This is caused by anything which causes an increased rate of RBC breakdown. The effect of which is to flood the plasma with bilirubin so rapidly that the liver, although functioning normally, cannot excrete the bilirubin as quickly as it is formed.
* The increased breakdown of RBC leads to an increase in the amount of unconjugated bilirubin present in blood and deposition of this unconjugated bilirubin into various tissues can lead to appearance of jaundice.
* In tropical countries,severe malaria causes this type of jaundice. Certain genetic diseases such as sickle cell anemia.
* The increased production of bilirubin leads to increased production of urine – urobilinogen. Bilirubin is not usually found in the urine because unconjugated is not water soluble, so, the combination of increased urine – urobilinogen with no bilirubin (since, unconjugated) in urine is suggestive of haemolytic jaundice.
* Laboratory findings include:
* Urine: no bilirubin present,urobilinogen>2 units
* Serum: increased unconjugated bilirubin.
* Kernicterus is associated with increased levels of unconjugated bilirubin.

**Hepatocellular/hepatic Jaundice**

* The pathology is located within the liver caused due to disease of the cells of the liver.

**Causes**

* + - Acute or chronic hepatitis.
    - Hepatotoxicity(drugs)
    - Cirrhosis
    - Drug induced hepatitis
    - Alcoholic liver disease
* Cell necrosis reduces the liver’s ability to metabolize and excrete bilirubin leading to a build-up of unconjugated bilirubin in the blood.
* Other causes include primary biliary cirrhosis leading to an increase in plasma conjugated bilirubin because there is impairment of excretion of conjugated bilirubin into the bile.
* The blood contains an abnormally raised amount of conjugated bilirubin and bile salts which are excreted in urine.
* Bilirubin transport across the hepatocyte may be impaired at any point between the uptake of unconjugated bilirubin into the cell and transport of conjugated bilirubin into the canaliculi. In addition, swelling of cells and edema due to inflammation cause mechanical obstruction of intrahepatic biliary tree.
* Hence in hepatocellular jaundice, concentration of both unconjugated and conjugated bilirubin rises in the blood. In hepatocellular disease, there is usually interference in all major steps of bilirubin metabolism-uptake,conjugation and excretion. However, excretion is the rate limiting step and usually is impaired to the greatest extent.
* The unconjugated bilirubin still enters the liver cells and becomes conjugated in the usual way. This conjugated bilirubin is then returned to the blood,probably by rupture of the congested bile canaliculi and direct emptying of the bile into the lymph leaving the liver.
* Thus most of the bilirubin in the plasma becomes the conjugated type, and this conjugated bilirubin which did not go to the intestine to become urobilinogen gives urine the dark colour.
* Laboratory findings depend on the cause of jaundice:
* Urine: conjugated bilirubin present,urobilinogen >2 units

**Obstructive/post hepatic Jaundice**

* The pathology is located after the conjugation of bilirubin in the liver caused due to obstruction of biliary passage. This is caused by an interruption to the drainage of bile containing conjugated bilirubin in the biliary sytem.

Causes

* Gallstones in the common bile duct,pancreatic cancer in the head of pancreas,biliary atresia,pancreatitis.
* In complete obstruction of the bile duct,no urobilinogen is found in urine,since bilirubin has no access to the intestine where it is converted to urobilinogen to be later released into the general circulation. In this case,presence of bilirubin (conjugated) in the urine without urine urobilinogen suggests obstructive jaundice,either intrahepatic or post hepatic.
* Obstructive jaundice of the extrahepatic type may be caused by occlusion of the bile duct by a gallstone, an inflammatory process, a tumor, or pressure from an enlarged organ.
* The obstruction may also involve the small bile ducts within the liver (ie, intrahepatic obstruction), caused, for example, by pressure on these channels from inflammatory swelling of the liver or by an inflammatory
* exudate within the ducts themselves. Intrahepatic obstruction resulting from stasis and inspissation (thickening) of bile within the canaliculi may occur after the ingestion of certain medications, which are referred to as cholestatic agents. These include phenothiazines, antithyroid medications, sulfonylureas, tricyclic antidepressant agents, nitrofurantoin, androgens, and estrogens.
* Whether the obstruction is intrahepatic or extrahepatic, and whatever its cause may be, bile cannot flow normally into the intestine but is backed up into the liver substance.
* It is then reabsorbed into the blood and carried throughout the entire body, staining the skin, mucous membranes, and sclerae.
* It is excreted in the urine, which becomes deep orange and foamy.
* Because of the decreased amount of bile in the intestinal tract, the stools become light or clay-colored. The skin may itch intensely, requiring repeated soothing baths. Dyspepsia and intolerance to fatty foods may develop because of impaired fat digestion in the absence of intestinal bile. AST, ALT, and GGT levels generally rise only moderately, but bilirubin and alkaline phosphatase levels are elevated.

**Signs and symptoms**

* Yellow tinge to the skin,eyes and the mucous membranes
* Pruritis
* Fatigue
* Abdominal pain
* Weight loss
* Vomiting
* Fever
* Pale coloured stools
* Dark urine

**Diagnosis**

* Patient’s history and physical examination,paying close attention to the abdomen;feel for masses in the abdomen and check for firmness of the liver;a firm liver indicates cirrhosis,while a rock hard liver indicates cancer.
* Liver function tests to find out if the liver is functioning properly.
* Blood bilirubin tests
* FHG
* Hepatitis A,B,C tests

If an obstruction in the liver is suspected, the liver structure will be looked at with the help of imaging tests:

* MRI scan: uses magnetic signals to create image “slices” of the soft tissue of the human body.
* Abdominal U/S: uses high frequency sound waves to create two dimensional image of the soft tissue inside the human body.
* CT scan: uses thin X ray beam to create image “slices” of soft tissue in the body.
* Liver biopsy: for checking for inflammation, cirrhosis, cancer and fatty liver.

**Management**

* Treatment typically requires a diagnosis of the specific cause in order to select suitable treatment options. Treatment would then target the cause, rather than the jaundice itself.
* Anemia induced jaundice may be treated by increasing the amount of iron in the blood: either by taking iron supplements or eating more iron rich foods.
* Hepatitis induced jaundice may be treated with anti-viral or steroid medication.
* Obstruction induced jaundice may be treated by surgery to remove the obstruction.
* Medication induced jaundice is treated by selecting an alternative medication and by discontinuing medications that cause jaundice.

**PORTAL HYPERTENSION**

* This is an increase in BP within the portal venous system.
* Veins coming from the stomach, intestine, spleen and pancrease merge into the portal vein, which then branches into the smaller vessels (sinusoids) and then travels through the liver.
* If the vessels in the liver are blocked due liver damage,blood cannot flow properly through the liver. As a result,high pressure in the portal system develops. This increased pressure in the portal vein may lead to the development of large swollen veins (varices) within the esophagus, stomach, rectum or umbilical area.varices can rupture and bleed,resulting in potentially life threatening complications.

**Causes**

* The most common cause is cirrhosis of the liver. This is scarring which accompanies the healing of liver injury caused by hepatitis,alcohol.
* In cirrhosis, the scar tissue blocks the flow of blood through the liver.
* Other causes include blood clots in the portal vein, blockages of the veins that carry the blood from the liver to the heart.

**Symptoms**

* Gastrointestinal bleeding marked by black,tarry stools or blood in the stools,or vomiting of blood due to spontaneous rupture and haemorrhage from the varices.
* Ascites (an accumulation of fluid in the abdomen)
* Encephalopathy or confusion caused by poor liver function.
* Reduced level of platelets, blood cells that help form blood clots, or white blood cells that fight infection.

**Diagnosis**

* This is made on the presence of ascites or dilated veins or varices seen during PE.
* X-rays endoscopic exams may also be used.

**Management**

* Most causes of portal hypertension cannot be treated. Instead treatment focuses on preventing or managing the complications.
* Medications such as antihypertensives may be prescribed to reduce pressure in the varices and further reduce the risk of bleeding.

**Prevention**

* Avoid alcohol and drug use.

ASCITES

This is a condition caused by severe liver disease. It causes excess fluid to build up in the abdomen making it swell and protrude.

**Causes**

Portal hypertension

* This happens when pressure builds up in the veins of the liver and the liver doesn’t work as it should.
* The pressure blocks blood flow in the liver, which over time keeps the kidney from removing excess salt from the body.

kidney and heart failure

* The failure of the liver to metabolize aldosterone increases sodium and water retention by the kidney. Sodium and water retention, increased intravascular fluid volume, and decreased synthesis of albumin by the damaged liver all contribute to fluid moving from the vascular system into the peritoneal space.
* Loss of fluid into the peritoneal space causes further sodium and water retention by the kidney in an effort to maintain the vascular fluid volume, and the process becomes self-perpetuating.
* As a result of liver damage, large amounts of albumin-rich fluid, 15 L or more, may accumulate in the peritoneal cavity as ascites.
* With the movement of albumin from the serum to the peritoneal cavity, the osmotic pressure of the serum decreases.
* This, combined with increased portal pressure, results in movement of fluid into the peritoneal cavity.
* This in turn causes fluid to build up.

**Symptoms**

* Feeling of fullness
* Rapid weight gain
* Shortness of breath
* Swelling in the legs and ankles
* Indigestion
* Vomiting
* Heartburn
* Loss of appetite
* Infection
* Hernia

**Diagnosis**

* PE are assessed by percussion of the abdomen. When fluid has accumulated in the peritoneal cavity,

the flanks bulge when the patient assumes a supine position.

* The presence of fluid can be confirmed either by percussing for shifting dullness or by detecting a fluid wave (,ultra sound or a CT scan
* Paracentesis(fluid is removed from the abdomen)

**Medical Management**

**Dietary Modification**

* The goal of treatment for the patient with ascites is to reduce sodium to reduce fluid retention. Cut down on the amount of salt consumed. Diuretics may be prescribed to flush extra fluids in the body. They help the kidneys remove sodium and water.

**Paracentesis**

* Paracentesis is the removal of fluid (ascites) from the peritoneal cavity through a small surgical incision or puncture made through the abdominal wall under sterile conditions.
* Use of large-volume (5 to 6 litres) paracentesis has been shown to be a safe method for treating patients with severe ascites.
* The procedure provides only temporary removal of fluid; it rapidly recurs, necessitating repeated removal.

**ESOPHAGEAL VARICES**

* These are abnormal, enlarged veins in the esophagus.

**How it occurs??**

* They develop when the normal blood flow to the liver is blocked by a clot or scar tissue in the liver.to go around the blockages, blood flows into smaller blood vessels that aren’t designed to carry large volumes of blood.
* The vessels can leak blood or even rupture, causing life threatening bleeding.

**Causes**

* Liver cirrhosis-liver diseases such as hepatitis infection, alcoholic liver disease, fatty liver disease, bile duct disorder.
* Blood clot in the portal vein and its branches.
* Parasitic infection (schistosomiasis) whereby liver flukes block the portal vein

**Symptoms**

Don’t usually cause symptoms unless they bleed. They include:

* Vomiting and seeing significant amounts of blood in the vomitus.
* Black, tarry or bloody stools.
* Light-headedness.
* Loss of consciousness

**Diagnosis**

* Upper endoscopy
* Ct scan of the splenic and portal vein

**Management**

* The main aim is to prevent bleeding,
* Give anti-hypertensive, blood transfusion to replace lost blood, prophylactic antibiotics, liver transplant to replace the diseased one.

**ESOPHAGEAL VARICES**

* Bleeding or hemorrhage from esophageal varices occurs in approximately one third of patients with cirrhosis and varices.

**Pathophysiology**

* Esophageal varices are dilated, tortuous veins usually found in the sub-mucosa of the lower esophagus, but they may develop higher in the esophagus or extend into the stomach.
* This condition nearly always is caused by portal hypertension, which in turn is due to obstruction

of the portal venous circulation within the damaged liver.

* Because of increased obstruction of the portal vein, venous blood from the intestinal tract and spleen seeks an outlet through collateral circulation (new pathways of return to the right atrium).
* The effect is increased pressure, particularly in the vessels in the sub-mucosal layer of the lower esophagus and upper part of the stomach.
* These collateral vessels are not very elastic but rather are tortuous and fragile and bleed easily.
* Less common causes of varices are abnormalities of the circulation in the splenic vein or superior vena cava and hepatic venothrombosis.
* Bleeding esophageal varices are life-threatening and can result in hemorrhagic shock, producing decreased cerebral, hepatic, and renal perfusion.
* In turn, there is an increased nitrogen load from bleeding into the GI tract and an increased serum ammonia level, increasing the risk for encephalopathy.
* Usually the dilated veins cause no symptoms unless the portal pressure increases sharply and the mucosa or supporting structures become thin.
* Then massive hemorrhage takes place.
* Factors that contribute to hemorrhage are muscular exertion from lifting heavy objects; straining at stool; sneezing, coughing, or vomiting; esophagitis; irritation of vessels by poorly chewed foods or irritating fluids; or reflux of stomach contents (especially alcohol).
* Salicylates and any medication that erodes the esophageal mucosa or interferes with cell replication also may contribute to bleeding.

**HEPATITIS**

* This is a systemic, viral infection in which necrosis and inflammation of liver cells. The condition can be self-limiting or can progress to fibrosis (scarring), cirrhosis or liver cancer.
* This is inflammation of the liver. It is usually the result of a viral infection or liver damage caused by drinking. There are several different types of hepatitis. some types will pass without any serious problems, while others can be long lasting (chronic) and cause scarring of the liver (cirrhosis),loss of liver function and in some cases liver cancer.

**Causes**

Hepatitis viruses

There are 5 main types of hepatitis viruses, referred to as type A,B,C,D and E. the 5 types are of greatest concerns because of the burden of illness and death they cause and the potential for outbreaks and epidemic spread.

Infections

Toxic substances (alcohol and certain drugs)

Autoimmune diseases

**HEPATITIS A**

* This is caused by the Hepatitis A virus.

MOT: consuming food and drink contaminated with poo of an infected person and is most common in places of poor sanitation.

* Eating food prepared by someone with the infection who hasn’t washed their hands or properly or washed them in contaminated with sewage.
* Drinking contaminated water.
* Close contact with someone who has Hepatitis A.
* Less commonly, having sex with someone who has the infection especially homosexuals.
* Someone with hepatitis A is most infectious from around two weeks before symptoms appear until about a week after the symptoms develop.
* The disease is self-limiting within two months. There is no specific treatment for it other than to relieve symptoms such as pain, nausea and itching.
* Vaccination against hepatitis A is recommended if travelling to endemic areas.

**Symptoms include:**

* Feeling tired and generally unwell.
* Joint and muscle pain.
* Loss of appetite.
* Feeling or being sick.
* Pain in the upper right part of the abdomen.
* Jaundice.
* Dark urine and pale stools
* Itchy skin.

**Diagnosis**

* Hepatitis A antigen may be found in the stool a week to 10 days before illness and for 2 to 3 weeks after symptoms appear.
* HAV antibodies are detectable in the serum, but usually not until symptoms appear.

**Vaccination against hepatitis A**

* Recommended for people at an increased risk including:
* Close contact of someone with Hepatitis A.
* People planning to travel or live in parts of the world where Hepatitis A is widespread.
* People with any type of long term liver disease.
* Homosexuals.
* People who inject illegal drugs.
* People who may be exposed to Hepatitis through their job (sewage workers, people working with monkeys, apes and gorillas,)

**Treatment for Hepatitis A**

There is no currently no cure for Hepatitis A, but it will normally pass on its own within a couple of months. You can usually look after yourself at home.

While you are ill, it’s a good idea to:

* Get plenty of rest.
* Take painkillers for any aches and pain.
* Maintain a cool, well ventilated environment, wear loose clothing, and avoid hot baths or showers to reduce any itching.
* Eat smaller, lighter meals to reduce nausea and vomiting.
* Avoid alcohol to reduce the strain on your liver.
* Stay off work or school and avoid having sex until at least a week after jaundice and other symptoms have started.
* Practise good hygiene measures, such as washing your hands with soap and water regularly.

**HEPATITIS B**

* This is an infection of the liver caused by Hepatitis B virus that’s spread through blood and body fluids.

MOT: the HBV is found in the blood and body fluids such as semen and vaginal fluids of an infected person.

* Infected pregnant women to their babies.
* Within families (Child to child contact) where the infection is common..
* Unprotected sex.
* Injecting drugs and sharing needles.
* Having a tattoo, body piercing or medical or dental equipment in an unhygienic environment with unsterilized equipment.
* Sharing tooth brushes or razors contaminated with infected blood.

HBV is not spread by kissing, holding hands, hugging, coughing, sneezing or sharing utensils.

NB: most adults infected with hepatitis B are able to fight off the virus and fully recover from the infection within a couple of months.

However, most people infected as children develop a long term infection. This is known as chronic hepatitis B and it can lead to cirrhosis and liver cancer. Antiviral medication can be used to treat it.

**Symptoms**

Many people with Hepatitis B wont experience any symptoms any symptoms and may fight off the virus without realising they had it.

If symptoms do develop, they tend to occur two or three months after exposure to the virus which includes:

* Flu like symptoms, including tiredness, fever, general aches and pains.
* Loss of appetite.
* Feeling and being sick.
* Diarrhoea
* Abdominal pain.
* Jaundice.

These symptoms will usually pass within one to three months (acute hepatitis B), although occasionally the infection can last for 6 months (chronic hepatitis B)

**Diagnosis**

* HBV antibodies are detectable in the serum.

**Management**

Treatment for Hepatitis B depends on how long you’ve been infected:

**If you’ve been exposed to the virus in the last few days,** emergency treatment can help stop becoming infected. This involves giving Hepatitis B vaccine at 0, 1 month and 6 months.

Hepatitis B immunoglobulin- a preparation for antibodies that work against the hepatitis B virus and can offer immediate but short term protection until the vaccine starts to take effect. Effective within 48 hours to one week after exposure.

**If you’ve had the infection for a few weeks or months (acute hepatitis B),** you may only need treatment to relieve symptoms while the body fights off infection.

Get plenty of rest.

Take painkillers for any aches and pain.

Maintain a cool,well ventilated environment,wear loose clothing, and avoid hot baths or showers to reduce any itching.

Take medications such as metoclopramide to stop you feeling sick and chlorphenaramine to reduce itching.

Most people completely recover in a couple of months,but it is advisable to have regular blood tests to check if you are free of the virus and haven’t developed chronic Hepatitis B.

**If you’ve had the infection for more than six months (chronic hepatitis B),** you may be offered treatment with medication that keeps the virus under control and reduce the risk of liver damage. Although they won’t necessarily cure the infection and some people need life-long treatment.medications include:

Peginterferon alfa 2a which stimulates the immune system to attack the HBV and regain control over it. It is usually given by injection once a week for 48 weeks.

Antiviral medications such as tenofovir or entecavir which are taken as tablets.

**Living with HBV:**

If you have hepatitis, you should;

Avoid having unprotected sex including anal and oral sex, unless you are sure your partner has been vaccinated against hepatitis B.

Avoid sharing needles used to inject drugs with other people.

Take precautions to avoid spread of infection-such as not sharing toothbrushes or razors with other people; close contacts such as family members may need to be vaccinated.

Avoid drinking alcohol-this can increase your risk of developing serious liver problems.

**Prevention**

Vaccination is available for people at high risk of the infection who include:

* Babies born to infected mothers
* Close family and sexual partners of someone with hepatitis B.
* People travelling to or from a part of the world where HBV is widespread such as Africa, Asia.
* People who inject drugs or have a sexual partner who injects drugs.
* People who change their sexual partners frequently.
* Homosexuals.

**HEPATITIS C**

* This is caused by the HCV.it is usually spread through blood to blood contact with an infected purpose.

MOT:

The HCV is usually spread through blood to blood contact.

Some ways the infection can be spread include:

* Sharing unsterilized needles by drug users.
* Sharing razors or tooth brushes.
* From a pregnant woman to her unborn baby.
* Rarely; through unprotected sex.

**Diagnosis**

Blood test for HCV.

**Management**

Pegylated interferon (a weekly injection) and ribavirin

**Prevention**

There is no vaccine for HCV but there are ways to reduce the risk of becoming infected: these include:

* Not sharing any drug injecting equipment with other people.
* Not sharing razors or tooth brushes that might be contaminated with blood.
* The risk of getting hepatitis C through sex is very low. However it may be higher if blood is present, such as menstrual blood or from minor bleeding during anal sex.

**HEPATITIS D**

* This is caused by the HDV.it only affects people who are already infected with hepatitis B, as it needs the hepatitis B virus to be able to survive in the body.

MOT: blood to blood contact or sexual contact.

There is no vaccine specifically for hepatitis D, but the Hepatitis B vaccine can help protect you from it.

**HEPATITIS E**

This is caused by the HEV.

MOT: consuming food and drink contaminated with the poo of an infected person.

The infection is a generally mild and short term infection that doesn’t require any treatment.

There is no vaccine for HEV but you can reduce the risk by practising good food and water hygiene measures

**NON VIRAL HEPATITIS**

**TOXIC HEPATITIS**

* This is inflammation of the liver in reaction to certain substances a person is exposed. Develops within hours or days of exposure to a toxin. Sometimes it may take months of regular use before signs and symptoms appear.

**Signs and symptoms**

* Jaundice
* Itching
* Abdominal pain in the right portion of the abdomen.
* Fatigue.
* Loss of appetite.
* Rash
* Weight loss
* Dark coloured urine.

**Causes**

Toxic hepatitis occurs when the liver develops inflammation because of exposure to a toxic substance.

The liver normally removes and breaks down most drugs and chemicals from the bloodstream. Breaking down toxins creates by products that can damage the liver. Although the liver has a great capacity for regeneration, constant exposure to toxic substances can cause serious, sometimes irreversible harm.

Alcohol: heavy drinking over the years can lead to alcoholic hepatitis.

Over the counter pain killers: especially paracetamol, brufen, aspirin can damage the liver especially if taken frequently or combined with alcohol.

Herbs and supplements

Industrial chemicals.

**Diagnosis**

Physical exam and history taking: in order to know all medication and herbs being taken,if you are working with industrial chemicals.

Blood tests look for certain liver enzymes.

Imaging tests

Liver biopsy

**Management**

Stopping exposure to the toxin causing liver inflammation will reduce signs and symptoms.

Liver transplant if severely impaired.

Autoimmune hepatitis

In some cases, the immune system mistakes the liver as a harmful object and begins to attack it. This causes on-going inflammation that can range from mild to severe often hindering liver function.

**LIVER CIRRHOSIS**

* This is a situation in which scar tissue (fibrosis) replaces healthy tissue. This scar tissue prevents the liver from working as it should. This is a complication of many liver diseases characterized by abnormal structure and function of the liver.
* The diseases that lead up to cirrhosis do so because they injure and kill liver cells, after which the inflammation and repair that is associated with the dying liver cells causes scar tissue to form.

**Effects**

* The relationship between blood and liver cells is destroyed.
* Obstructs blood flow through the liver and to the other liver cells.
* Disturbed relationship between liver and the channels through which bile flows.(canaliculi)
* Digestion in the intestine is reduced.

**Causes**

* Long term, heavy use of alcohol.
* Chronic viral hepatitis.
* Autoimmune disease of the liver.
* Blocked bile ducts. (primary biliary cirrhosis)
* Idiopathic.
* Signs and symptoms
* Fatigue
* Easy bruising from decreased production of blood clotting factors by the diseased livers.
* Jaundice
* Weight loss.
* Edema and ascites.
* Bleeding from enlarged veins in the digestive tract.
* Confusion

**Diagnosis**

* Physical examination and history taking may uncover history of excessive and prolonged intake of alcohol, history of hepatitis. A cirrhotic liver feels firm and irregular than normal liver.
* Abdominal ultrasound, CT scan, tissue biopsy.
* Blood tests reveals reduced level of albumin in the blood and reduced blood clotting factors, abnormal elevation of liver enzymes.

**Management**

Treatment of cirrhosis includes 3 factors:

* Preventing further damage to the liver.
* Treating the complications of cirrhosis.
* Preventing liver cancer or detecting it early.
* Liver transplantation.

**Prevent further damage to the liver the following are recommended:**

* Consume a balanced diet and multivitamins daily because there is impairment of fat soluble vitamins may need additional vitamin D and K.
* Avoid alcohol/drugs that cause liver damage.
* Eradicate viral hepatitis by using antiviral medications.
* Suppress the immune system with corticosteroids and immunosuppressant to decrease inflammation of the liver in autoimmune hepatitis.
* Immunize patients with cirrhosis against infection with hepatitis A and B.

**Treatment of complications of hepatitis.**

* Edema and ascites: restrict sodium intake, give diuretics to promote elimination of salt and water into the urine.
* Bleeding from varices: anti-hypertensives.
* Peritonitis from ascites give prophylactic antibiotics.

**Prevention and early detection of liver cancer.**

* By early screening when patients presents with cirrhosis,treatment of liver diseases,liver transplant if extensively damaged.

**Liver transplantation:**

* Cirrhosis is irreversible,in advanced cases liver transplantation is the only option for treatment.

**Disorders of the Gallbladder**

* Several disorders affect the biliary system and interfere with normal drainage of bile into the duodenum. These disorders include inflammation of the biliary system and carcinoma that obstructs the biliary tree. Gallbladder disease with gallstones is the most common disorder of the biliary system.
* Although not all occurrences of gallbladder inflammation (**cholecystitis**) are related to gallstones (**cholelithiasis**), more than 90% of patients with acute cholecystitis have gallstones.

**CHOLECYSTITIS**

* This is inflammation of the gallbladder. Inflammation is the body’s response to injury. It is a process by which the body’s white blood cells and substances they produce protect us from infection with foreign organisms, such as bacteria and viruses.

**Causes**

* The causes can be grouped into two main categories:
  + - Calculous and acalculous cholecystitis.

**Calculous cholecystitis**

* This is the most common and usually less serious type. Accounts for 95% of cases. This develops when the main opening to the gallbladder, called the cystic duct, gets blocked by a gallstone or a substance known as biliary sludge. This is a mixture of bile and salt crystals.
* The blockage in the cystic duct causes bile to build up in the gallbladder, increasing pressure inside it and causing it to become inflamed.
* A gallstone stuck in the cystic duct. This blocks fluid from passing out of the gallbladder. This results in an irritated and swollen gallbladder.

**Acalculous cholecystitis**

* This is less common, but usually the more serious type. It usually develops as a complication of a serious illness, infection or injury that damages the gallbladder.
* Can be caused by accidental damage to the gallbladder during major surgery, serious injuries or burns, blood poisoning (sepsis),severe malnutrition or AIDS
* Infection or trauma, such as an injury from a car accident, can also cause.

**Symptoms**

* Pain in the upper right abdomen that sometimes move around to the back or right shoulder.
* Nausea or vomiting.
* Tenderness in the right abdomen.
* Fever
* Pain gets worse during a deep breathe.
* Pain for more than 6 hours, particularly after meals.

**Diagnosis**

* Physical examination
* Ultrasound which reveals gallstones, thickening of the gallbladder wall,extra fluid.
* Blood tests: FHG to check for signs of inflammation in the body.

**Management**

* Resting the bladder by not eating or drinking.
* Receiving fluids through a drip directly into a vein to prevent dehydration.
* Taking painkillers.
* Antibiotics if the cause was infection.
* After initial treatment, any gallstones that may have caused acute cholecystitis usually fall back into the gallbladder and the inflammation will always settle.

**Possible complications**

* The death of gallbladder tissue (gangrenous cholecystitis)- which can cause a serious infection that could spread throughout the body.
* The gallbladder splitting open (perforated bladder)-which can spread infection within the abdomen (peritonitis) or lead to a build-up of pus (abscess)

**Prevention**

* Adopt a healthy balanced diet and reduce the number of high cholesterol foods you eat, as it is thought to contribute to the formation of gallstones.
* Control weight within normal limits.

**CHOLELITHIASIS/GALLSTONES**

* They aren’t really stones. They are pieces of solid material that form in the gallbladder.
* These are small stones usually made of cholesterol, that form in the gallbladder. In most cases they don’t cause any symptoms and don’t need to be treated.
* However if a gallstone becomes trapped in an opening (duct) inside the gallbladder, it can trigger a sudden, intense abdominal pain that usually lasts between one and five hours. This type of pain is known as biliary pain.

**Types**

There are two main kinds:

**Cholesterol stones**: These are usually yellow green in colour. They are the most common type.

**Pigmented stones**: These stones are smaller and darker. They are made up of bilirubin, which comes from bile.

**Causes**

* There may-be several reasons; including’ genes, weight, problems with the gallbladder, diet.
* Bile can be part of the problem. The body needs bile, but if it has too much cholesterol in it, that makes gallstones likely.
* It can also happen if the gallbladder can’t empty properly.
* Pigmented stones are more common in people with certain medical conditions, such as cirrhosis and sickle cell disease.

**Risk factors**

* Obesity: raises cholesterol levels and also make it harder for the gallbladder to empty completely.
* DM: people with this condition tend to have high level of cholesterol.
* Losing weight too quickly: liver makes extra cholesterol

**Symptoms**

* Pain in the upper belly and upper back that can last for several hours.
* Nausea
* Vomiting
* Digestive problems as: bloating, indigestion, heart burn and gas.

**Diagnosis**

* Blood tests to check for signs of infection or obstruction
* U/S to see the bladder.
* CT scan/MRI to visualize the liver and gallbladder.

**Management**

* Surgery to get out the gallbladder.

**PANCREATITIS**

* This is inflammation of the pancreas. Pancreatic damage happens when the digestive are activated before they are released into the small intestine and begin attacking the pancreas.

Pancreatitis is categorised as being either acute or chronic.

**Acute pancreatitis:** Develops suddenly, and it is usually a short term (a few days to weeks) illness that typically resolves with appropriate medical management.

**Chronic pancreatitis**; develops after multiple episodes of acute pancreatitis, isa long term condition that can last for months or even several years.

**Causes**

Normally digestive enzymes released by the pancreas are not activated to break down fats and proteins until they reach the small intestine. However, when these digestive enzymes are activated while still in the pancreas, inflammation and local damage to the pancreas occurs leading to pancreatitis. Causes include:

* Alcohol consumption
* Gallstones
* High triglyceride levels
* Abdominal injury or surgery.
* Certain medications.
* Exposure to certain chemicals.
* Smoking.
* Family history of pancreatitis.
* Pancreatic cancer.

**Signs and symptoms**

* Upper abdominal pain which can range from mild to severe.
* The pain may come on suddenly or it may develop gradually. often the pain will start or worsen after eating, which can also occur with gall bladder or ulcer pain.

**S&S of acute pancreatitis may include:**

* Abdominal pain that may radiate to the back, it may be aggravated by eating, especially foods high in fat.
* Nausea and vomiting.
* Worsening pain after eating.
* Tenderness to touch of the abdomen.
* Swollen abdomen.
* Fever and chills.
* Weakness and lethargy.

In chronic pancreatitis, abdominal pain also can be present but it is often not as severe, and some people may not have any pain at all.

**Signs and symptoms of chronic pancreatitis may include:**

* Abdominal pain
* Foul smelling oily stool.

**Diagnosis**

* Liver and kidney functions tests.
* FHG (anaemia and WBC)
* CT scan: of the abdomen may be ordered to visualize the pancreas and to evaluate the extent of inflammation, may also detect gallstones and biliary system.
* U/Scan maybe used to look for gallstones and abnormalities of the biliary system.
* Tissue biopsy.

**Management**

* Depending on the underlying cause of pancreatitis, management may vary to address the specific cause.
* Fasting to help the pancreas to rest and recover.
* IVF to prevent dehydration while fasting. pain medications as it can be very painful.
* If pancreatitis is due to an obstructing gallstone, surgical intervention may be required to remove gallstone and or remove the gallbladder.
* If alcohol consumption is the cause, abstain.
* If medications or a chemical exposure is found to be the cause, then removal of medication or offending exposure is recommended.
* Medication to control nausea.
* Low fat diet that is high in nutrients is recommended.

**Complications**

* Diabetes: due to disruption in the secretion of insulin.
* Malnutrition: damage to the pancreas can lead to a decrease or absence of digestive enzymes produced, which can affect the absorption of various nutrients. This may lead to malnutrition and unintentional weight loss.

**Pancreatic cancer.**

* Cancer that develops within the pancreas.

Can be divided into two major categories

**Cancer of the endocrine pancreas** (the part that makes insulin and other hormones) they are referred to as the islet cell cancers.

**Cancer of the exocrine pancreas** (the part that makes enzymes) these develop from the cells that line the system of ducts that deliver enzymes to the small intestine and are commonly referred to as pancreatic adenocarcinomas.

**Causes and risk factors**

* Increasing age above 60 years.
* Tobacco use.
* Obesity.
* Diabetes.
* Sedentary lifestyle.
* Pancreatitis.
* Fatty diet.
* Chronic infections such as Hepatitis B and H.pylori

**Signs and symptoms**

In general the signs and symptoms of pancreatic cancer can be produced by exocrine or endocrine cancer cells.

Exocrine pancreatic cancer signs and symptoms can include:

* Jaundice
* Dark urine
* Itchy skin
* Light coloured stools.
* Pain in the abdomen or back.
* Poor appetite and weight loss.
* Digestive problems characterised by pale and greasy stools, nausea and vomiting.
* Blood clots.
* Enlarged gallbladder.

The signs and symptoms of endocrine pancreatic cancers are often related to the excess hormones that they produce and consequently to a variety of different symptoms:

**Insulinomas:** these are insulin producing tumours that lowers blood glucose levels can cause low blood sugars, weakness, confusion, coma and even death.

Glucagonomas: glucagon producing tumours can increase glucose levels and can cause symptoms of diabetes that is thirst, increased urination, diarrhoea and skin changes.

**Diagnosis**

* CT scan, ultra sounds, MRI and endoscopy.

**Management**

* If pancreatic cancer is found at an early stage and is contained locally within or around the pancreas, surgery maybe recommended. If it has metastasised to nearby structures chemotherapy and radiotherapy is recommended to minimize the symptoms.