Short course of surgery

General surgery

الطب والجراحة لدنة

عَبْدي إذا ما الهَمُّ أرخى وِزرَهُ فالبَذلُ يُشفي مِنْ مَآسي ضُرَّهُ
خَيْرُ الأيادى مَنْ سَخَتْ في بَذلها مِنْ فضل رَبي ذاكَ فِعْلٌ سَرَّهُ

هذا ما آمن به الفريق الأكاديمي- لجنة الطب و الجراحة دوماً ..و على ذاك عاهدت نفسها .. و الآن تفي بوعدها ..و تضع بين أيديكم دوسية الجراحة الجديدة .. نسخة جديدة منقحة ومحدثة تشمل جميع مواضيع الخطة بشقيها خطة السنة الرابعة والسادسة شاملة للمعلومات التي ذكرها المدرّسون في محاضراتهم ومدقّقة.. لا نتزهها من الخطأ لكننا بذلنا في سبيلها الكثير من الأوقات والجهود لكي نضع بين يديكم أكمل ما نستطيع ..

فلكل من ساهم بإنجاح هذا العمل له منّا و باسمكم جزيل الشكر و العرفان و نخص بالشكر أولا ياسر لافي وأبرار الصرايرة على ما بذلوه من جهود كبيرة في التخطيط والتنظيم والتنسيق ونشكر أيضا كل من ساهم في هذا العمل من كتابة مواضيع وتلخيصها وتدقيقها وتاليا أسماؤهم :

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ACID-BASE

ACID-BASE CONSIDERATIONS

Nearly all biochemical reactions in the body are dependent on maintenance of a physiological hydrogen ion concentration. In any aqueous solution, water molecules reversibly dissociate into hydrogen (H+) and hydroxide ions (OH-). pH (the hydrogen number) is determined by the balance between the two and usually reflects the hydrogen ion concentration [H+]. A pH in general is affected by the balance between positive and negative charges. Neutral pH is 7. the Blood is a bit alkaline and has a pH of (pH 7.35-7.45).

ACID-BASE BALANCE

[H+] in the body and specifically the blood is vital and critical in effect as it determines the function of many enzymes in the body, include those involved in the respiratory process, the cardiac tissue and those involved in the clotting cascade.

The hydrogen ion is thus closely regulated because changes in [H+] produce widespread organ dysfunction. This regulation often referred to as acid-base balance is of prime importance.

Hydrogen ion is generated by daily metabolism including oxidation of amino acids and anaerobic metabolism of glucose to lactic and pyruvic acid 1 mEq/kg per day. Also a large amount is produced by the introduction carbon dioxide (as a result of aerobic metabolism) into the blood and formation bicarbonic acid (15 mEq/day). the last source of hydrogen ion is closely regulated by respiration.

Physiological responses to changes in [H+] are characterized by three phases: (1) immediate chemical buffering, (2) respiratory compensation (whenever possible), and (3) a slower but more effective renal compensatory response that may nearly normalize arterial pH even if the pathological process is still present.

IMMEDIATE CHEMICAL BUFFERING

A buffer system is a solution that resists the changes in pH. Physiologically important buffers in humans include bicarbonate (H2CO3/HCO3-), hemoglobin (HbH/Hb-), other intracellular proteins (PrH/Pr-), phosphates (H2PO4-/HPO42-), and ammonia (NH3/NH4+). Acid loads can also demineralize bone and release alkaline compounds (CaCO3and CaHPO4). Alkaline loads (NaHCO3) increase the deposition of carbonate in bone.

Buffering by plasma bicarbonate is almost immediate, while other buffers are slower. Thus in addition to the fact that bicarbonate (HCO3-) is present in relatively high concentrations in extracellular fluid, this makes it the most important in the body.

RESPIRATORY COMPENSATION

Changes in alveolar ventilation responsible for pulmonary compensation of PaCO2 are mediated by chemoreceptors within the brain stem. This is achieved by changing the rate of ventilation, the lungs are responsible for eliminating the approximately 15 mEq of carbon dioxide produced every day as a product of carbohydrate metabolism.

Decreases in arterial blood pH (acidemia) stimulate medullary respiratory centers. The resulting increase in alveolar ventilation lowers PaCO2 and tends to restore arterial pH toward normal. PaCO2 normally decreases 1-1.5 mmHg below 40 mmHg for every 1 mEq/L decrease in plasma [HCO3-].

Increases in arterial blood pH (alkalemia) depress respiratory centres. The resulting alveolar hypoventilation tends to elevate PaCO2 and restore arterial pH toward normal. As a general rule, PaCO2 can be expected to increase 0.25-1 mmHg for each 1 mEq/L increase in [HC03-].

RENAL COMPENSATION

The ability of the kidneys to control the amount of HCO3- reabsorbed from filtered tubular fluid, form new HCO3_, and eliminate H+ in the form of titratable acids and ammonium ions allows them to exert a major influence on pH. In fact, the kidneys are responsible for eliminating the approximately 1 mEq/kg per day of incompletely oxidized organic acids that are normally produced by the metabolism of dietary and endogenous proteins. The renal response to acidaemia is (1) increased reabsorption of the filtered HCO3-, (2) increased excretion of titratable acids, and (3) increased production of ammonia.

might otherwise have been excreted are reabsorbed, resulting in metabolic alkalosis) and (2) those associated with potassium depletion (hypokalaemia augments H+ secretion (and HCO3- reabsorption) and will also propagate metabolic alkalosis and (3) those that occurs due to gastric juice loss and thus hydrogen ion depletion (such as occurs in pyloric stenosis, gastric outlet obstruction and continuous vomiting).

As with other acid-base disorders, correction of metabolic alkalosis is never complete until the underlying disorder is treated. The treatment of choice for metabolic alkalosis due to fluid depletion is administration of intravenous saline (NaCl) and potassium (KCI).

FLUIDS THERAPY

The relationship between total body weight and total body water (TBW) is relatively constant for an individual and is primarily a reflection of body fat. The highest percentage of TBW is found in newborns, with approximately 80% of their total body weight composed of water. This decrease to about 65% by 1 year of age and thereafter remains fairly constant. The body of a healthy 70 kg male contains about 42 litres of water. That is, total body water constitutes about 60% of his total body weight. Lean tissues such as muscle and solid organs have higher water content than fat and bone. As a result, young, lean males have a higher proportion of body weight as water than elderly or obese individuals. An average young adult male will have 60% of his total body weight as TBW, while an average young adult female's will be ~55%.

TBW is divided into two functional fluid compartments, the extracellular and intracellular. The extracellular fluid compartment comprises about one third of the TBW and the intracellular compartment the remaining two thirds. The extracellular water comprises 20% of the total body weight and is divided between plasma (¼ of the extracellular water volume, or 5% of body weight; also referred to as the intravascular compartment) and interstitial fluid (¾ of the extracellular water volume, or 15% of body weight). Intracellular water makes up approximately 40% of an individual's total body weight.

The osmolality of extracellular fluid is determined primarily by sodium and chloride concentrations. The following equation is a calculation of the serum osmolality;

Osmolality= 2 sodium + glucose/18 +BUN/2.8

Osmotic pressure is measured in units of osmoles (osm) or milliosmoles (mOsm) that refer to the actual number of osmotically-active particles. For example, one millimole (mmol) of sodium chloride contributes to 2 mOsm (one from sodium and one from chloride). The principal determinants of osmolality are the concentrations of sodium, glucose, and urea (blood urea nitrogen [BUN]); whereas the major intracellular ions are potassium, magnesium, phosphate and sulphate. The osmolality of the intracellular and extracellular fluids is maintained between 290 and 310 mOsm/kg water in each compartment. Because cell membranes are permeable to water, any change in osmotic pressure in one compartment is accompanied by a redistribution of water until the effective osmotic pressure between compartments is equal.

Sodium is confined to the extra-cellular fluid compartment, and because of its osmotic and electrical properties, it remains associated with water. Therefore, sodium-containing fluids are distributed throughout the extracellular fluid and add to the volume of both the intravascular and interstitial spaces.

The distribution of electrolytes is maintained by membrane ion pumps) which is essential for normal cellular function, and is an energy-dependent process that uses a significant proportion of basal energy requirements.

The distribution of fluid between the intra- (the plasma) and extravascular compartments (interstitial fluids) is dependent on the oncotic pressure of plasma and the permeability of the endothelium. The plasma oncotic pressure is determined by the presence of colloid particles, of which albumin is the most important.

REGULATION OF WATER BALANCE

The kidney is the most important organ in the regulation of water balance. It can control the amount of water by controlling the amount of its excretion. This is achieved by controlling the amount of water filtration and reabsorption. The amount of filtration can be controlled by the hormone angiotensin ll which increases filtration in cases of kidney hypoperfusion. The amount of reabsorption can be controlled by the hormones aldosterone and ADH. Both Aldosterone and ADH increases water retention, except that in the former, sodium is conserved,

and in the latter (because only water is reabsorbed), sodium is diluted.

The hormone angiotensin II is released in response to the renin-angiotensin system which is activated by hypo-perfusion to the kidney. Aldosterone is released in response to the hormone angiotensin and an increase in the serum potassium. The hormone ADH is released from the posterior pituitary in response to stimulation of the hypothalamus by a decrease in water volume or an increase in serum osmolality.

INPUT-OUTPUT

The normal daily input-of water is about 2000 ml of water, an additional 200-300 ml per 24 hours is provided endogenously by oxidation of carbohydrate and fat (i.e. metabolic water). This is consistent with daily water losses (output). A healthy individual loses fluid and electrolytes by three routes: via the kidneys, via the gastrointestinal tract, and by evaporation from the skin and respiratory tract (insensible water loss). The table above presents the amount of fluid and electrolytes that are lost in-a 24 hours period from these routes.

normal daily output			
Route of loss	**volume (ml)**	**Na+ (mEq)**	**K+ (mEq)**
Urine	**1500**	90	**60**
Insensible losses (skin and respiratory tract)	**700**		
Feces	**200**	**-**	**10**
Total	**2400**	**90**	**70**

Almost all sodium loss is via the urine (90 mEq/24 hrs). Potassium is also excreted mainly via the kidney (60- 100 mEq/24 hrs) and about 10 mEq /day is lost via the gastrointestinal tract.

Pyrexia increases water loss from the skin by approximately 200 ml/day for each 1 °C rise temperature. Sweating increases fluid loss considerably, by up to 1/ litre per hour, but is difficult to quantify. Sweat contains significant amounts of sodium (20-70 mEq/l) -and potassium (10 mEq/l), which should be considered when assessing losses.

Hyperventilation increases insensible water loss via the respiratory tract. This should occur when the normal humidifying mechanisms of the respiratory tracts are overcame by extremely dry air (e.g. ventilators with non-humidified air).

THE EFFECT OF SURGERY

Many patients undergoing surgery do not ingest oral fluids, either in preparation for surgery or as a result of the surgery itself. This will decrease the daily input, however it is opposed by the following (the endocrine response to surgery);

- ADH release conserves water and typically reduces urine volume to 1000-1500 ml for 2-3 days following major surgery.

- Aldosterone secretion conserves sodium and further contributes to oliguria. In the first 2 days after operation, urinary excretion of sodium typically falls to approximately 30 mEq/24 hrs. Potassium excretion is increased during this period to approximately 120 mEq/day (plus the release of potassium from damaged tissues).

This is called renal conservation. Because of this opposition, parenteral fluid replacement for patients kept NPO and are undergoing surgery should be appropriate and not excessive, or else fluid overload and hyponatremia will result.

TYPES OF PARENTERAL FLUIDS

An electrolyte profile is mandatory before selecting the type of solution.

CRYSTALLOIDS

Crystalloid solutions are aqueous solutions of low-molecular-weight ions (salts) with or without glucose. A wide variety of solutions is available as is shown in the table below. Solutions are chosen according to the type of fluid loss being replaced. For losses primarily involving water, replacement is with hypotonic solutions, also called maintenance-type solutions. If losses involve both water and

electrolytes, replacement is with isotonic electrolyte solutions, also called replacement-type solutions.

DEXTROSE SOLUTION 5%

Dextrose solution 5% (5 g of dextrose/100 ml water) does not contain any electrolytes. The dextrose is rapidly metabolized in the body, such that dextrose solution is equivalent to administering water; which distributes rapidly and evenly throughout the entire body fluid compartments.

It follows that 1 litre of intravenous dextrose solution expands the ECF compartment by 330 ml and the intravascular compartment, by only about 70 ml.

5% dextrose is therefore of value for replacing water losses but has no use as a resuscitation fluid to expand the intravascular volume. It is also useful for replacing water deficit, in patients with hypernatremia. Because it contains sugar, it is employed as a measure to prevent the catabolic state (i.e. hypoglycaemia and ketosis) that follows prolonged fasting (e.g. keeping the patient NPO before surgery).

More concentrated dextrose solutions (10%, 20% and 50%) are available, but their use is limited to the management of diabetic patients or patients with hypoglycaemia. These solutions are irritant to veins.

LACTATED RINGER AND SODIUM CHLORIDE 0.9% ('NORMAL SALINE'

Because most intraoperative fluid losses are isotonic (water and solutes), replacement-type solutions are generally used. These fluids are isotonic to ECF. After intravenous administration they distribute rapidly into the ECF compartment, and are appropriate when the main fluid deficiency derives from this source: for example, gastrointestinal losses or intra-operative losses other than bleeding. It follows that 1 litre of these fluids administered intravenously will increase the intravascular volume by about 220 ml after equilibration (complete within 30-60 minutes), and thus are also useful as plasma volume expanders, but it must be remembered that only about one-quarter remains in the circulating volume after redistribution (justifying the 3:1 or 4:1 replacement rule, in haemorrhage).

The most common solution used is Lactated ringer (LR) or ringer's lactate or Hartmann's solution. This solution is slightly hypo-tonic and provides approximately 100 mL of free water per litre and tending to lower serum sodium to 130 mEq/L, however, in most settings, the benefits of replacing such a balanced solution (balanced means that it mimics the plasma composition) when large volumes are needed outweigh the risk. The lactate (used as a buffer) present in Ringer's lactate solution is rapidly metabolized in the liver. This generates bicarbonate ions. Bicarbonate cannot be directly added to the solutions because it is unstable (tends to precipitate). Normal saline (NS)'or Sodium chloride 0.9% is slightly hypertonic and contains more chloride than ECF. When used in large volumes, mild hyperchloremic (non-anion gap) metabolic acidosis (plasma bicarbonate concentration decreases as chloride concentration increases) results. This makes NS the preferred solution for hyperchloremic metabolic alkalosis. Many patients with hyperkalaemia, including patients with renal failure, routinely receive normal saline because it contains no potassium. Because it is nearly isotonic, normal saline is an ideal solution for dilution of packed red blood cells (ringer's solution is not used because it contains lactate and calcium).

OTHER REPLACEMENT-TYPE SOLUTIONS

PlasmaLyte is a family of balanced crystalloid solutions with multiple different formulations available worldwide according to regional clinical practices and preferences. It closely mimics human plasma in its content of electrolytes, osmolality, and pH. These solutions also have additional buffer capacity and contain anions such as acetate, gluconate. They also contain magnesium.

HYPERTONIC SOLUTIONS

Hypertonic salt solutions are less commonly used, and their sodium concentrations range from 250 to 1200 mEq/L. The greater the sodium concentration, the; lower the total volume required for satisfactory resuscitation. This difference reflects the movement due to osmotic forces of water from the intracellular space into the extracellular space, these solutions must be administered slowly (preferably through a central venous catheter) because they readily cause haemolysis. There is little evidence at present that

they have any benefit over more traditional solutions. Potential indications include the treatment of cerebral oedema and raised intracranial pressure, hyponatraemic seizures and 'small volume' resuscitation of hypovolaemic shock. Hypertonic 3% saline is employed in therapy of severe symptomatic hyponatremia.

Crystalloid fluids									
	Category	Na* (mEq/L)	cl^- (mEq/L)	K* (mEq/L)	Glucose (g/L)	Lactate (mEq/L)	Ca^{2+} (mEq/L)	Mg^{2}* (mEq/L)	Others
5% dextrose in water (DsW)	Hypo(253)				5				
Normal saline (NaCI 0.9%)	iso (308)	154	154						
Ringer's lactate	Iso (273)	130	109	4		28	3		
Half normal saline (½NS)	Hypo (154)	77	77						
D_5LR	Hyper (525)	130	109	4	5	28	3		
D_5NS	Hyper (586)	154	154		5				
D_5 ½NS	Hyper (432)	77	77		5				
3% Saline	Hyper (1026)	513	513						
5% Saline	Hyper (1710)	855	855						
Plasmalyte	Iso (294)	140	98	5				3	acetate 27 mEq/L gluconate 23 mEq/L

COLLOID SOLUTIONS

Colloid solutions are those containing particles that exert an oncotic pressure. These particles may occur naturally or be synthetic (refer to the table on the next page). When a colloid solution is administered, the solution remains in the circulation until the colloid particles are removed (predominantly by the reticulo-endothelial system), after which it distributes into the ECF volume because it also contains electrolytes. Colloid solutions are good resuscitation fluids (i.e. plasma volume expanders) because all the volume administered stays in the circulation. In contrast to crystalloid solutions which approximately require 3 or 4 times the volume of fluids lost; colloids solutions are replaced in a 1:1 ratio. This justifies their indication for the use when more than 3-4 litres of crystalloid solution has been injected.

Some starch solutions have a greater oncotic pressure than normal plasma, so fluid is drawn into blood vessels and the circulating volume is increased by more than the volume administered.

Because they contain albumin and because they increase the oncotic pressure, they are preferable when there is hypovolemia and hypoalbuminemia. Most solutions remain in the circulation for between 6 and 24 hours.

COLLOID SOLUTIONS

	Na^+(mEq/l)	K^+(mEq/i)	Cl- (mEq/l)	Misc. (mmol/l)	Oncotic pressure (mmH2O)	Typical plasma half-life	pH
Hemaccel (succinylated gelatin)	145	5.1	145	Ca^{2+} 6.25	370	5 hr	7.4
Gelofusine (Po- lygeline gelatin)	154	0.4	125	Ca^{2+}0.4	465	4 hr	7.4
Hetastarch (hydroxyethyl starch)	154	0	154	Mg^{2+}0.4	310	17 days	5.5
Human albumin solution 4.5% (HAS)	150	0	120	0	275	0	7.4

APPROACH TO FLUID THERAPY

In surgical practice, many patients undergoing surgery or are expected to undergo surgery are kept nil per os. (NPO), fluid replacement by the parenteral route is thus necessary, as in most procedures, an amount of fluid is expected to be lost. Keeping the patient on NPO will cause an output of fluid without an opposing input. These output (via kidney, feces, skin and lungs as discussed earlier) If not replaced properly, patients may enter the surgery with pre-existing deficit that will predispose them to complications such hypovolemia and acute renal failure.

MAINTENANCE FLUID THERAPY

In the absence of oral intake, fluid and electrolyte deficits can rapidly develop as a result of continued urine formation, gastrointestinal secretions, and insensible losses from the skin and lungs. Normal maintenance requirements based on the body weight can be estimated from formula in the table below.

Estimating maintenance fluid requirements	
Weight	**Rate**
-first 10 kg	*4* mL/kg/h (or 100 mL/kg per day)
-next 10 kg	Add 2 mL/kg/h (or 50 mL/kg per day)
-for each kg above 20 kg	Add 1 mL/kg/h (or 20 mL/kg per day)

Using this formula;

A 35 kg child would require (10 x 4 ml) + (10 x 2 ml) + (15 x 1 ml) = 40 + 20 + 15 = 75 ml/hr and a 25-kg child would require 40 + 20 + 5 = 65 mL/h of maintenance fluids.

Electrolyte and mineral requirements are also calculated by body weight, the daily requirement for sodium and potassium in children is approximately 2-3 mEq/kg BW/day. In adults the daily sodium and potassium daily requirement is 1-2 mEq/kg BW/day. Chloride daily requirement is 1.5 mEq/kg/day.

The most common maintenance fluid used is D5½2NS with 20 mEq KCL. This fluid contains about 77 mEq/L of sodium and 77 mEq of chloride with the added 20 mEq potassium. For a 70 kg man the needed daily sodium is (by the previous formula) (70 x 1.5= 105 mEq) 105 mEq. A 70 kg man will require 2.5 litres of fluid.

In patients requiring intravenous fluid replacement for more than 3–4 days, supplementation of magnesium and phosphate may also be required as guided by direct measurement of plasma concentrations. The provision of parenteral nutrition should also be considered in this situation.

For maintenance. If we use D5½2NS + KCL, 2.5 litres will contain about 194 mEq of sodium. 80 mEq of potassium can be given in 4 bags. This will cover the electrolytes needs.

DEFICIT

Patients presenting for surgery after an overnight fast without any fluid intake will have a pre-existing deficit/ proportionate to the duration of the fast. The deficit can be estimated by multiplying the normal maintenance rate by the length of the fast. For the average 70-kg person fasting for 8 h, this amounts to (40 + 20 + 50) mL/h x 8 h, or 880 mL. (In reality, this deficit will be somewhat less as a result of renal conservation.). The fluid infusion rate for normal patients should be set to deliver three to four times the maintenance rate until the calculated deficit has been corrected.

When the period of fast is unknown, or is known but the patient has abnormal fluid losses (sweating, pyrexia, ascites), the amount of fluid deficit cannot be accurately estimated. The best method to estimate fluid deficit is approximation based on the clinical data. Physical examination is relatively reliable preoperatively. Invaluable clues to hypovolemia include skin turgor, the hydration of mucous membranes, fullness of a peripheral pulse, the resting heart rate and blood pressure and the (orthostatic) changes from the supine to sitting or standing positions, and urine output. This can give an approximation of the amount of fluid lost, however, this is affected by the chronicity of the condition

and many drugs can alter these signs (see the table below).

Signs of Fluid Loss			
Sign	Fluid loss as percentage of the body weight		
	5%	10%	15%
Mucous membranes	Dry	Very dry	Parched
Level of consciousness	Normal	Lethargic	Obtunded
Orthostatic hypotension			> 10 mmHg
Orthostatic tachycardia			> 15 bpm
Urine output.	Mildly decreased	decreased	Markedly decreased
Heart rate	Normal or increased	Increased > 100 bpm	Increased > 120 bpm
Blood pressure	Normal	Normal	Start to decrease

A more rational approach is to first diagnose volume deficit and then to correct this deficit according to the restoration of vital signs and maintenance of adequate urine output (0.5 to 1 mL/kg per hour in an adult), and correction of base deficit. Usually an infusion of 1 to 2 L of isotonic fluid (bolus= 1 litre/1hr) followed by a continuous infusion and monitoring is the mainstay.

The fluid used is an isotonic crystalloid, depending on the particular electrolyte profile. If symptomatic electrolyte abnormalities accompany volume deficit, the abnormality should be corrected to the extent that the acute symptom is relieved prior to surgical intervention.

REPLACEMENT

Replacement of fluid pre- and intra-operatively involves knowing which compartment is losing fluid and then replacing with solutions based on the knowledge of the fluid lost. The term fluid replacement is usually used invariably with the term deficit. Usually deficit refer to the replacement of fluid that has been already lost, while the term replacement (in this lecture) refers to both the replacement of deficits and future or present ongoing losses. The sources of fluid loss are as follows;

BLOOD LOSS

The most commonly used method for estimating blood loss is measurement of blood in the surgical suction container and visually estimating the blood on surgical sponges and laparotomy pads ("laps"). A fully soaked sponge (4 x 4) is said to hold 10 mL of blood, whereas a soaked “lap" holds 100-150 mL, serial haematocrits may be of value in long procedures.

Every 1ml of lost blood will require 3 ml of crystalloid fluid or 1 ml of colloid fluid.

THIRD SPACE LOSS

Internal redistribution of fluids (often called "third spacing" because it refers to a space other than the ICF and ECF) can cause massive fluid shifts and severe intravascular depletion. Traumatized, inflamed, or infected tissue (as occurs with bums, extensive injuries, surgical dissections, or peritonitis) can sequester large amounts of fluid in its interstitial space and can translocate fluid across serosal surfaces (ascites) or into bowel lumen. The result is an obligatory increase in a non-functional component of the extracellular compartment, as this fluid does not readily equilibrate with the rest of the compartments. This fluid shift is at the expense of both the functional extracellular and intracellular fluid compartments.

The amount and components of the third space losses, is usually difficult to identify. Aspiration of the fluid losses (as in ascites or using a nasogastric tube in a patient with ileus) will give an idea about the amount and composition of that fluid.

Example

A 70 kg patient with ileus had lost about 2000 ml of fluid in a nasogastric aspirate in one day. The aspirate contains about 240 mEq of sodium and 20 mEq of potassium. How would you calculate the replacement and which fluid would you use?

The maintenance for this patient is 2.5 L. add this to the 2 litres he lost, he then would require 4.5 litres of fluid. Because he requires about 380 mEq of sodium (add the 105 of maintenance plus 240 lost= 345) and 125 mEq potassium. 2 litres of normal saline would cover 300 mEq of sodium. 1 litre of 1/4NS would supply about 39 mEq of sodium. Add these to 1 litre of dextrose water with 120 KCL (6 bags each 20 mEq).

Intraoperatively, third space losses has been estimated based on the type of the procedure performed, third-space fluid losses are dependent on the size of the incision and the extent of tissue trauma and dissection and can be replaced with an appropriate volume of lactated Ringer's solution. Small incisions with minor tissue trauma (e.g. inguinal hernia repair) result in third-space losses of approximately 1-3 mL/kg per hour. Medium-sized incisions with moderate tissue trauma (e.g., uncomplicated sigmoidectomy) result in third-space losses of approximately 3-7 mL/kg per hour. Larger incisions and operations with extensive tissue trauma and dissection (e.g., pancreatico-duodenectomy) can result in third-space losses of approximately 9-11 mL/kg per hour or greater.

GASTROINTESTINAL LOSSES

The magnitude and content of gastrointestinal, fluid losses depend mainly on the site of loss. The approximate electrolyte content and volumes of various gastrointestinal fluids are shown in the table below. The fluids used should be similar in composition to the fluids lost. In cases of intestinal fistulae, it may be useful to measure the electrolyte content of fistula fluid in order to determine the type of fluid replacement required to maintain balance.

INTRAOPERATIVE REPLACEMENT

Ideally, blood loss should be replaced with crystalloid or colloid solutions to maintain intravascular volume (normovolemia) until the danger of anemia outweighs the risks of transfusion. At that point, further blood loss is replaced with transfusions of red blood cells to maintain haemoglobin concentration (or haematocrit) at that level. In practice, most clinicians give lactated Ringer's solution in approximately three to four times the volume of the blood lost, or colloid in a 1:1 ratio, until the transfusion point is reached. At that time, blood is replaced unit for unit as it is lost, with reconstituted packed red blood cells.

As explained before, the fluid replacement for evaporative and third space losses will require additional fluids depending on the size of the wound and the type of the procedure. These values are only guidelines, and actual needs vary considerably from patient to patient.

COMMON DISTURBANCES OF FLUIDS AND ELECTROLYTES IN THE SURGICAL WARD

VOLUME DEPLETION

Pure water depletion (or water deficit) is rare in surgical practice, it implies the loss of water alone with accompanying electrolytes and it is usually caused by inadequate water intake (with secondary urinary, gastrointestinal and insensible losses which are considered hypotonic), hot climate (i.e. sweating), fever and diabetes insipidus (no ,or no response to, ADH with resultant water diuresis out of proportion to natriuresis). Other than the signs of dehydration, pure water depletion is manifested with symptoms and signs of hypernatremia; i.e. muscle rigidity, tremors, seizures, lethargy , or coma. Patients usually have hypernatremia and a low urine sodium (< 15 mEq/L) due to renal conservation of water. The correction of hypernatremia depends on the correction of the accompanying water deficit (see later).

Most often in the surgical ward, water loss is combined with electrolytes loss. This is termed isotonic volume Joss. The most common etiology of volume depletion and isotonic volume loss in surgical patients is a loss of gastrointestinal fluid, but volume depletion due to trauma, peritonitis and the use of diuretics is also seen. Acute volume deficit is associated with cardiovascular and central nervous system signs, while chronic deficits display tissue signs, such as a decrease in skin turgor and sunken eyes, in addition to cardiovascular and central nervous system signs (see the table below).

symptoms and signs of volume deficit	
Symptoms • **thirst** • **anorexia** • **weight loss**	
Signs • **decreased skin turgor** • **small wrinkled tongue** • **collapsed veins** • **sunken eyes**	Chronic signs (slow response)
Vital signs • **Tachycardia** • **thready pulse** • **narrow pulse pressure** • **orthostatic hypotension** • **low temperature** • **liguria; but concentrated urine**	Acute (fast response)

The urine is affected with the severity of dehydration. Severe dehydration will reveal a high specific gravity (> 1.020), high urine osmolality (>500 mosm/kg.H2O), low urine sodium (due to the effect of aldosterone, < 10 mEq/l) and fractional excretion of sodium < 1%; findings consistent with pre-renal renal failure. In dehydration, the BUN to creatinine ratio increases to about 20:1 (normally it is 10:1)

The management depends on the site of loss, the amount of loss and the electrolytes associated with the lost fluid along with the present levels of electrolytes in the patient. Accordingly, the fluid replacement solution is selected as have been discussed in the previous lecture (refer to fluid therapy for more information).

WATER EXCESS

Water excess is common in the surgical practice and is usually seen in the post-operative period. The commonest cause is excessive administration of 5% dextrose or in gastrointestinal washout or during TUR of Prostate. renal conservation due to the effect of ADH and aldosterone as an endocrine response to stress, can predispose the patients to

water excess if they have been replaced with large amounts of fluids. The large amounts are usually given because they are overestimated secondary to inability to accurately quantify third space or ongoing gastrointestinal losses. Water excess may also occur in patients with poor cardiac function or renal failure, or those with hepatic failure or hypoalbuminemia.

CLINICAL FEATURES

Patients with water excess usually remain well, but may develop *dependent edema* with weight gain.' When there is associated cardiac insufficiency (already present or as a result of water excess) distention of jugular veins and elevated jugular venous pressure is seen. In cardiac and renal failure, water accumulation can result in *pulmonary edema,* gallop rhythm (tachycardia with an S3 or S4) indicates cardiac failure.

MANAGEMENT

Patients with high serum sodium (those replaced with slightly hypertonic normal saline, or those with liver failure), should have sodium restriction. Those with water excess with low sodium (i.e. excessive dextrose 5%) should have-water restriction. Treatment of the cause should be considered and treatment with diuretics is indicated in severe water excess as in the case of pulmonary edema.

HYPONATREMIA

Hyponatremia is defined as serum sodium less than 135 mEq/L

ETIOLOGY

The causes of hyponatremia are either pure water excess (dilution hyponatremia or water intoxication) or sodium loss) in the surgical ward, hyponatremia most often develops as a result of pure water excess, specifically, more in the post-operative period due to excessive administration of dextrose 5%. Gastrointestinal losses comes second and usually due to diarrhea, vomiting or large amounts of fluid in nasogastric aspirate or fistula: Loss from skin through burns is also seen. Other causes are shown in the table below.

causes of hyponatremia

Dilutional hyponatremia

- **Iatrogenic water excess**
- **SIADH or psychogenic polydypsia**
- **•. Cirrhosis**
- **Nephrotic syndrome**
- **Congestive heart failure**

Sodium loss

- **Gastrointestinal losses (vomiting, diarrhea)**
- **Skin loss (e.g. burns, sweating)**
- **Renal loss (e.g. Diuretics, Adrenal insufficiency)**

Pseudohyponatremia

- **Hyperglycemia (decrease by 1.6 mEq/L for every 100 mg/di increase in sugar)**
- **Hyperlipidemia**
- **Hyperproteinemia**

CLINICAL FEATURES

The clinical features of hyponatremia are mainly neurologic and depend on how fast the level of sodium drops, rather on the level of sodium. If the drop was slow patients may be symptomless until about 125 mEq/L symptoms range from mild confusion to obtundation to seizures and coma.

MANAGEMENT

the treatment of hyponatremia is directed at correcting both the underlying disorder as well as the plasma sodium. Isotonic saline is generally the treatment-of choice for hyponatremic patients with decreased total body sodium content. Once the extracellular fluid deficit is corrected, spontaneous water diuresis returns plasma sodium to normal. Conversely, water restriction is the primary treatment for hyponatremic patients with normal or increased total body sodium.

Acute symptomatic hyponatremia (usually less than 125) requires prompt treatment. In such instances, correction of plasma sodium to > 125 mEq/L is usually sufficient to alleviate symptoms. The amount of NaCI necessary to raise plasma sodium to the desired value, the Na^+ deficit, can be estimated by the following formula:

Na^+ deficit= TBW x (desired Na* - present Na^+)

Very rapid correction of hyponatremia has been associated with demyelinating lesions in the pons (central pontine myelinolysis), resulting in serious permanent neurological sequelae. Generally the rate of rise should no excess 0.5 mEq/L/hr (or more than 12 mtq/L/hr).

EXAMPLE;

An 80-kg woman is lethargic and is found to have a plasma [Na^+] of 118 mEq/L How much NaCI must be given to raise her plasma [Na^+] to 130 mEq/L?

TBW is approximately 50% of body weight in females, thus when applying to the formula;

Na* deficit= 50% x 80 x (130-118)= 40 x 12 = 480 mEq

Because normal (isotonic) saline contains 154 mEq/L, the patient should receive 480 mEq ÷ 154 mEq/L, or 3.12 L of normal saline. For a correction rate of 0.5 mEq/L/h, this amount of saline should be given over 24 h (130 mL/h).

SIADH

SIADH or syndrome of inappropriate antidiuretic hormone secretion is encountered **in** surgical practice after head injuries. It is also seen after burns and severe pain and the hormone ADH is released from some forms of cancers (bronchus cancer). The hall - mark is an increase in the level of ADH hormone with increased water reabsorption. This well cause a mild water overload with secondary dilutional hyponatremia and shutdown of aldosterone with resultant sodium loss. It presents with symptoms of hyponatremia. The urine is usually dark and concentrated with high osmolality. The diagnosis is made by the presence of high urine osmolality with low serum sodium. Fluid restriction is usually enough for treatment, however in severe cases an infusion of normal saline with a loop diuretic can increase the sodium level.

HYPERNATREMIA

Hypernatremia in surgical practice is generally not caused by an excess of sodium, but rather by a relative deficit of pure Water in the body. Another case is seen after head injuries that involves posterior pituitary and results in what is termed central diabetes **insipidus,** though this is less frequent

CLINICAL FEATURES AND MANAGEMENT

Patients may present with neurologic symptoms ranging from lethargy to seizures and coma, depending on the level of sodium and the rate of its increase, patients with diabetes insipidus will have polyuria with dilute urine.

Treatment of hypernatremia aims at correcting the underlying water deficit, Water deficits, should generally be corrected over 48 h with a hypotonic solution such as 5% dextrose in water. The water deficit can be calculated from the following formula;

Water deficit = TBW x (serum Na+ -140 (or desired Na+))/140 (desired Na+)

Rapid correction of hypernatremia can result in seizures, brain edema, permanent neurological damage, and even death. In general, plasma sodium concentration should not be decreased faster than 0.5 mEq/L/h.

EXAMPLE;

A 70-kg man is found to have a plasma sodium of 160 mEq/L. What is his water deficit.

Applying the previous formula,

water deficit = 60% x 70 (160-140)/140= 6 liters to replace this deficit over 48 h, it is necessary to give 5% dextrose in water intravenously, 6000 mL over 48 h, or 125 mL/h

POTASSIUM BALANCE

Intracellular potassium concentration is estimated to be 140 mEq/L, whereas extracellular potassium concentration is normally about 4.5 mEq/L (between 3.5-5.5). Dietary potassium intake averages

80 mEq/d in adults (range, 40-140 mEq/d). About 70 mEq of that amount is normally excreted in urine, whereas the remaining 10 mEq is lost through the gastrointestinal tract.

Extracellular potassium is a major determinant of aldosterone secretion from the adrenal gland. Hyperkalaemia stimulates aldosterone secretion, whereas hypokalaemia suppresses aldosterone secretion. Renal tubular flow in the distal nephron may also be an important determinant of potassium secretion because high tubular flow rates (as during osmotic diuresis) increase potassium secretion by keeping the capillary to renal tubular gradient for potassium secretion high.

Intercompartmental shifts of potassium are known to occur following changes in extracellular pH, circulating insulin levels and circulating catecholamine activity. Insulin and catecholamines are known to directly affect Na+-K+ ATPase activity and decrease plasma potassium (shifting it to the ECF). Changes in extracellular hydrogen ion concentration (pH) directly affect extracellular potassium because the ICF may buffer up to 60% of an acid load. During acidosis, extracellular hydrogen ions enter cells, displacing intracellular potassium ions; the movement of potassium ions out of cells maintains electrical balance but increases extracellular and plasma potassium. Conversely, during alkalosis, extracellular potassium ions move into cells to balance the movement of hydrogen ions out of cells; as a result, plasma potassium decreases. Although the relationship can be quite variable, a useful rule of thumb is that plasma potassium concentration changes approximately 0.6 mEq/L per 0.1 U change in arterial pH (range 0.2-1.2 mEq/L per 0.1 U).

HYPOKALEMIA

Hypokalemia is defined as plasma potassium less than 3.5 mEq/L. it is the commonest electrolyte disorder in the surgical ward. It can be mild (3.5-3), moderate (3-2.5) or severe (<2.5).

ETIOLOGY

can occur as a result of (1) increased potassium loss (through kidney or Gl tract), (2) an intercompartmental shift of potassium (see above) or (3) an inadequate potassium intake.

Most commonly hypokalemia is due to loss through the gastrointestinal tract usually by vomiting, diarrhea or suction. In the surgical practice, Renal output can usually be caused by diuretics (loop or thiazide diuretics) or by increased aldosterone activity (hyperaldosternosim; conn's syndrome)

Intercompartmental shift resulting in hypokalemia is usually caused by alkalosis as occurs in gastric outlet obstruction, or as a result of hyperinsulinemia; either exogenous or due to an insulinoma. Less encountered, catecholamines excess (e.g. salbutamol) can result in hypokalemia.

CLINICAL FEATURES

Hypokalemia is known to affect many organs. Effect on the heart is reflected by arrhythmias and decreased cardiac contractility. The decrease in the contractility of gastrointestinal muscles results in paralytic ilues. Neuromuscular effects of hypokalemia include skeletal muscle weakness and if the respiratory, muscles are affected, results would be fatal.

ECG manifestations are primarily due to delayed ventricular repolarization and include T-wave flattening and inversion, an increasingly prominent U wave, ST? segment depression, increased P-wave amplitude, and prolongation of the P-R interval (Figure 1).

TREATMENT OF HYPOKALEMIA

For mild hypokalemia, oral replacement with potassium chloride solutions is generally safest (60-80 mEq/d).

Rapid infusion of potassium more than the recommended rates can result in diastolic cardiac arrest. Hypokalemia in spite of replacement therapy (refractory hypokalemia) indicates coexistent magnesium deficiency.

Intravenous replacement of potassium chloride should usually be reserved for patients with moderate hypokalemia or those at risk for serious cardiac manifestations or muscle weakness. Peripheral intravenous replacement should not exceed 8 mEq/h (40 mEq/L/hr) because of the irritative effect of potassium on peripheral veins. Dextrose-containing solutions should generally be avoided because the resulting hyperglycemia and secondary insulin

secretion may actually lower plasma potassium even further.

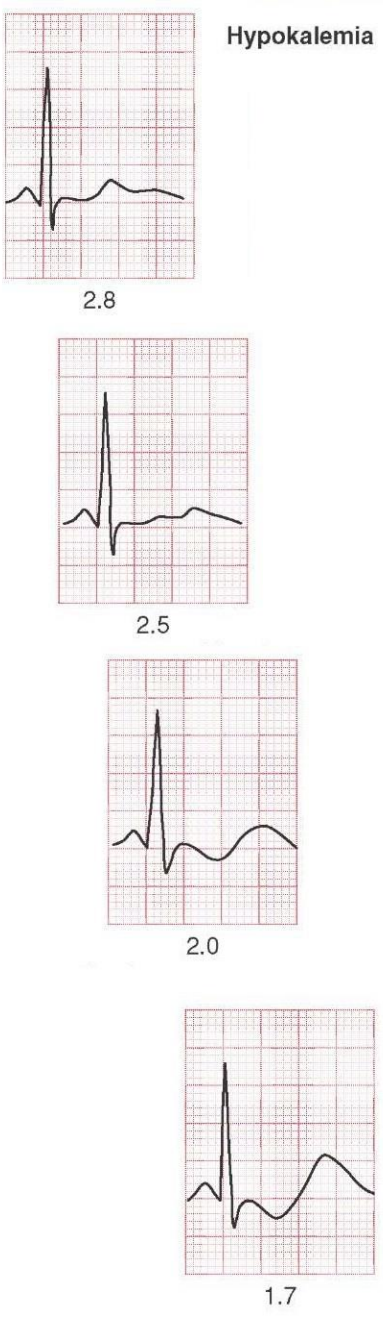

Figure 1 ECG of hypokalemia

HYPERKALEMIA

Hyperkalemia exists when plasma potassium exceeds 5.5 mEq/L it can be mild (5.5-6), moderate (6-6.5) or severe (urgency 6.5-7, emergency >7).

ETIOLOGY

Hyperkalemia can result from (1) an intercompartmental shift of potassium ions, (2) decreased urinary excretion of potassium, or, rarely, (3) an increased potassium intake. Most commonly in surgical practice, intercompartmental shift can occur due to acidosis or

Faster intravenous replacement (10-20 mEq/h) are needed in severe hypokalemia and requires a central venous catheter (some may use femoral catheters to decrease local potassium at the heart level) and close monitoring of the ECG.

the release of potassium from cells after their destruction as occurs after burns, crush injuries, hemolysis or severe catabolic states (i.e. sepsis). Decreased urinary excretion can occur as a result of renal failure, or as a result of aldosterone shutdown (e.g. Addison's disease).

In vitro release of potassium from white cells in a blood specimen can also falsely indicate increased levels in the measured plasma potassium (pseudohyperkalemia) when the leukocyte count exceeds 70,000 x 10^9/L. A similar release of potassium from platelets occurs when the platelet count exceeds 1,000,000 x 10^9/L. this can also occur due to hemolysis with in the specimen.

CLINICAL FEATURES

Hyperkalemia is hardly identified clinically. Sometimes there is muscle weakness, intestinal colic or diarrhea. Cardiac manifestations can occur and are more appreciated on the ECG monitor. ECG changes characteristically progress (in order) from symmetrically peaked T waves (often with a shortened QT interval), widening of the QRS complex, prolongation of the P-R interval, less of the P wove, loss of R-wave amplitude, ST- segment depression (occasionally elevation), an ECG that resembles a sine wave before progression to ventricular fibrillation and asystole (figure 2).

Management

Because of its lethal potential, hyperkalemia exceeding 6 mEq/L should always be treated. Treatment is monitored with reversal of ECG signs.

Calcium (5-10 mL of 10% calcium gluconate or 3-5 mL of 10% calcium chloride) partially antagonizes the cardiac effects of hyperkalemia and is useful in patients with marked hyperkalemia. It's effects are short lived (1 hour).

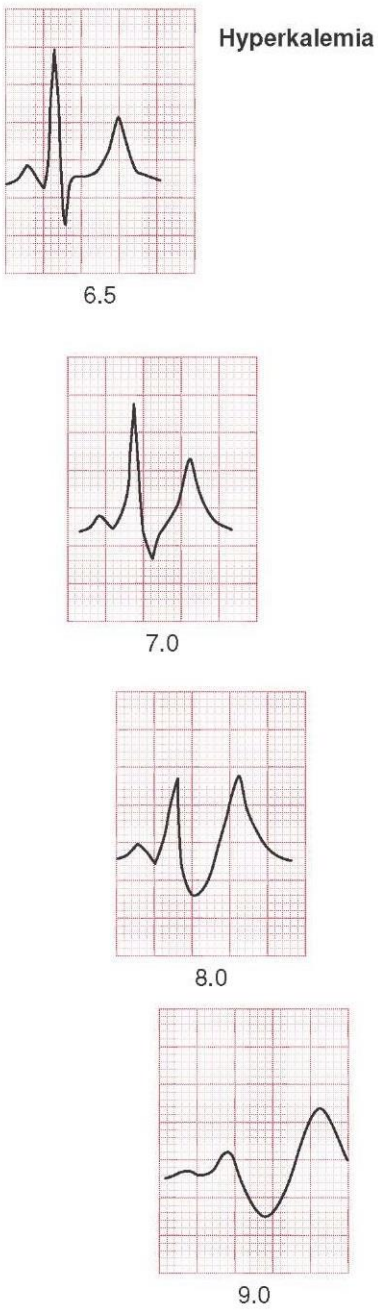

Figure 2 ECG changes in hyperkalemia

intravenous sodium bicarbonate (usually 45 mEq) will promote cellular uptake of potassium and can decrease plasma potassium within 15 min, this should not be given in the same IV line as potassium as it may precipitate. It is usually employed when there is evident metabolic acidosis (still controversial).

An intravenous infusion of glucose and insulin (100 ml of 50% glucose with 20 units of insulin) is also effective in promoting cellular uptake of potassium and lowering plasma potassium, but often takes up to 1 h for peak effect. The use of ß-agonists promote cellular uptake of potassium is restricted to certain situations.

For patients with some renal function, loop diuretics is a useful adjunct in increasing urinary excretion of potassium. In the absence of renal function, elimination of excess potassium can be accomplished only with nonabsorbable cation-exchange resins such as oral or rectal sodium polystyrene sulfonate (Kayexalate). Each gram of resin binds up to 1 mEq of K^+ and releases 1.5 mEq of Na^+; the oral dose is 20 g in 100 mL of 20% sorbitol (sorbitol prevents constipation). Dialysis is indicated in symptomatic patients with severe or refractory hyperkalemia or in those when renal failure is the cause.

CALCIUM BALANCE

Although 98% of total body calcium is in bone. Calcium intake in adults averages 1-3 gm/d. about 80% of those are lost in feces. The kidneys are responsible for calcium excretion. Renal calcium excretion averages 100 mg/d. Normally, 98% of the filterable calcium is reabsorbed. The reabsorbtion occurs at the proximal and distal tubules, but is controlled by the parathyroid hormone at the distal tubule.

The normal plasma calcium concentration is 8.5-10.3 mg/dL (2-2.5 mmol/L). Approximately 50% is in the free ionized form; 40% is protein bound?(mainly to albumin), and 10% is complexed with anions such as citrate and amino acids. It is the free ionized calcium concentration ([Ca2+]).that is physiologically most important. Plasma [Ca2+] is normally 4.75-5.3 mg/dl (2.38-2.66 mEq/L or 1.19-1.33 mmol/L). Changes in plasma albumin concentration affect total but not ionized calcium concentrations: for each increase on decrease of 1 g/dL in albumin (normally it is 3.5-5 g/dI) the total plasma calcium concentration increases or decreases approximately 0.8-1.0 mg/dL, respectively.

Changes in plasma pH directly affect the degree of protein binding and thus ionized calcium concentration. Ionized calcium increases approximately 0.16 mg/dL for each decrease of 0.1 unit in plasma pH and decreases by the same amount for each 0.1 unit increase in pH.

The level of calcium is regulated by the parathyroid hormone, vitamin D and to a lesser degree by the hormone calcitonin.

When the calcium concentration of the blood falls below normal, the parathyroid gland secrete parathyroid hormone (PTH), or parathormone. parathormone increases bone resorption, and calcium reabsorption from the kidneys. The net result of PTH secretion is an increase in calcium concentration in body fluids.

PTH also stimulates the formation and secretion of 1,25-dihydroxyvitamin D3 (vitamin D) at the kidneys. In general, the effects of calcitriol complement or enhance those of PTH, but one major effect of calcitriol is the enhancement of calcium and phosphate absorption by the digestive tract.

Calcitonin is produced by thyroid C cells and functions as an antihypercalcemic hormone by inhibiting osteoclast-mediated bone resorption. At the kidney, calcitonin increases phosphate excretion by inhibiting its reabsorption. It has minimal or no role in calcium regulation.

HYPOCALCEMIA

Hypocalcemia is defined as a serum calcium level below the normal range of 8.5 to 10.3 mg/dl, or a decrease in [Ca^{2+}] below the range of 4.75-5.3 mg/dl. Because the diagnosis can only be made based on the results of ionized calcium level [Ca^{2+}], when direct measurements of plasma [Ca^{2+}] are not available, the total calcium concentration must be corrected for decreases in plasma albumin concentration (see previous).

ETIOLOGY

Hypocalcemia due to hypoparathyroidism is a relatively common cause of symptomatic hypocalcemia. Second in incidence is renal failure and pancreatitis. This is encountered commonly after thyroid surgeries that involve the thyroid gland. Magnesium deficiency is postulated to impair the secretion of PTH and antagonize its effects on bone, thus resulting in hypocalcemia. This is seen in patients put on parenteral nutrition for long periods.

The causes are shown in the table below.

Common causes of hypocalcemia in the surgical ward
• **Hypoprathyroidism**
• **Hypomagnesemia**
• **Severe pancreatitis**
• **Renal failure**
• **Severe blood loss (albumin loss)**
• **Massive blood transfusion**
• **Crush injuries (destroyed muscle accumulates calcium)**
• **Necrotizing fasciitis**
• **Others (e.g. calcitonin-secreting medullary carcinomas)**

CLINICAL FEATURES

A fall in the plasma calcium level increases neuromuscular excitability causing cramps or even tetany or seizures in severe cases. An early symptom of hypocalcemia is paraesthesia, especially around the lips. Clinical tests for hypocalcemia include tapping over the parotid gland. This provokes transient contraction of the facial muscles and is known as Chvostek's sign. A further test involves inflating a sphygmomanometer cuff on the upper arm to above systolic pressure. this induces carpal spasm within about 3 minutes (Trousseau's sign or carpopedal spasm). Deep tendon reflexes are exaggerated in hypocalcemia.

Cardiac irritability can lead to arrhythmias. Decreased cardiac contractility may result in heart

failure, hypotension, or both. ECG signs include prolongation of the QT interval.

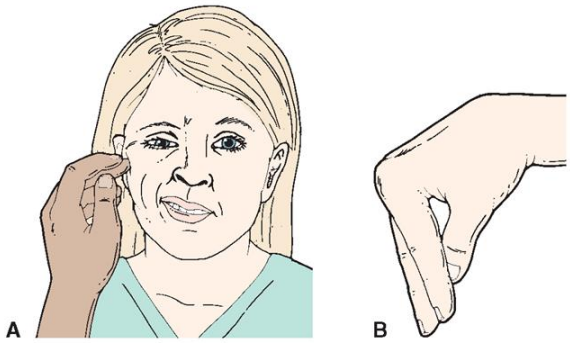

Figure 3 signs related to hypocalcemia; (A) chvosteks sign, (B) carpopedal spasm.

The table below presents important diagnostic clues to the etiology of hypocalcemia.

Diagnostic clues to hypocalcemia
• **History of thyroid or neck surgery** • **Calcium level (corrected total and ionized)** • **Albumin** • **ABGs for pH** • **Magnesium** • **Kidney function tests** • **Parathyroid hormone**

MANAGEMENT

Acute symptomatic hypocalcemia is a medical emergency and should be treated immediately with intravenous calcium chloride (3-5 mL of a 10% solution) or calcium gluconate (10-20 mL of a 10% solution). Serial ionized calcium measurements are mandatory. Repeat boluses or a continuous infusion (Ca^{2+} 1-2 mg/kg/h) may be necessary.

Before an attempt to give calcium, blood pH is checked at first. If there is an associated alkalosis, it should be corrected. Usually the total calcium is not affected. This is commonly encountered in patients who are hysterical and hyperventilate to the degree of respiratory alkalosis.

In *chronic* hypocalcemia , oral calcium ($CaCO_3$) and vitamin D replacement (1-α-vitamin D_3) are usually necessary. Marked hyperphosphatemia is thought to lower plasma [Ca^{2+}] by precipitation and deposition of calcium phosphate in bone and soft tissues. Hyperphosphatemia is generally treated with phosphate- binding antacids such as aluminum hydroxide or aluminum carbonate Plasma magnesium concentration should be checked to exclude hypomagnesemia.

HYPERCALCEMIA

Hypercalcemia is defined as a serum calcium level above the normal range of 8.5 to 10.3 mg/dl, or an increase in the ionized calcium level above 4.75-5.3 mg/dl. However, the definition is broader than this. It is the level of the parathyroid hormone that determines wither calcium levels are high or not. For exampie patients with high parathyroid hormone levels and a level of calcium that is high normal (e.g. 10.3 mg/dl) are deemed hypercalcemic.

ETIOLOGY

Primary hyperparathyroidism (due to adenoma, hyperplasia or carcinoma, see the lecture on the parathyroid glands) as a cause of hypercalcemia is more commonly encountered in surgical than secondary or tertiary types.

Patients with cancer can present with hypercalcemia. This is seen most commonly after metastasis to bone by an osteolytic form of cancer. In surgical practice this is seen most commonly after breast cancer. Less frequent are those seen after prostate, bronchial, thyroid and kidney tumors, secretion of humoral mediators of hypercalcemia (PTH-like substances) is probably responsible for hypercalcemia in most patients with bronchial, ovarian and kidney cancers.

For now the above causes comprise about 90% of the incidence of hypercalcemia. Less common causes are shown in the table below.

Causes of hypercalcemia in surgical

- **Hyperparathyroidism**
- **Cancer metastasis to bone**
- **Carcinomas with endocrine secretion (e.g. bronchial cancers)**
- **Vitamine D intoxication**
- **Sarcoidosis**
- **Hyperthyroidism**
- **Prolonged immobilization**
- **milk-alkali syndrome (i.e. increase in calcium intake)**

CLINICAL FEATURES

Hypercalcemia often produces anorexia, nausea, vomiting, weakness, and polyuria (with concomitant dehydration and resultant polydipsia and oliguria). Hypertension is often present initially before hypovolemia (due to diuresis) supervenes. Ataxia, irritability, lethargy, or confusion can rapidly progress to coma. Other manifestations of hyperparathyroidism include peptic ulceration, acute and chronic pancreatitis

The symptoms of primary hyperparathyroidism can be remembered by the aide memoire; "bones, stones, abdominal groans and psychic moans" (further is explained at the parathyroid glands lecture).

ECG signs include a shortened ST segment and a shortened QT interval.

MANAGEMENT

Symptomatic hypercalcemia requires rapid treatment. The most effective initial treatment is rehydration followed by a brisk diuresis with administration of intravenous saline infusion and a loop diuretic to accelerate calcium excretion. Premature diuretic therapy prior to rehydration may aggravate the hypercalcemia by additional volume depletion. Severe hypercalcemia (> 15 mg/dL) usually requires additional therapy with a bisphosphonate or calcitonin.

Additional treatment depends on the underlying cause of the hypercalcemia and may include glucocorticoids in the setting of vitamin D-induced hypercalcemia such as granulomatous disease states. Older agents such as plicamycin (mithramycin) or phosphates are seldom used today because of their potential adverse effects. Dialysis should be considered when renal failure is the cause of hyperparathyroidism.

MAGNESIUM BALANCE

Magnesium is an important intracellular cation that functions as a cofactor in many enzyme pathways. Only 1-2% of total body magnesium stores is in the ECF compartment; 67% is contained in bone whereas the remaining 31% is. intracellular. Magnesium intake averages 20-30 mEq/d (250 mg/d) in adults. Plasma [Mg2+] is closely regulated between 1.7 and 2.1 mEq/L (0.7-1 mmol/L). Although the exact mechanisms involved remain unclear, they involve interaction of the gastrointestinal tract (absorption), bone (storage), and the kidneys (excretion). Approximately 50-60% of plasma magnesium is unbound and diffusible.

HYPOMAGNESEMIA

Hypomagnesemia is a common and frequently overlooked problem, particularly in critically ill patients. Deficiencies of magnesium are generally the result of inadequate intake, reduced gastrointestinal absorption, or increased renal excretion. Hypokalemia is seen with hypomagnesemia is 90% of cases and is due to promotion of potassium renal loss. Patients usually present with manifestations of hypokalemia. The classic sign of severe hypomagnesemia (< 1.2 mg/dL) is hypocalcemia, the mechanism is controversial.

hypomagnesemia can be treated orally (magnesium sulfate heptahydrate or magnesium oxide) or intramuscularly (magnesium sulfate; 2meq/kg/day).

HYPERMAGNESEMIA

In surgical practice, increases in plasma [Mg2+] are nearly always due to renal impairment (GFR < 30 mL/min).

Symptomatic hypermagnesemia typically presents with neurological, neuromuscular, or cardiac manifestations. Hyporeflexia, sedation, and skeletal muscle weakness and paralysis are characteristic features. Vasodilation, bradycardia, and myocardial depression can lead to hypotension at high levels. ECG signs are inconsistent but often include prolongation of the P-R interval, absence of P waves, tall tented T waves and widening of the QRS complex and may mimic those of hyperkalemia.

Marked hypermagnesemia can lead to respiratory arrest.

Intravenous calcium (1 g calcium gluconate) can temporarily antagonize most of the effects of hypermagnesemia. A loop diuretic along with an infusion of 1⁄2 normal saline in 5% dextrose enhances urinary magnesium excretion. Dialysis may be necessary in patients with marked renal impairment.

WOUND

Wounds are discontinuities in the tissues that result from trauma.

CLASSIFICATION

Wounds can be classified according to the mechanism of injury;

- **Abrasions**; These result from friction damage to the body surface and are characterized by superficial bruising and loss of a varying thickness of skin and underlying tissue.

- **incised wounds**; caused by sharp instrument and usually have regular edges with no associated structures involved, other than the skin itself.

- **Lacerated wounds**; usually caused by blunt objects, and are associated with irregular edges and involvement of structures other than skin. It is usually felt with dirt and is more likely to be infected.

- **Penetrating (or stabbing) wounds**; these are caused by sharp penetrate through a very small opening and cause deep penetration, so that the extent of the wound is more than it is obvious.

- **Degloving injuries**; These result from shearing forces that cause parallel tissue planes to move against each other: for example, when a hand is caught between rollers or in moving machinery. Large areas of apparently intact skin may be deprived of their blood supply by rupture of feeding vessels (the non-viable skin is more extensive than you think).

- **Crush injuries**; These are due to severe pressure and is considered a severe form of blunt trauma. Even though the skin may not be breached, there can be massive tissue destruction, edema can make wound closure impossible. Increasing pressure within fascial compartments can cause ischemic necrosis of muscle and other structures (compartment syndrome).

- **Gunshot (missile) wounds**; These may be low- velocity (i.e. low energy e.g. shotguns) or high- velocity (i.e. high energy e.g. military rifles). Bullets fired from high-velocity rifles cause massive tissue destruction after skin penetration.

Most surgical wounds are incised. Wounds can also be classified depending on the duration. Acute wounds are those which have been present for a period of less than 4-6 weeks. Wounds are termed chronic when they have been present for more than 4-6 weeks. According to the risk of infection wounds can be classified as follows (this classification is important for operative wounds);

- **Clean wounds**; contamination is not expected and should not occur. In clean operations (such as hernia repair or thyroid surgery), the wound infection rate should be less than 1.5%.

- **Clean-contaminated wounds**; are those in which no frank focus of infection is encountered but where a significant risk of infection is nevertheless present, perhaps because of the opening of a viscus, such as the colon or urinary tracts. Infection rates is in excess of 7.5%.

- **Contaminated wounds**; in here the risk increases from the that of the previous category because of a major break in sterile techniques, the presence of an infection focus or the spillage of a viscus contents. The infection rate can reach 15% or more.

- **Dirty wounds**; are those in which gross contamination is inevitable and the risk of wound infection is high (up to > 40%); an example is emergency surgery for perforated diverticular disease.

WOUND HEALING

Soft tissue healing can be subdivided into three phases;

- **Inflammatory phase**; this extends to about 2-5 days after the initial injury. It starts by a haemostatic phase where by the bleeding is stopped by vasoconstriction and platelets aggregation. Upon the release of various cytokines (e.g. PDGF, TGF, EGF) from the platelets (and the damaged tissue), vasodilatation and chemotaxis allows white blood cells to accumulate at the site of injury.

White blood cells, especially macrophages will get rid of foreign material and dead tissue and release

further cytokines which propagates the inflammatory response.

• **Proliferative phase**; this extends from the end of the inflammatory phase to about 3 weeks from the time of injury. This Many process occur during the proliferative phase these include the formation of new capillary loops (angiogenesis), the deposition of ground substance (glycosaminoglycans, fibronectin and elastin) and collagen (type Ill) by fibroblasts (the process of fibroplasias), early in this phase, the wound supporting tissue is called granulation tissue. Epithelial cells at the edge of the wound lose their adhesion to each other and migrate across the wound until they meet cells from the other side. As they migrate, they are replaced by new cells formed by the division of basal cells near the wound edge. The cells that have migrated undergo mitosis and the new epithelium thickens, eventually forming normal epithelial cover for the scar produced by the dermis. This process is referred to as the process if re-epithelialization.

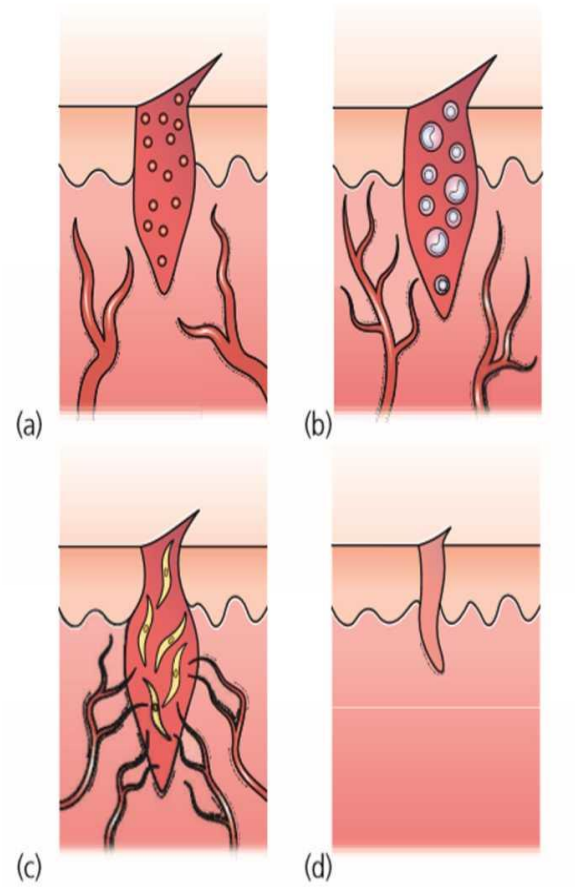

Figure 1. The phases of healing, (a) Early inflammatory phase with platelet-enriched blood clot and dilated vessels, (b) Late inflammatory phase with increased vascularity and increase in polymorphonuclear lymphocytes and lymphocytes (round cells), (c) Proliferative phase with capillary buds and fibroblasts, (d) Mature contracted scar.

Re-epithelialization is complete in less than 48 hours in the case of approximated incised wounds, but may take substantially longer in the case of larger wounds, where there is a significant epidermal/dermal defect.

Wound contraction starts with the process of fibroplasia and is maximal at 5-15 days after injury. It is caused by the presence of myofibroblasts.

• **The remodelling phase**; during the proliferative phase, the collagen synthesized is type III. The remodelling phase is characterized by maturation of collagen (type I replacing type III until a ratio of 4:1 is achieved). The cellular contents decrease in the formed scar tissue, and most of the water content is reabsorbed at the 21st day of injury. Orientation of collagen fibres in the direction of local mechanical forces increases tensile strength. However, skin and fascia usually recover only 80% of their original tensile strength. The process ex¬tends from about 3 weeks to 2 years of injury.

Scar remodeling continues for many (6 to 12) months postinjury, gradually resulting in a mature, avascular, and acellular scar. The mechanical strength of the scar never achieves that of the uninjured tissue.

Factors which might adversely affect wound healing are shown in the table below.

Factors adversely affecting wound healing
Local factors
• Decreased Blood supply
• Hypoxia
• Infection
• Hematoma
• Mechanical stress (e.g. compression from sutures, bed sores)
• Type of tissue (nerves are less likely to re-generate)
• Surgical technique
• Suture material
Systemic factors
• Age (young children and old age)
• Anemia
• Malnutrition;
Protein deficiency
Vitamin C deficiency (required for hydroxylation of collagen proline residues)
Zinc deficiency (important cofactor for many anabolic enzymes)
Calcium and magnesium deficiencies
• Drugs
Steroids (prevent inflammation and collagen syn- theis)
Cytotoxic drugs (prevent inflammation)
• Malignancy
• Diseases (i.e. diabetes and uremia)

MANAGEMENT OF THE ACUTE WOUND

The first step in managing apparent wounds is to stop bleeding, this is achieved by local pressure (or the use of tourniquets in limbs). Stabilization of the cardiovascular status of the patient takes priority over the definitive repairs.

A wound should be explored and debrided to the limit of blood staining. Devitalised tissue must be excised until bleeding occurs with the obvious exception of nerves, vessels and tendons. The use of copious saline irrigation or pulsed jet lavage (where the instrumentation is available) can be less destructive than knife or scissors when debriding. Removing the non-viable tissue and proper cleaning will improve healing.

The choice of suture material is important, absorbable, sutures (e.g, polyglycolic acid) are appropriate for internal layers (e.g. fascias, muscle). Non absorbable (e.g. nylon) sutures are more useful for external layers (skin). The suturing technique is important and should emphasize on not making the sutures two tight as it might cause poor healing due to decreased blood supply. An incised wound that is clean and sutured closed is said to heal by primary intention (figure 2). The wound edges are usually close and there is minimal scar tissue. When the wound is infected or has a poor blood supply, the resultant wound would be filled with granulation tissue. This granulation tissue filled wound will re-epithelialize and end with a large, usually ugly scar. This process is referred to as healing by secondary intention. Contraction of the wound would be pulling the edges together. Delayed primary intention healing (or heeling by tertiary intention) means to leave the wound open, when the conditions for healing are unfavourable, such as the presence of poor blood supply, or infection and then until, favourable conditions are restored, closure of the wound is commenced, meanwhile the wound is left open (drained). This allows for better healing and minimal scaring tissue, along with a decreased rate of infection.

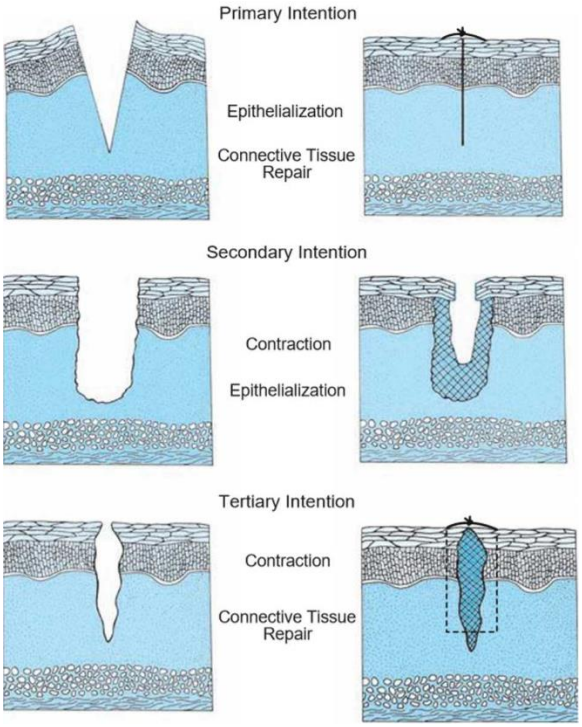

Figure 2. Different clinical approaches to the closure and healing of acute wounds.

in a new blood supply and can be used to cover tendon, nerve, bone and other structures that would not provide a suitable vascular base for a skin

graft. A skin graft has no inherent blood supply and is dependent on the recipient site for nutrition.

Hematomas should be evacuated when they are symptomatic, or when might have an effect on the healing process. Degloving injuries are usually more extensive than one can see, and thus require the use of flourscein dying to detect viable and non-viable skin and remove the later.

MANAGING CHRONIC WOUNDS

A form of chronic wounds is referred to as leg ulcers and is the most common form. The most common cause of leg ulcers are vascular in nature (arterial or venous). They can be also caused by self-infliction, infections (e.g. syphilis) or malignancies (e.g. squamous cell carcinoma). Pressure sores (or bed sores) is the other variety of chronic wounds and is caused by chronic obstruction of the capillary network (closing pressures 30 mmHg), usually by pressure (such as the immobilized critically ill patient).

Treatment of leg ulcers depend on the cause and require thorough assessment of the circulation (as healing is impaired). Biopsy for excluding malignant ulcers (marjolin's ulcer) is essential. When healing is not expected, surgery using meshed skin grafts (skin grafts that has been systematically cut) is an option.

The treatment of pressure sores follows that of acute wounds. Adequate debridement is essential.

Once clean, all the structures inside the wound are repaired. Each structure has especial line of treatment. Nerves, vessels and tendons require the use of 8/0 or 10/0 monofilament nylon sutures under magnification (loupes or microscope). Tendon repairs require early mobilization to prevent adhesion to the fibrous sheath (and thus limitation of movement).

Skin cover by flap or graft may be required as skin clo¬sure should always be without tension and should allow for the edema typically associated with injury and the inflammatory phase of healing. A flap brings

Large skin flaps are then used to cover the resultant space.

In both leg ulcers and pressure sores, the use of what is referred to as the vacuum assisted wound closure (VAC) or Negative-pressure wound therapy (NPWT) can be of value. It is said that the application of negative pressure intermittently through a dressing at the site of the wound, draws fluid and thus increase the local circulation. This in turn can improve healing for both conditions, whereby the cause is disrupted circulation.

Complications of wound healing

Infection

Wound infection is a common complication and might be life threatening if not identified. Signs indicating wound infection include swelling with serous discharge between the sutures or from the gap of the wound. Patients are usually pyrexic. Signs such as crepitus (due to the presence of gas forming bacteria), swelling beyond the margin of erythema), blistering and tenderness that is out of proportion to the severity of the wound, should be alarming to the fact that there might be a necrotizing skin infection (e.g. necrotizing fasciitis).

Antibiotic treatment of acute wounds must be based upon organisms suspected to be found within the infected wound and the patient's overall immune status. When a single specific organism is suspected, treatment may be commenced using a single antibiotic. Conversely, when multiple organisms are suspected, as with enteric contamination or when a patient’s immune function is impaired by diabetes, chronic disease, or medication, treatment should commence with a broad-spectrum antibiotic or several agents in combination. Antibiotics can also be delivered topically.

Wound dehiscence

This is caused by the breaking the wound open along a surgical suture. It might be due to error in the surgical technique (e.g. excessive tension) or due to patient internal risk factors (e.g. connective tissue disease (e.g. ehlar-danlos syndrome), obesity , steroid use).

Pathological fibrosis

This is caused by the formation of excessive collagen in the scar tissue. A hypertrophic scar is defined as excessive scar tissue that does not extend beyond the boundary of the original incision or wound and never continue to worsen after 6 months. It is caused by prolonged inflammatory phase and thus is more cellular and vascular, making them appear red and raised. It is seen more in children and on flexor surfaces. A widened scar may appear in wounds perpendicular to the skin tension lines.

A keloid scar is defined as excessive scar tissue that extends beyond the boundaries of the original incision or wound and continue to enlarge after 6 months. They are of unknown cause, but they might be related to individuals with lower ability to break collagen, as studies have shown that people who form keloids have a lower level of α2-macroglobulin. lt is seen more in black women of middle ages and in an area of the body that represents a triangle with its angles being the xiphisternum, and the tips of both shoulders.

ULCER FISTULA & CYST

ULCERS

DEFINITION:

A break in the epithelial continuity OR discontinuity of the skin or mucous membrane which occurs due to the microscopic death of the tissues.

AETIOLOGY:

- **Vascular**: **1)** Venous Disease (Varicose Veins) .**2)** Arterial Disease; Large vessel (Atherosclerosis) or Small vessel (Diabetes).
- **Arteritis**: Autoimmune (Rheumatoid Arthritis, Lupus).
- **Trauma**.
- **Chronic Infection**: TB/Syphilis (1ry chancre - 2ry snail track ulcer - 3ry gumma).
- **Neoplastic**: Squamous or BCC, Sarcoma.

WAGNER'S GRADING OF ULCERS:

Grade 0 - Preulcerative lesion/healed ulcer.

Grade 1 - Superficial ulcer.

Grade 2 - Ulcer deeper to Subcutaneous tissue exposing soft tissue or bone.

Grade 3 - Abscess formation or osteomyelitis.

Grade 4 - Gangrene of part of tissues/limb/foot.

Grade 5 - Gangrene of entire one area/foot.

CLASSIFICATION:

A. CLINICAL:

- Spreading: (Edge - Inflamed & Edematous) No sign of healing.

- Healing: (Edge is sloping with healthy red granulation tissue & serous discharge) sign of healing.

- Callous: (Floor contains pale unhealthy granulation tissue with indurated edge), Very slow healing, see in DF.

B. PATHOLOGICAL:

1.**Nonspecific**

- **Traumatic Ulcer**
- **Arterial Ulcer**
- **Venous Ulcer**
- **Trophic ulcer**
- **Neurogenic Ulcer**
- **Infective Ulcer**
- **Diabetic Ulcer**

2.Specific

- **Tuberculosis**
- **Syphilis**
- **Actinomycosis**

3.Malignant

- **Squamous cell ca**
- **Basal cell ca**
- **Malignant melanoma**

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TRAUMATIC ULCER

1. Mechanical- Dental ulcer on tongue (jagged tooth) .

2. Physical- Electrical burn.

3. Chemical- Application of caustics. Like drinking alcohol or Kaz

Acute, Superficial, Painful, Tender.

TROPHIC ULCER

- Pressure Sore or Decubitus Ulcer.
- In pt bedridden OR paraplegia.
- Punched out edge with slough on the floor.
- Ex: Bed Sores & Perforating ulcers.
- Develop as a result of Prolonged Pressure.
- Sites: Ischial Tuberosity (اذا كان قاعد)>Greater Trochanter نائم على جنبه> Sacrum (نائم على ظهره) > Heel > Malleolus > Occiput.

VASCULAR ULCER

Arterial Ulcer	Venous ulcers
- **Caused due to peripheral vascular disease.** - **Lower Limb: Atherosclerosis.** - **UL: Cervical Rib, Raynaud's.** - **Chief complaint: Severe Pain.** - **Toes, Feet, Legs & UL Digits.**	- **Medial aspect of lower 3rd of lower limb**. <u>medial malleolus.</u> - Ankle (Gaiters Zone): **Chronic Venous HTN.** - Ulcers are **Painless.** - Varicose Veins or Post thrombotic (Phlebitic) limb (PTS)

- If the problem in the great saphenous vein it's medial venous ulcer.
- If the problem in the short saphenous vein it's lateral venous ulcer.
- After DVT patient may have chronic venous HTN-- venous ulcer.

TROPICAL ULCER

- Tropical regions: Africa, India, S.America
- Trauma or Insect Bite
- Fusobacterium fusiformis & Borrelia vincentii
- Abrasions, Redness, Papules & Pustules
- Severe Pain.

DIABETIC ULCER

• It may be caused due to diabetic Neuropathy(mostly) & Diabetic Microangiopathy.

• Increased Glucose: Increased Infection.

• Foot (Plantar), Leg, Back, Scrotum, Perineum.

• Ischemia then, Septicemia then, Osteomyelitis.

INVESTIGATIONS:

- CBC, ESR
- Urine and blood examination to rule out diabetes
- Chest X-ray - PA. view to rule out TB
- Pus for culture/sensitivity
- Lower limb angiography in cases of arterial diseases
- X-ray of the part to see for Osteomyelitis
- Biopsy: Non-healing/malignant ulcers

TREATMENT:

- Address cause
- Correct deficiencies
- Control pain, infection
- Debridement, dressing
- Closure of defect

Treatment of The Ulcers: clinical classification not pathological.

1)Treatment of Spreading Ulcers:

- Pus Culture/Sensitivity report
- Appropriate Antibiotics
- Repeated Dressings

2)Treatment of Healing Ulcers:

- Regular dressings are done for a few days
- Antiseptic creams like, Zinc Oxide or Silver Sulphadiazine.
- Culture swab is taken to rule out Streptococcus Haemolyticus (contraindication for skin grafting)
- Ulcer is
    - small - Heals by itself (Epithelialization)
    - Large - Free Split Skin Graft applied or flap.

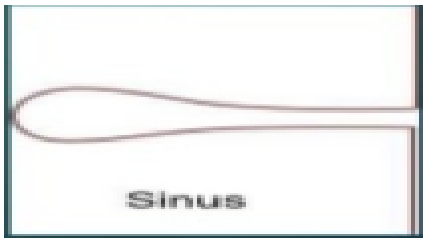
3)Treatment of Chronic Ulcers:

- These do not respond to conventional methods of treatment.
- The following are tried:
- Infrared radiation, short-wave therapy, ultraviolet rays decrease the size of the ulcer.
- Amnion helps in epithelialization.
- Chorion helps in granulation tissue.
- These ulcers ultimately may require skin grafting.

4)Treatment of The Underlying Disease

SINUS

Definition: **Blind track** lined by granulation tissue leading from epithelial surface down into the tissues. **Latin**: Hollow (or) a bay



CAUSE:

Congenital	Acquired
Preauricular sinus	• TB sinus • Pilonidal sinus • Actinomycosis

FISTULA

Definition:

Is an abnormal communication between two epithelium lined surfaces. (a pip or tube).

CAUSES:

a) congenital:

- Branchial fistula
- Tracheo-esophageal
- Umbilical
- Congenital AV fistula
- Thyroglossal fistula

b) Acquired:

- traumatic:
  A. following surgery: e.g., intestinal fistulas (faecal, biliary, pancreatic).
  B. following instrumental delivery (or) difficult labour e.g., vesicovaginal, rectovaginal, **ureterovaginal** fistula.
- Inflammatory: intestinal actinomycosis, tb, crohn

malignancy: when growth of one organ penetrates into the nearby organ. E.g., rectovesical fistula in carcinoma rectum

- **Iatrogenic**: avf for hemodialysis.

Fistula can be ether internal or external. (E.g.in the table below)

External	internal
Orocutaneous	Tracheo-esophageal
Appendicular	Colovesical
Thyroglossal	Rectovesical
Branchial	AVF (arteriovenous)
Enterocutaneous	Cholecystoduodenal

CAUSES FOR PERSISTENCE OF SINUS (OR) FISTULA:

- Presence of necrotic tissue underneath. e.g., sequestrum.
- Insufficient (or) non-dependent drainage. e.g., TB sinus
- Persistent drainage like urine/faeces/CSF
- Dense fibrosis
- Malnutrition Specific causes. e.g., TB, actinomycosis
- Ischemia, Drugs. e.g., steroids
- interference by the patient

can be abbreviated in **HIS FRIEND:**

- High-output fistula (<500 cc/day)
- Intestinal destruction (50% of circumference).
- Short segment fistula > 2.5 cm
- Foreign body. E.g., suture material
- Radiation
- Infection
- Epithelialisation (colostomy) (or) endothelisation of the track. E.g., avf
- Neoplasm
- Distal obstruction. E.g., faecal (or) biliary fistula

Clinical Features:

- Usually asymptomatic but when infected manifest as Recurrent/ persistent discharge.

- Pain.
- Constitutional symptoms if any deep-seated origin.

INVESTIGATIONS

- CBP (Hb, Total Leucocytosis, Differential Leucocytosis, ESR).
- Discharge for C/S, AFB (acid fast bacilli), cytology, Gram staining.
- X-RAY of the part to rule out OM, foreign body.
- X-RAY KUB and U/S abdomen in cases of lumbar fistula to rule out staghorn calculi.
- MRI (gold stander).
- BIOPSY from edge of sinus, if non healing
- CT Sinusogram
- Fistulography/ Sinusography:
    - For knowing the exact extent/origin of sinus (or)fistula.
    - Water soluble or ultrafluid lipoidal iodine dye is used.
    - Lipoidal iodine is poppy seed oil containing 40% iodine

Treatment Basic Principles:

- Antibiotics, if infection.
- Adequate excision, after excision specimen SHOULD be sent for HPE.
- Adequate drainage.
- Treating the cause. e.g. ATT (Antitubercular therapy) for TB sinus.
- removal of any foreign body.
- sequestrectomy for OM.

CYST

DEFINITION:

abnormal sac or closed cavity lined with epithelium and filled with fluids, air, or semi-solid material.

Classification of Cysts:

A- According to lining endothelium:

- True Cysts are usually lined by epithelium or endothelium, if infections supervene, the lining may be composed of granulation tissue. Like: Thyroglossal cyst, Dermoid cyst, and Bartholin cyst.
- False Cysts, they are usually exudation (discharge) & degeneration cysts. Like: cystic degeneration in a tumour.
- Pseudo-cyst of the pancreas is an encysted collection of fluids in the lesser sac.

B- According to ethology:

1. Congenital

- Sequestration dermoid: due to displacement of epithelium along the suture line during closure.
- Tubulo-embryonic (tubulo-dermoid) : due to
    - Abnormal budding e.g. Thyroglossal cyst.
    - Dilation of vestigial remnants e.g. Urachal cyst, vitello-intestinal cysts,
    - Hydatid cyst of Morgagni and branchial cysts.
- Cysts of embryonic remnants

2. Acquired:

- Retention: Due to Blocking of a glandular duct e.g. Sebaceous cyst, ranula, Bartholin's gland cyst or hydronephrosis.
- Distension: e.g. Dilated acini of thyroid follicles or in the ovary or lymphatic cyst as cystic hygroma.
- Exudation: exudation into an anatomical space already lined with epithelium e.g. Hydrocele.
- Trauma: hematoma may resolve into a cyst which becomes lined with endothelium (cure requires excision not aspiration).
- Cystic swellings
- Implantation
- Degeneration

3. Parasitic:

- Hydatid

- Trichiniasis
- Cysticercosis

CLINICAL FEATURES OF A CYST:

1. Swelling.
2. Fluctuation.

COMPLICATIONS:

1. Infections (abscess).
2. Haemorrhage.
3. Torsion.
4. Calcification
5. Cachexia Ovarica

HEMORRHAGE

Hemorrhage is the most common cause of shock in the surgical patients. Because of the special precautions that should be taken while managing a patient with shock due to hemorrhage, it merits some attention.

CAUSES OF HEMORRHAGE

The causes of hemorrhage are all too many, they include loss of blood due to surgery, trauma, spontaneously, coagulopathy or any form of blood diseases, blood vessel diseases or the use of drugs.

CLASSIFICATION

The classification of hemorrhage usually depends on the timing, location, according to severity and amount, and on the type of the source vessel (artery or vein, not very important).

ACCORDING TO TIMING

Primary hemorrhage is hemorrhage occurring immediately as a result of an injury (or surgery). Reactionary hemorrhage is delayed hemorrhage (within 24 hours) and is usually caused by dislodgement of clot by resuscitation, normalization of blood pressure and vasodilatation or from a technical failure such as slippage of a ligature. Secondary hemorrhage is caused by sloughing of the wall of a vessel, and it usually occurs 7-14 days after injury and is precipitated by factors such as infection, pressure necrosis (such as from a drain) or malignancy.

ACCORDING TO LOCATION

Hemorrhage may be revealed or concealed. Revealed hemorrhage is a visible usually external hemorrhage, such as bleeding from an open arterial wound or hematemesis in a patient with a duodenal ulcer. Concealed hemorrhage is contained within the body cavity (intrabdominal, intrathoracic) or within a soft tissue (retroperitoneal, subcutaneous) and must be suspected, actively investigated and controlled. Examples of concealed hemorrhage include gastrointestinal bleeding and ruptured aortic aneurysm.

ACCORDING TO THE SEVERITY OR AMOUNT LOST

There is about 70 ml of blood in each kg body weight of an adult. This equates to about 5 liters of blood, approximately.

The estimation of blood lost by measuring the amount of blood, is very inaccurate. It is usually an under estimation. Estimation of lost blood is done through measuring the amount lost in soaked swabs and in suction tubes in the operating room. The degree of hemorrhage can be classified into classes 1-4 based on the estimated blood loss required to produce certain physiological compensatory changes (see the table next page). However, it is better to depend on the clinical features (vital signs, preload assessment, base deficit, urine output) rather than the amount lost, to estimate severity.

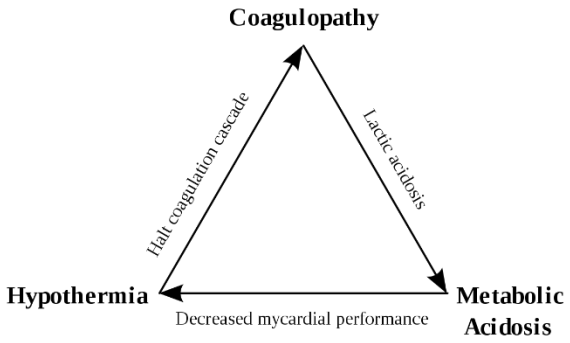
PHYSIOLOGICAL BASIS OF TREATMENT

Hemorrhage is treated by arresting the bleeding, and not by fluid resuscitation or blood transfusion, hemorrhage will result in a state of shock that will result in *acidosis*. The acidosis will alter the coagulation factors structure and function and thus results in *coagulopathy*. The coagulopathy will result in further bleeding. Because one of the compensatory mechanisms to the shock state, is vasoconstriction of the skeletal muscle vessels, the heat that these muscle produce will not be transmitted to the rest of the body, which will cause the body to go into *hypothermia,* further affecting the function of the coagulation factors which will result in further bleeding and acidosis. This vicious circle of acidosis, coagulopathy and hypothermia is the cause of death in patients with hemorrhage, and is termed the death triad.

Resuscitating the patients with fluids before stopping the site of hemorrhage from bleeding will be catastrophic. Giving more fluid will increase the blood pressure and cause further and more bleeding, that is plus the effect of diluting the coagulation factors in the blood. Intra-venous blood and fluids are cold and exacerbate hypothermia, bleeding and many crystalloid fluids are themselves acidic (e.g. normal saline has a pH of 6.7). This will accelerate the process of the death triad. Thus,

Classification of hemorrhage according to severity				
Class	Amount lost	Severity	Clinical features	Urine output
Class I	<15%	Mild	Pallor, Cool extremities, Diaphoresis (cold sweats), Diminished capillary refilling (> 2 seconds), Collapsed subcutaneous veins	Normal
Class II	15-30%	Moderate	As with mild, plus tachycardia	Decreased (< 5
Class III	30-40%	Moderate-severe	and postural hypotension, lethargy	ml/kg/hr)
Class IV	>40%	Severe	As with moderate, but there is mental changes, restlessness or apathy	Decreased (< 5 ml/kg /hr)

before starting any fluid therapy the bleeding should be stopped.



MANAGEMENT

IDENTIFY THE SITE

Identification of the site of hemorrhage is the first step. If the site is external (i.e. revealed), then direct pressure on the site of bleeding is important, if not (i.e. concealed), then rapid investigations considered. The type of investigation is directed by a brief history and examination. For example, a patient who comes with a history of a road traffic accident and marks of seat belt on the abdomen, should have a diagnostic peritoneal lavage, to identify whether there is bleeding in the abdomen, or a patient with history of NSAIDs use and a tarry black stool (i.e. melena), will require endoscopy.

Once the bleeding site (not the exact site, instead the source) is identified, proper methods of treatment are carried through (e.g. endoscopy, surgery).

Surgery may need to be limited to the minimum necessary to stop bleeding and control sepsis. More definitive repairs can be delayed until the patient is physiologically capable of sustaining the procedure. This concept of tailoring the operation to match the patient's physiology and staged procedures to prevent physiological exhaustion is called 'damage control surgery'. Once hemorrhage is controlled, patients should be aggressively resuscitated and warmed and coagulopathy corrected.

RESUSCITATION

Once bleeding is controlled, the next step, is restoration of tissues perfusion. This is achieved by installing two short wide bore I.V lines. Blood for CBC, urea arid electrolytes, ABGs and cross matching is withdrawn. O negative packed red cells should be present.

In the case of hemorrhage, the ideal fluid to replace is blood. However, it is in some cases not available, thus crystalloid is required. Crystalloids replacement should follow the 3:1 or 4:1 whereby every one litre of blood is replaced by 3 or 4 litres of crystalloid fluids. This is because only about one quarter to one third of the volume of the crystalloid fluids

remains in the intravascular compartment after distribution. The intravascular compartment is the one that needs to be resuscitated, the amount is calculated according to the severity of hemorrhage.

It is more proper to order blood after the results of the hemoglobin level. Usually, blood transfusion is needed when the haematocrit is less than 30%. However, when the fluid therapy has been enough in resuscitating the patient, avoiding blood transfusion is better.

In patients with concealed hemorrhage, a bolus of fluid is given, the patient should be checked for being a responder or a transient responder (see the lecture on shock). Transient responders are active bleeders.

Inotropic drugs are given after hypovolemia is corrected.

MONITORING

Further monitoring of the patient with heart rate (ECG), blood pressure (non-invasively), urine output, central venous pressure and serum lactate and base deficit levels is very important.

Restoring tissue oxygenation with a face mask is also very important, the results of the arterial blood gas analysis will give the indication for using mechanical ventilation.

SHOCK

Shock is a state of sever physiological abnormality associated with abnormal cellular metabolism due to decreased tissues perfusion. It is an emergency state that is responsible for most of the deaths in the surgical field, but is encountered in every other field of medicine.

PATHOPHYSIOLOGY

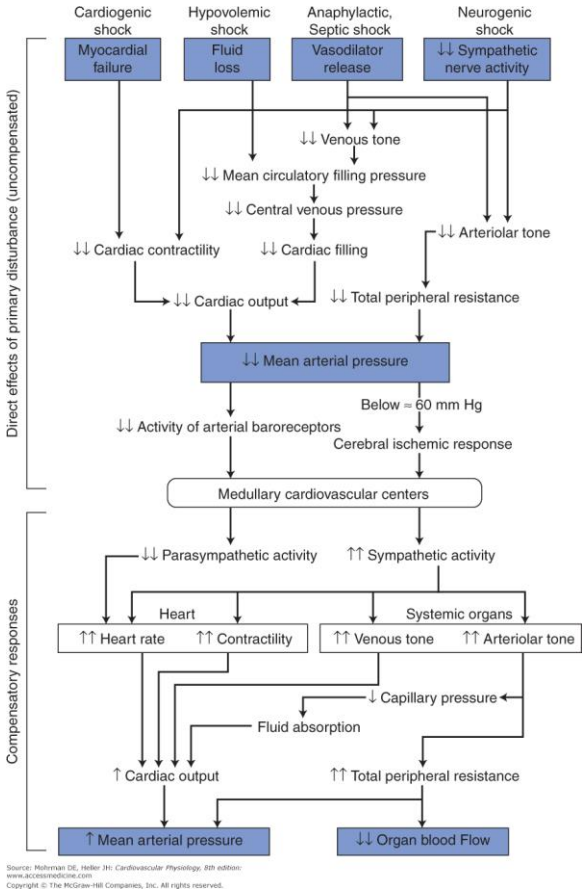
ORGAN SYSTEMS

the hallmark of shock is *decreased tissues perfusion*. This may result from many causes (see later), but in most of them (except in sepsis), there is low cardiac output. The low cardiac output, will result in an increased sympathetic activity (from carotid baroreceptors and adrenal catecholamines) which causes *tachycardia and* systemic *vasoconstriction*. Along with the metabolic acidosis (see later) and hypoxia, this will also *increase the respiratory rate* The low cardiac output (or the decrease in peripheral vascular resistance as is seen in sepsis and other forms of distributive shock) will activate the renin-angiotensin system which will increase sodium and water retention; and cause vasoconstriction. Vasopressin (antidiuretic hormone (ADH)) is also released due to a decrease in blood volume and causes increased water reabsorption from the kidneys. The vasoconstriction will shift the blood from organs such as the skin, gastrointestinal tract, liver and spleen toward vital organs such as the heart and brain (which do not respond to vasonctrictors, but instead to locally produced vasodilators). The water and salt retention, will prevent further loss of fluid from the kidneys.

These mechanisms can overcome the decrease in blood pressure to a certain extent (compensated shock), but when they are overwhelmed by further loss of blood volume, a state of shock (or decompensated shock) is said to occur, the deterioration may continue if not untreated, to involve more than 2 major systems of the body (multiple organ failure).

CELLULAR LEVEL

At the cellular level, the decreased oxygen delivery to the cells will result in the shift to anaerobic metabolism, with its product, lactic acid. Its accumulation in the blood will result in metabolic acidosis.



Once glucose is consumed by this process, ATP depletion will take place and the NA^+-K^+ ATPase pump will cease to function. This will cause accumulation of sodium and water inside the cell (causing it to swell), and potassium outside the cell. More potassium is released into the blood stream after cells are damaged by the action of hydroleases released from lysosomes (autodigestion).

MICROCIRCULATION

The local changes of ischemia and acidosis will activate the the coagulation system and cause small clots (which may further deteriorate the local cells status). They will also activate the immune system and cause the generation of cytokines and oxygen radicals at the local level.

STAGES OF SHOCK

COMPENSATED SHOCK

This term applies to the loss of fluid in amounts (usually <15% of blood volume) that still allow the compensatory mechanisms to overcome the drop in

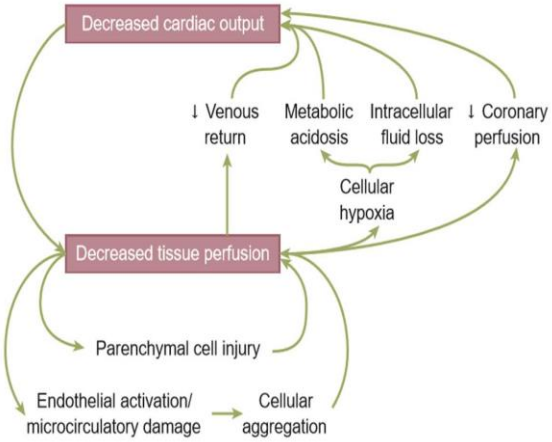


Figure . The "vicious cycle of shock." Regardless of the etiology, decreased tissue perfusion and shock results in a feed-forward loop that can exacerbate cellular injury and tissue dysfunction.

blood pressure, patients often have tachycardia with cool peripheries with a mild decrease or no change in urine' output;

DECOMPENSATED SHOCK

When blood volume becomes lost so severely, that compensatory mechanisms are not effective in maintaining the blood pressure, the term 'decompensated shock' becomes evident.

According to the degree of blood volume decrease, clinical features are described. Patients with mild) shock have tachycardia, tachypnoea, fall in pulse pressure, cool sweaty peripheries, and mild anxiety; Those with moderate have the same manifestations, but start to have a low blood pressure, low urine output (< 0.5 ml/kg/hr) and are usually drowsy or confused; Those with severe shock, have hypotension, zero urine output (anuria) and are unconscious.

In all of those, the capillary refill time (the time taken to restore the colour of the nail after pressure) is usually increased, except in sepsis (and other causes of distributive shock, see later).

IRREVERSIBLE SHOCK

The term irreversible shock applies to patients who became shocked for a long time, so that they will not respond to therapy. This is interchangeable with the term 'unresuscitable shock'. Several mechanisms have been proposed for explaining this phenomenon including;

- At the microvascular level, capillary permeability increases after endothelial damage (due to prolonged hypoxia) resulting in local tissue edema and further compromise of tissue perfusion. Also, cells may swell from the dysfunction of the NA^+-K^+ ATPase pump (explained earlier).

- Myocardial depression occurs due to prolonged hypoxia and the release of toxins (in some forms of shock).

The failure of the circulation will make the patient unresponsive to fluid therapy, and the myocardial depression will make the patient unresponsive to inotropic therapy. Thus, the term 'unresuscitable'.

MULTIPLE ORGAN FAILURE

Prolonged untreated shock may result in damage to multiple organ systems (more than 2 organ systems).

The effects on the specific organ systems is shown in the table below. Management in this case is supportive.

Effects of shock on specific organ system
• **Lung; Acute respiratory distress syndrome (ARDS)**
• **Kidney; Acute renal insufficiency (acute tubular necrosis (ATN))**
• **Liver; Acute liver insufficiency**
• **Clotting; Coagulopathy**
• **Brain; neuronal damage (coma)**
• **Cardiac; Cardiovascular failure**

TYPES OF SHOCK

HYPOVOLEMIC SHOCK

Hypovolemic shock is caused by a reduced circulating volume. Hypovolemia may be due to hemorrhagic or non hemorrhagic causes.

Non-hemorrhagic causes include poor fluid intake' (dehydration) and excessive *fluid* loss. because of vomiting, diarrhoea, urinary loss (e.g. diabetes), evaporation and **'third-spacing',** in which fluid is lost into the gastrointestinal tract and interstitial spaces, as for example in bowel obstruction or pancreatitis. *Plasma and fluid* loss are seen in burns.

Hemorrhagic shock (the most common type of hypovolemic shock) is seen after trauma; spontaneous bleeding;(e.g. gastrointestinal bleeding), and loss of blood after surgeries.

In the management of shock, the cause should be assumed hypovolemic until proven otherwise.

CARDIOGENIC SHOCK

Cardiogenic shock is due to primary failure of the heart to pump blood to the tissues. It is caused by any disease that affects the heart muscle, most commonly, ischemic heart disease (which manifests as this, when more than 50% of the wall of the ventricles is damaged).

Cardiogenic shock usually occurs in association with *venous hypertension', generalized edema, or pulmonary edema the* pulmonary edema in these patients will exacerbate the condition.

OBSTRUCTIVE SHOCK

This usually occurs due an obstruction of the blood inflow to the heart, decreasing the preload, as occurs most commonly in cardiac tamponade, tension pneumothorax and massive pulmonary embolism or air embolism. In this form there is also signs of venous hypertension.

DISTRIBUTIVE SHOCK

Distributive shock describes the pattern of cardiovascular responses characterized by inadequate organ perfusion accompanied by vascular dilatation with hypotension, *low systemic vascular resistance)* inadequate afterload and a resulting abnormally *high cardiac output.*

In anaphylaxis (anaphylactic shock), vasodilatation is caused by histamine release, whereas in high spinal cord injury (neurogenic shock) there is failure of sympathetic outflow arid adequate vascular tone. The terms psychogenic shock and vasovagal shock, falls under this classification.

The cause in sepsis (septic shock) is less clear but is related to the release of bacterial products (endotoxins contained with the cell wall of gram negative bacteria, or exotoxins released by gram positive bacteria) and the activation of cellular and humoral components of the immune system. The released inflammatory mediators will cause systemic vasodilatation. There is maldistribution of blood flow at a microvascular level with *arteriovenous shunting* and *dysfunction of the cellular utilisation of oxygen:* this most commonly affects children, people of old age and those who are immunocompromised. The mortality rate is high reaching 50%.

Sepsis most commonly follows catheterization in the urinary tract, badly infected wounds (i.e. dirty wounds), abscesses, peritonitis and strangulated bowel.

Patients often exhibit high grade fever. In the later phases of septic shock there is hypovolemia from fluid loss into the interstitial spaces and there may be concomitant myocardial depression, which complicates the clinical picture, thus patients may have a mixed picture of hypovolemic, distributive and cardiogenic shock. The development of micro-blood clots and lead to consumptive coagulopathy and disseminated intravascular coagulation (DIC).

Patients who develop sepsis usually have evidence of what is referred to as the systemic inflammatory response syndrome (SIRS, see the table below). One can say that sepsis is SIRS with a documented infection.

systemic inflammatory response syndrome
Two or more of the following
• hyperthermia (> 38 °C) hypothermia (< 36 °C)
• tachycardia (> 90 min/l)
• tachypnea (> 20 /min) or $PaCO_2$ < 32 mmHg
• white cell count > 12 x 10^9/l or < 4 x 10^9/l
• immature bands

MANAGEMENT

immediate recognition of the patient with shock is of utmost importance for saving his/her life. The clinical examination is usually not perfect, but is enough. It is safer to assume that the patient is in a state of shock and start resuscitation, rather than waiting for further investigations.

GENERAL MEASURES

General measures such as laying the patient fiat in bed and raising their legs, using antishock garment or trousers will increase the venous return to the heart and may have some benefits. Keeping the patient warm is important.

HYPOVOLEMIC SHOCK

Patients with shock should be treated as having hypovolemia until proven otherwise. Initially venous access is important using 2 short, wide bore cannulas (16 G or larger) that will allow rapid infusion. Patients should be provided with oxygen. If vasoconstriction is intense, access may be difficult and central vein cannulation or a formal cut-down should be considered. This is performed in the antecubital fossa or on to the long saphenous vein in front of the medial malleolus.

Before starting infusion of fluid, the site of bleeding (assuming first that its hypovolemic shock due to hemorrhage) should be identified and controlled very well. This is very important, because starting fluid therapy without controlling the bleeding source will increase the bleeding from that site (as fluids increase blood pressure), and the fluids if given in large amounts, will dilute the haemoglobin and coagulation factors; the results would be catastrophic.

The type of replacement fluid is not very important, there is little privilege of using crystalloids'(ringer lactate, Hartman's solution, normal saline) over colloids (albumin and others) in terms of efficacy of volume expansion, complications and price. When the fluid lost is blood, the ideal fluid to be used is blood; but crystalloids may be used until the blood is prepared. The shock status can be determined dynamically by the cardiovascular response to the rapid administration of a fluid bolus. In total, 250-500 ml of fluid is rapidly given (over 5-10 min) and the cardiovascular responses in terms of heart rate, blood pressure and central venous pressure (CVP) are observed. Patients can be divided into;

- Responders show an improvement in their cardiovascular status, which is sustained. These patients are not actively losing fluid but require filling to a normal volume status.
- Transient responders show an improvement but then revert to their previous state over the next 10-20 min. These patients either have moderate on-going fluid losses (either overt hemorrhage or further fluid shifts reducing intravascular

Distinction among the types of shock

	Hypovolaemic	Cardiogenic	Septic	Anaphylactic
Skin colour	**Pale**	**Pale**	**Flushed**	**Urticarial rash**
Sweating	**Present**	**Present**	**Absent**	**Absent**
Temperature	**Cold**	**Cold**	**Warm**	**Warm**
Capillary refill	**Slow**	**Slow**	**Rapid**	**Normal or rapid**
Central venous pressure	**Low**	**High**	**Low**	**Low**
Mental status	**Restless**	**Quiet**	**Drowsy**	**Variable**

volume), these patients require operative intervention, to identify the site of bleeding and stop it.

- Non-responders are severely volume depleted and are likely to have major on-going loss of intravascular volume, usually through persistent uncontrolled hemorrhage.

The use of inotropic agents should not be used as first line therapy, because the inotropic action may consume the oxygen and energy stores (by stimulation and decreasing the preload and thus coronary perfusion) in the heart muscle and cause myocardial depression if the hemodynamic status is not restored first.

CARDIOGENIC SHOCK

Patients should be provided with oxygen and sedation. inotropic drugs (dobutamine) are used in this case, if hypovolemia turned out to be absent.

SEPTIC SHOCK

Oxygen is provided for those patients. Intubation and' mechanical ventilation should be considered in obtunded patients.

Patients should be thoroughly resuscitated using crystalloid solutions with their central venous pressure monitored (see below). Broad spectrum antibiotics should be started once the patient is stable and samples from the blood and all body fluids (including wounds) are collected for culture. Haemolysis can result from sepsis, and blood transfusion is important in this case.

Vasopressor agents (phenylephrine, dopamine (< 5 mg/kg/min), noradrenaline) are indicated in sepsis and other distributive shock states. In cases refractory to treatment with vasopressors, the use of glucocorticoids (100 mg IV q 8 hours for 24-48 hours) may improve the response, as it sensitizes the cells to catecholamine's. the use of this was also justified based upon the finding, that cases of sepsis are associated with adrenal insufficiency. Vasopressin' may be used as an alternative vasopressor in resistant cases.

In cases of distributive shock due to anaphylaxis, the cause (e.g. drugs) should be stopped. Patients should be given vasopressors, antihistamines and glucocorticoids and properly managed with parenteral fluids.

METHODS OF MONITORING

The patient's heart rate (ECG), peripheral perfusion (pulse oximetry) and non-invasive blood pressure monitoring provide a very

good continuous monitoring of the patient cardiovascular status and at least they should be provided. The level of consciousness is also a good continuous monitor of brain perfusion.

- Urine output; the transurethral insertion of a bladder catheter connected to a graduated collecting device will allow an *hourly* measurement of urine output (normally > 0.5 ml/kg/hr). his can be used as an indirect measurement of vital organ perfusion: namely, that of the kidney.

- Central venous pressure (CVP); a catheter is inserted into the superior vena cava and provides a secure route of administering fluids, and measurement of the pressure inside the right atrium. CVP is also used when the vasoconstriction in the peripheries is intense, so that peripheral lines cannot be inserted. The normal rise of CVP after (5-10 minute) a fluid bolus (250-500) is 2-5 cmH_2O (euvolemic response), when there is no rise, this indicates that patients require more fluids (hypovolemic response). If there is a sustained rise, this indicates a large preload, and a form of cardiac insufficiency (hypervolemic response).

- Metabolic monitoring; In addition to venous blood being sent for full blood count and cross-matching, urea and electrolyte estimation should be undertaken. Blood gas analysis is important for showing hypoxia (give mask) and hypercarbia (may require ventilation). Serum lactate and base deficit are increased in shock. They are good indicators that the patient is stable, as they are measured routinely, but they do not provide minute- minute monitoring of therapeutic response. They also provide a good indicator of occult hypoperfusion (see later).

- Cardiac output; measured by pulmonary catheters and Doppler ultrasound can help distinguish the type of shock (cardiogenic) and can monitor the cardiovascular response to fluid therapy by real time monitoring.

ISCHEMIA-REPERFUSION SYNDROME

During the period of systemic hypoperfusion, cellular and organ damage progresses because of the direct effects of tissue hypoxia and local activation of inflammation. Further injury occurs once normal circulation is restored to these tissues. The acid and potassium load that has built up can lead to direct myocardial depression, vascular dilatation and further hypotension. The cellular and humoral elements activated by the hypoxia (complement, neutrophils, microvascular thrombi) are flushed back into the circulation where they cause further endothelial injury to organs such as the lungs and kidneys. This leads to acute lung injury, acute renal injury, multiple organ failure and death.

Reperfusion injury can currently only be attenuated by reducing the extent and duration of tissue hypoperfusion.

END-POINTS OF RESUSCITATION

Traditionally patients have been resuscitated until they have a normal pulse, blood pressure and urine output; however, these parameters are monitoring organ systems whose blood flow is preserved until the late stages of shock. Therefore, a patient may be resuscitated to restore central perfusion to the brain, lungs and kidneys and yet the gut and muscle beds continue to be underperfused. Thus, activation of inflammation and coagulation may be on-going and, when these organs are finally perfused, it may lead to reperfusion injury and ultimately multiple organ failure.'

This state of normal vital signs and continued underperfusion is termed occult hypoperfusion (OH). With current monitoring techniques it is manifested only by a persistent lactic acidosis. The duration that patients spend in this hypoperfused state has a dramatic effect on outcome. Patients with OH for more than 12 hours have a two to three times higher mortality rate than that of patients with a limited duration of shock.

Resuscitation algorithms directed at correcting global perfusion endpoints (base deficit, lactate, mixed venous oxygen saturation) rather than traditional endpoints have been shown to improve mortality and morbidity in high-risk surgical patients.

SEPSIS ANS SIRS

Sepsis as a word means "rot"

FEW DEFINITIONS:

Infection: Microbial phenomenon characterized by an inflammatory response to the presence of microorganisms or the invasion of normally sterile host tissue by those organisms.

Inflammation: Is the term used to describe how blood vessels & immune cells react to injury >> allows inflammatory cells, plasma proteins, and fluid to exit blood vessels >> and enter the interstitial space.

Acute:

Immediate response with limited specificity

Arises in response to 2 important stimuli:

1- Infection "particularly bacterial ": To eliminate the pathogen.

2- Necrosis: The goal is to eliminate debris.

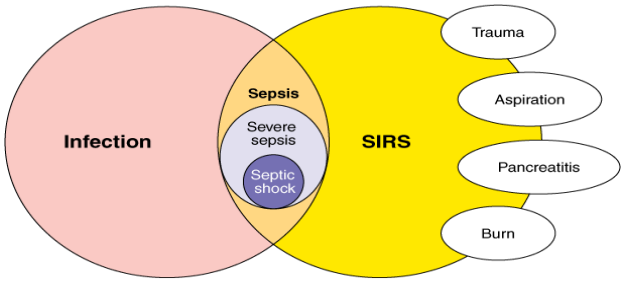
Chronic:

There is Antibodies and T-cells to a very specific Antigen

5 cardinal signs of inflammation: rubor, tumor, dolor, calor and functio laesa

Bacteremia: presence of viable bacteria in the bloodstream

Systematic inflammatory response syndrome: defined as clinical response to a nonspecific insult of either infectious or noninfectious origin.



Sepsis: is the systemic response to infection and is defined as the presence of SIRS in addition to a documented or presumed infection

SIRS defined as the presence of two of the following:

1- Fever of more than 38°C or less than 36°c .

2- Heart rate of more than 90 beats per minute.

3- Respiratory rate of more than 20 breaths per minute or arterial carbon dioxide tension ($PaCO_2$) of less than 32 mm Hg.

4- Abnormal white blood cell count >12,000/μL or < 4,000/μL or >10% band B cells

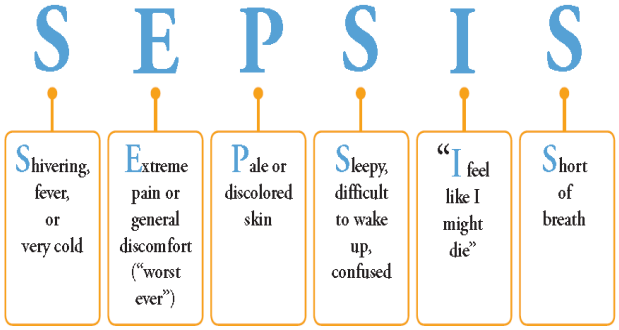
Sepsis causes a drop of a systolic blood pressure of less than 90 mm Hg or a reduction of more than 40 mm Hg from baseline in the absence of other causes of hypotension and despite adequate fluid resuscitation. (septic shock)

causes of sepsis Like: pneumonia, urinary tract infection, peritonitis …etc.

SIGNS AND SYMPTOMS OF SEPSIS:

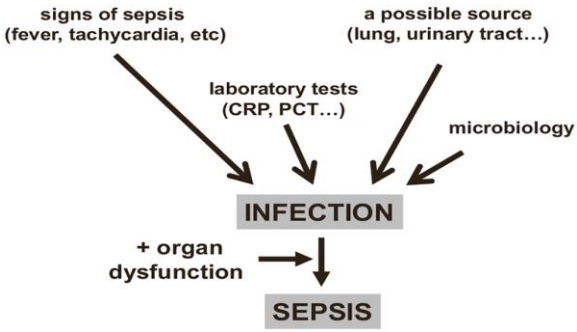
- Signs of shock (hypotension, oliguria, lactic acidosis..etc)
- fever or may be hypothermic (hypothermia is more common in the very young, elderly, debilitated, and immunocompromised)

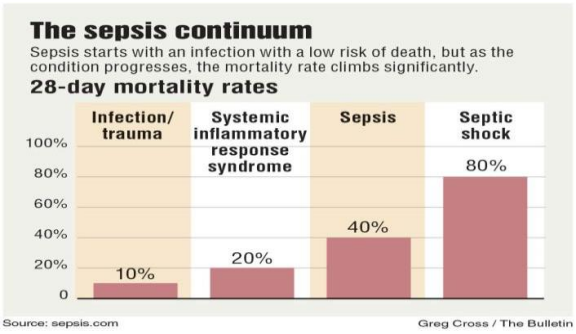
MULTIPLE ORGANS DYSFUNCTION (MODS):



the presence of altered organ function in an acutely ill patient so that homeostasis cannot be maintained without intervention and it is of two types:

- 1-Primary MODS: a well-defined insult, occurs early and can be directly associated to the insult itself (e.g., renal failure due to rhabdomyolysis).
- 2-Secondary MODS: not in direct response to the insult itself, but as a consequence of a host response (SIRS)





The mortality increase when the condition progress..

Markers of sepsis:

- Procalcitonin:

A hormone that is synthesized by the parafollicular C cells of thyroid:

- normal level <0.05ng/ml
- <0.5 rules out infection
- 0.5-2 suspected sepsis
- >2- 100 sepsis severity

The rise in concentration start after 6 hrs. and the t1/2 is 24 hrs.

- C-reactive protein (CRP): acute phase reactant used to assess the severity of sepsis

normal value <6mg/l

Differentiate between bacterial &viral infection

Rises in concentration up to 1000-fold in the blood in response to infection

TREATMENT STRATEGIES FOR SEVERE SEPSIS:

- The early goal directed therapy
- Lung protective ventilation
- administration of adequate antibiotic therapy
- Steroids
- Activated protein C
- goal directed hemodynamic resuscitation
- Maintenance of normal glucose level (4-6 mmol/L)

The goals during the first 6 hours of resuscitation should be:

1. CVP 8-12mm Hg (12-15 on ventilated patients)
2. Mean arterial pressure > 65 mm Hg
3. Urine output > 0.5 mL/kg/hr

- Transfusion of packed red blood cells to a hematocrit of 30% and/or dobutamine infusion (to maximum 20 µg/kg/min) are considered optional therapies
- Fluid Therapy

A fluid administration is continued as long as the hemodynamic improvement continues

(e.g., arterial pressure, heart rate, urine output)

The rate of fluid administration be reduced when cardiac filling pressures (central venous pressure or

pulmonary artery balloon-occluded pressure) increase without concurrent hemodynamic improvement

- Target a tidal volume of 6 mL/kg in patients with (acute lung injury/adult respiratory distress syndrome).
- Target an initial upper limit plateau pressure 30 cm H2O. Consider chest wall compliance when assessing plateau pressure

❖ If plateau pressure remain > 30 after reduction of tidal volume to 6 ml/kg, tidal volume should be reduced further to as low as 4 ml/kg

❖ Start antibiotic therapy in the first hour of recognition of severe sepsis after obtaining appropriate cultures

❖ Empiric combination therapy includes metronidazole plus either levofloxacin, aztreonam, cefepime, or ceftriaxone.

❖ If fluid administration fails to restore adequate blood pressure and organ perfusion; Mean arterial pressure (MAP) be maintained (≧ 65) .

❖ The first line agents to correct hypotension in septic shock are norepinephrine or dopamine

❖ Norepinephrine more potent than dopamine and more effective at reversing hypotension

❖ Dopamine is preferred in patients with compromised systolic function but it causes many side effects like tachycardia and is arrhythmogenic

❖ Epinephrine is the alternative agent in patient that is poorly responsive to norepinephrine or dopamine

❖ Consider intravenous hydrocortisone for adult septic shock when hypotension responds poorly to adequate fluid resuscitation and Vasopressors

❖ Administer intravenous hydrocortisone 200-300 mg/day for 7 days in three or four divided doses or by continuous infusion

❖ Shown to reduce mortality rate in patients with relative adrenal insufficiency

❖ Do not use corticosteroids to treat sepsis in the absence of shock unless the patient's endocrine or corticosteroid history warrants it

❖ Activated protein C had anti-thrombotic, anti-inflammatory and pro-fibrinolytic properties

❖ recommended in patients at a high risk of death

❖ Sepsis-induced multiple organ failure, or

❖ Septic shock, or

❖ Sepsis induced acute respiratory distress syndrome

❖ Treatment should begin as soon as possible once patient identified as high risk of death

❖ Patients should have no absolute or relative contraindication related to bleeding risk

BLOOD TRANSFUSION

The process of transferring blood or blood products from one person to the circulatory system of another. the first successful transfusion in 1818

BLOOD AND BLOOD PRODUCTS

Blood is collected from donors who have been previously screened before donating.

In the UK, up to 450 mL of blood is drawn, a maximum of three times each year. Each unit is tested for evidence of hepatitis B, hepatitis C, HIV-1, HIV-2 and syphilis. Donations are leukodepleted as a precaution against variant Creutzfeldt–Jakob disease (this may also reduce the immunogenicity of the transfusion).

The ABO and rhesus D blood group is determined, as well as the presence of irregular red cell antibodies. The blood is then processed into subcomponents.

INDICATIONS FOR BLOOD TRANSFUSION

Blood transfusions should be avoided if possible, and many previous uses of blood and blood products are now no longer considered appropriate use. The indications for blood transfusion are as follows:

• acute blood loss, to replace circulating volume and maintain oxygen delivery;

• perioperative anaemia, to ensure adequate oxygen delivery during the perioperative phase;

• symptomatic chronic anaemia, without haemorrhage or impending surgery

Whole blood	**Rarly available. whole blood transfusion has significant advantages over packed cells as it is coagulation factor rich and, if fresh, more metabolically active than stored blood.**
Packed RBC	Packed red blood cells are spun-down and concentrated packs of red blood cells. Each unit is approximately 330 mL and has a haematocrit of 50–70 per cent .the shelf life of red blood cells is 42 days
Fresh-frozen plasma (FFP)	Fresh-frozen plasma (FFP) is rich in coagulation factors and is removed from fresh blood and stored at −40 to −50°C with a two-year shelf life. It is the first-line therapy in the treatment of coagulopathic haemorrhage
Cryoprecipitate	Cryoprecipitate is a supernatant precipitate of FFP and is rich in factor VIII and fibrinogen. It is stored at −30°C with a two year shelf life. It is given in low fibrinogen states or factor VIII deficiency.
Platelets	Platelets are supplied as a pooled platelet concentrate and contain about 250 × 109/L. Platelets are stored on a special agitator at 20–24°C and have a shelf life of only 5 days. Platelet transfusions are given to patients with thrombocytopenia or with platelet dysfunction who are bleeding or undergoing surgery.
Prothrombin complex concentrates	Prothrombin complex concentrates (PCC) are highly purified concentrates prepared from pooled plasma. They contain factors II, IX and X. Factor VII may be included or produced separately. It is indicated for the emergency reversal of anticoagulant (warfarin) therapy in uncontrolled haemorrhage

TRANSFUSION TRIGGER

Historically, patients were transfused to achieve a haemoglobin >10 g/dL. This has now been shown to not only be unnecessary but also to be associated with an increased morbidity and mortality compared to lower target values. A haemoglobin level of 6 g/ dL is acceptable in patients who are not actively bleeding, not about to undergo major surgery and are not symptomatic. There is some controversy as to the optimal haemoglobin level in some patient groups, such as those with cardiovascular disease, sepsis and traumatic brain injury. Although conceptually a higher haemoglobin improves oxygen delivery, there is little clinical evidence at this stage to support higher levels in these groups

Perioperative red blood cell transfusion criteria.	
<6	Probably will benefit from transfusion
6–8	**Transfusion unlikely to be of benefit in the absence of bleeding or impending surgery**
>8	**No indication for transfusion in the absence of other risk factors**

COMPLICATIONS OF BLOOD TRANSFUSION

Nonhemolytic Reactions. Febrile, nonhemolytic reactions are defined as an increase in temperature (>1°C) associated with a transfusion and are fairly common (approximately 1% of all transfusions). Preformed cytokines in donated blood and recipient antibodies reacting with donated antibodies are postulated etiologies. The incidence of febrile reactions can be greatly reduced by the use of leukocyte-reduced blood products. Pretreatment with acetaminophen reduces the severity of the reaction.

Allergic Reactions. Allergic reactions are relatively frequent, occurring in about 1% of all transfusions. Reactions are usually mild and consist of rash, urticaria, and flushing. In rare instances, anaphylactic shock develops. Allergic reactions are caused by the transfusion of antibodies from hypersensitive donors or the transfusion of antigens to which the recipient is hypersensitive. Allergic reactions can occur after the administration of any blood product but are commonly associated with FFP and platelets. Treatment and prophylaxis consist of the administration of antihistamines. In more serious cases, epinephrine or steroids may be indicated

Respiratory Complications. Respiratory compromise may be associated with transfusion-associated circulatory overload (TACO), which is an avoidable complication. It can occur with rapid infusion of blood, plasma expanders, and crystalloids, particularly in older patients with underlying heart disease. Central venous pressure monitoring should be considered whenever large amounts of fluid are administered. Overload is manifest by a rise in venous pressure, dyspnea, and cough. Rales can generally be heard at the lung bases. Treatment consists of diuresis, slowing the rate of blood administration, and minimizing fluids while blood products are being transfused.

Complications from a single transfusion	*Complications from massive transfusion*

• **incompatibility haemolytic transfusion reaction**
• **febrile transfusion reaction**
• **allergic reaction**
• **infection**
  • **bacterial infection (usually due to faulty storage)**
  • **hepatitis**
  • **HIV**
  • **malaria**
• **air embolism**
• **thrombophlebitis**
• **transfusion-related acute lung injury (usually from FFP).**
	• coagulopathy
	• hypocalcaemia
	• hyperkalaemia
	• hypokalaemia
	• hypothermia.
	• In addition, patients who receive repeated transfusions over long periods of time (e.g. patients with thalassaemia) may develop iron overload. (Each transfused unit of red blood cells containapproximately 250 mg of elemental iron.)

The syndrome of TRALI is defined as noncardiogenic pulmonary edema related to transfusion.89 It can occur with the administration of any plasma-containing blood product. Symptoms are similar to circulatory overload with dyspnea and associated hypoxemia. However, TRALI is characterized as noncardiogenic and is often accompanied by fever, rigors, and bilateral pulmonary infiltrates on chest x-ray. It most commonly occurs within 1 to 2 hours after the onset of transfusion but virtually always before 6 hours. Treatment of TRALI entails discontinuation of any transfusion, notification of the transfusion service, and pulmonary support, which may vary from supplemental oxygen to mechanical ventilation.

Hemolytic Reactions. Hemolytic reactions can be classified as either acute of delayed. Acute hemolytic reactions occur with the administration of ABO-incompatible blood and can be fatal in up to 6% of cases. Contributing factors include errors in the laboratory of a technical or clerical nature or the administration of the wrong blood type. Immediate hemolytic reactions are characterized by intravascular destruction of red blood cells and consequent hemoglobinemia and hemoglobinuria. DIC can be initiated by antibody-antigen complexes activating factor XII and complement, leading to activation of the coagulation cascade. Finally, acute renal insufficiency results from the toxicity associated with free hemoglobin in the plasma, resulting in tubular necrosis and precipitation of hemoglobin within the tubules.

Delayed hemolytic transfusion reactions occur 2 to 10 days after transfusion and are characterized by extravascular hemolysis, mild anemia, and indirect (unconjugated) hyperbilirubinemia. They occur when an individual has a low antibody titer at the time of transfusion, but the titer increases after transfusion as a result of an anamnestic response. Reactions to non-ABO antigens involve immunoglobulin G-mediated clearance by the reticuloendothelial system. If the patient is awake, the most common symptoms of acute transfusion reactions are pain at the site of transfusion, facial flushing, and back and chest pain. Associated symptoms include fever, respiratory distress, hypotension, and tachycardia. In anesthetized patients, diffuse bleeding and hypotension are the hallmarks. A high index of suspicion is needed to make the diagnosis. The laboratory criteria for a transfusion reaction are hemoglobinuria and serologic criteria that show incompatibility of the donor and recipient blood. positive Coombs' test indicates transfused cells coated with patient antibody and is diagnostic. Delayed hemolytic transfusions may also be manifest by fever and recurrent anemia. Jaundice and decreased haptoglobin usually occur, and low-grade hemoglobinemia and hemoglobinuria may be seen. The Coombs' test is usually positive, and the blood bank must identify the antigen to prevent subsequent reactions. If an immediate hemolytic transfusion reaction is suspected, the transfusion should be stopped immediately, and a sample of the recipient's blood drawn and sent along with the suspected unit to the blood bank for comparison with the pretransfusion samples. Urine output should be monitored and adequate hydration maintained to prevent precipitation of hemoglobin within the tubules. Delayed hemolytic transfusion reactions do not usually require specific intervention.

MASSIVE TRANSFUSION

the replacement by transfusion of more than 50 percent of a patient's blood volume in 12 to 24 hours.

The most frequent indication:

1.Hemorrahgic shock secondary to trauma,

2. ruptured aortic aneurysm,

3. massive GI hemorrhage

4.liver transplantation

AUTOLOGOUS BLOOD

It is possible for patients undergoing elective surgery to predonate their own blood up to 3 weeks before surgery for retransfusion during the operation. Similarly, during surgery blood can be collected in a cellsaver which washes and collects red blood cells which can then be returned to the patient.

In emergency situations, type O-negative blood may be transfused to all recipients. O-negative and type-specific red blood cells are equally safe for emergency transfusion.

ACUTE_ISOTONIC_DILUTION

This process dilutes your own blood so you lose less concentrated blood during surgery. This technique has been widely used in cardiothoracic surgery.

BLOOD_SALVAGE

blood is collected and processed for reinfusion

a. peri operative: Blood lost from surgical site may be saved

b. Post-operative: wound drains

COMPARTMENT SYNDROME

INTRODUCTION

The muscle groups of the human limbs are divided into sections, or compartments, formed by strong, unyielding fascial membranes. Compartment syndrome occurs when increased pressure within a compartment compromises the circulation and function of the tissues within that space.

Compartment syndrome may occur acutely, often following trauma, or as a chronic syndrome, seen most often in athletes, that presents as insidious pain. Acute compartment syndrome (ACS) is a surgical.

EPIDEMIOLOGY AND RISK FACTORS

Acute compartment syndrome (ACS) most often develops soon after significant trauma, particularly involving long bone fractures However, ACS may also occur following minor trauma or from nontraumatic causes. Common sites include the leg and forearm]. ACS can also occur in the foot, thigh, and gluteal region

ACS is seen more often in patients under 35 years of age This may be explained by the relatively larger muscle mass of men contained within fascial compartments that do not change in size once growth is complete.

Of note, patients who develop ACS without an associated fracture are at significantly greater risk for delayed diagnosis and treatment (ie, fasciotomy)

Risk associated with fracture treatment — Both closed and open fracture treatment can increase compartment pressure and the risk for ACS.

Trauma without fracture — Other forms of trauma not involving a fracture can predispose a patient to ACS. Possible causes include:

- Forceful direct trauma to a tissue compartment (eg, crush injury)
- Severe thermal burns
- Constrictive bandages, splints, or casts (usually circumferential)
- Penetrating trauma
- High-pressure injection
- Injury to vascular structures
- Animal bites and stings

Vascular, particularly arterial, injury is an important cause of ACS []. Arterial bleeding increases compartment pressures and muscle deprived of arterial blood flow becomes ischemic and prone to reperfusion injury, which in turn causes swelling and a further increase in compartment pressures. In addition, muscle that has sustained a previous ischemic insult is less tolerant of increased tissue pressure . Venous injury (eg, traumatic deep vein harvest, direct vein trauma) is also associated with an increased risk of ACS .

Nontraumatic causes — Nontraumatic causes of ACS occur less frequently but may stem from a wide range of conditions or events.

- Hematologic: ischemia-reperfusion injury, thrombosis, bleeding disorders, vascular disease, spontaneous hemorrhage
- Anticoagulation
- Nephrotic syndrome (or other conditions that decrease serum osmolarity)
- Toxic: animal envenomations and bites; injection of recreational drugs
- IV fluids: extravasation of fluid; massive fluid resuscitation (eg, severe thermal burns, sepsis)
- Prolonged limb compression (eg, following severe drug or alcohol intoxication; poor positioning during surgery)
- Revascularization procedures or treatments (eg, extremity bypass surgery, embolectomy, thrombolysis)
- Group A streptococcus infections of muscle; systemic inflammatory response

Of note, anticoagulation following surgery, such as prophylaxis against deep vein thrombosis, may contribute to ACS]. postoperative pain making the diagnosis difficult.

PATHOPHYSIOLOGY

Multiple explanations for the complex pathophysiology of acute compartment syndrome (ACS) exist. In all cases, the final common pathway is cellular anoxia due to local ischemia. Such ischemia is

thought to begin once local blood flow, restricted by the rise in compartment pressure, fails to meet the metabolic demands of local tissue.

Compartment pressures capable of compromising perfusion develop when they rise to within 10 to 30 mmHg of diastolic pressure; muscle oxygenation decreases as tissue pressure approaches mean arterial pressure []. Therefore, ACS develops based upon both compartment and systemic blood pressures. Compared with a normotensive patient, a patient with hypotension is less likely to tolerate any given increase in tissue pressure.

CLINICAL FEATURES

- **any** tense painful muscle compartment represents a possible ACS. When the diagnosis of ACS is suspected on clinical grounds, it is often confirmed by measuring compartment pressures. As part of the initial assessment, a careful and complete neurologic examination of the extremity should be performed and documented.
- Pain out of proportion to apparent injury (early and common finding)
- Persistent deep ache or burning pain
- Paresthesias (onset within approximately 30 minutes to two hours of ACS; suggests ischemic nerve dysfunction)

amination findings suggestive of ACS include the following:

- Pain with passive stretch of muscles in the affected compartment (early finding)
- Tense compartment with a firm "wood-like" feeling
- Pallor from vascular insufficiency (uncommon)
- Diminished sensation
- Muscle weakness (onset within approximately two to four hours of ACS)
- Paralysis (late finding)
- The classic findings associated with arterial insufficiency are often described as signs of ACS, but this is inaccurate . Of the five classic signs of arterial insufficiency (five P's: pain, pallor, pulselessness, paresthesias, poikilothermia [cold skin temperature]), only pain is commonly associated with compartment syndrome, particularly in its early stages. Paresthesias may also occur.

LABORATORY STUDIES

Acute compartment syndrome (ACS) is diagnosed on the basis of clinical findings and in many cases the measurement of compartment pressures. Laboratory values are not used for diagnosis. If the diagnosis is suspected, surgical consultation, possibly including the measurement of compartment pressures, should not be delayed in order to obtain a laboratory result.

Nevertheless, as ACS develops and muscle breakdown ensues, lab abnormalities develop, including elevations in the serum creatine kinase (CK) (conversely, CK rises in patients with rhabdomyolysis, which may go on to cause ACS). Myoglobinuria can develop within four hours of the onset of
ACS above.)

MEASUREMENT OF COMPARTMENT PRESSURES

Compartment pressures are not required for diagnosis, and the surgeon may opt to take the patient to the operating room on the basis of the history and examination findings alone without obtaining measurements . Conversely, the surgeon may measure compartment pressures in an effort to avoid unnecessary fasciotomies in patients with suggestive findings.

Direct measurement techniques — Although multiple techniques for direct compartment pressure measurement have been described three methods have been used most frequently: a handheld manometer (eg, Stryker device , a simple needle manometer system, and the wick or slit catheter technique.

In summary:

- ACS delta pressure = diastolic blood pressure – measured compartment pressure
- ACS delta pressure <20 to 30 mmHg indicates need for fasciotomy (we use <30 mmHg)

MANAGEMENT

Perhaps the most important aspect of diagnosis is to maintain a high index of suspicion among patients at risk for acute compartment syndrome (ACS) . Frequent serial examinations are important

in such patients. Immediate surgical consultation should be obtained if ACS is suspected.

Immediate management of suspected ACS includes relieving all external pressure on the compartment. Any dressing, splint, cast, or other restrictive covering should be removed. The limb should neither be elevated nor placed in a dependent position. Placing the limb level with the heart helps to avoid reductions in arterial inflow and increases in compartment pressures from dependent swelling, both of which can exacerbate limb ischemia .

Analgesics should be given and supplementary oxygen provided. Hypotension reduces perfusion, exacerbating tissue injury, and should be treated with boluses of intravenous isotonic saline.

Fasciotomy to fully decompress all involved compartments is the definitive treatment for ACS in the great majority of cases . Delays in performing fasciotomy increase morbidity, including the need for amputation

Fasciotomy is occasionally not indicated or may not be necessary. As an example, it should be avoided when the muscle is already dead. Fasciotomy in such instances provides no benefit and can increase the risk of infection. Definitive treatment for such injuries often involves amputation

Hyperbaric oxygen has been described as adjunct treatment for ACS .

ACUTE COMPARTMENT SYNDROME OF THE EXTREMITY – RAPID OVERVIEW.

ACS occurs when the pressure in a muscle compartment rises sufficiently to cause tissue ischemia leading to muscle or nerve damage. Impending ACS occurs when tissue pressure has begun to increase and tissue perfusion is reduced but is not sufficient to cause muscle or nerve damage.

RISK FACTORS

Severe trauma: Long bone fracture, crush injury.

Prolonged extremity ischemia with reperfusion.

Spontaneous bleeding, hematoma.

Burn injury, massive fluid resuscitation, SIRS.

Others:* Myositis, myonecrosis, rhabdomyolysis, prolonged immobilization, bites and stings, high pressure injection, intravenous extravasation injury, soft tissue infection, intra-arterial injection, birth injury.

CLINICAL FEATURES

Physical examination alone has limited sensitivity and specificity for ACS. Serial examinations are important in patients at risk. Clinical features include:

Significant extremity pain is the primary feature; pain can be "out of proportion" to apparent injury.

Pain and other features can progress rapidly over a few hours.

Tense, firm compartment (note: deep posterior compartment of the leg cannot be palpated).

Pain exacerbated by passive stretch of muscle within the compartment.

Compartment-specific neurovascular findings (eg, paresthesias, reduced sensation, muscle weakness, diminished pulses).¶

OTHER CLINICAL FINDINGS THAT SUGGEST IMPENDING ACS INCLUDE:

Excessive or disproportionate increase in extremity girth.

Acidosis or hyperkalemia following reperfusion.

Clinical evidence of rhabdomyolysis (eg, high CK >30,000 units).

INITIAL MEASURES AND REASSESSMENT.

Normalize extremity perfusion (eg, fluid resuscitation, align fractures).

Relieve external pressure on the compartment (eg, bivalve or remove cast, escharotomy for circumferential burns).

MEASURE COMPARTMENT PRESSURES.

Whenever possible, pressure measurements should be obtained by the surgeon who will perform fasciotomy.

Compartment pressures typically measured with handheld manometer.

Needle (eg, 18 gauge) attached to a pressure transducer (eg, arterial line setup) can be used.

FASCIOTOMY

Extremity fasciotomy is the only recognized treatment. Early fasciotomy (ideally within four hours of symptom onset) can save the extremity.

Indications:

High clinical suspicion.

Compartment pressure within 30 mmHg of diastolic pressure.

Contraindications: Established late compartment syndrome is not likely to benefit from fasciotomy. Tissue damage becomes irreversible 4 to 8 hours after compartment pressure has increased. Fasciotomy for established ACS after 6 hours of onset increases the rate of infection and amputation.

TECHNIQUES:

Leg: A four-compartment fasciotomy is recommended using either a one- or two-incision approach.

Forearm: One or two incisions are made on the volar surface and one on the dorsal surface.

Fasciotomies of the buttock, thigh, shoulder, upper arm, or hand are needed less often.

OBSERVATION

Patients in whom clinical suspicion is not high.

Hourly reassessment for clinical features of ACS.

Repeat compartment pressures, as needed.

MINIMALLY INVASIVE SURGERY (MIS)

ADVANTAGES OF MINIMAL ACCESS SURGERY

- ✓ Decrease in wound size
- ✓ Reduction in wound infection, dehiscence, bleeding, herniation and nerve entrapment
- ✓ Decrease in wound pain
- ✓ Improved mobility
- ✓ Decreased wound trauma
- ✓ Decreased heat loss
- ✓ less disfiguring
- ✓ Improved visualization
- ✓ It can offer cost-effectiveness to both health services and employers by shortening operating times , shortening hospital stays, and allowing faster recuperation ,postoperative pain, less trauma

LIMITATIONS OF MINIMAL ACCESS SURGERY

- ✓ Reliance on remote vision and operating
- ✓ Loss of tactile feedback
- ✓ Dependence on hand–eye coordination
- ✓ Difficulty with haemostasis
- ✓ Reliance on new techniques
- ✓ Extraction of large specimens
- ✓ Need experienced surgeon and Special instrument
- ✓ Gas insufflator complication(hemodynamic and metabolic)

- **open procedure**;

- ✓ reducing mobility contributes to an increased incidence of pulmonary atelectasis, chest infection, paralytic ileus and deep venous thrombosis
- ✓ The wound is often the cause of morbidity, including infection, dehiscence, bleeding, herniation and nerve entrapment
- ✓ fluid loss by evaporation

-The core principles of minimal access surgery (independent of procedure or device) can be summarized by the acronym **(I-VITROS)**:

- ✓ **I**nsufflate/create space – to allow surgery to take place in the minimal access setting
- ✓ **V**isualize – the tissues, anatomical landmarks and the environment for the surgery to take place
- ✓ **I**dentify – the specific structures for surgery
- ✓ **T**riangulate – surgical tools (such as port placement) to optimize the efficiency of their action, and ergonomics by minimising overlap and clashing of instruments
- ✓ **R**etract – and manipulate local tissues to improve access and gain entry into the correct tissue planes
- ✓ **O**perate – incise, suture, anastomose, fuse
- ✓ **S**eal/hemostasis.

-MINIMAL ACCESS TECHNIQUES CAN BE CATEGORZSED AS FOLLOWS:

LAPAROSCOPY

A rigid endoscope (laparoscope) is introduced through a port into the peritoneal cavity. This is insufflated with carbon dioxide to produce a pneumoperitoneum. Further ports are inserted to enable instrument access and their use for dissection

-Creating a pneumoperitoneum

There are two methods for creation of a pneumoperitoneum: open and closed.

The closed method involves blind puncture using a Verres needle. Although this method is fast and relatively safe, there is a small but significant potential for intestinal or vascular injury on introduction of the needle or first trocar.

- ✓ Less exposure field
- ✓ More postop. pain
- ✓ Less physiological derangement
- ✓ Can done with standard (non laparoscopic) instruments

The Open method according to hasson or modified hasson approach (efficient and safe compared with closed method)
Hasson approach : a 1 cm vertical or transverse incision is made at the level of the umbilicus. Two small retractors are used to dissect bluntly the subcutaneous fat and expose the midline fascia. Two sutures are inserted each side of the midline incision (into the rectus sheath confluence), followed by the creation of a 1 cm opening in the fascia.

Free penetration into the abdominal cavity is confirmed by the gentle introduction of a finger. Finally, a Hasson trocar (or other blunt-tip trocar) is inserted and anchored with the fascial sutures

- ❖ **The umbilicus usually is selected as the preferred point of access because ,in this location , the abdominal wall is quite thin even in obese patients**
- ❖ **Pneumoperitoneum can be achieved with air,N2O or CO2 insufflation**

-PREOPERATIVE PROBLEMS

- ✓ Previous abdominal surgery (assess the type and location of surgical scars)
- ✓ Obesity (technical difficulties in obtaining pneumoperitoneum, reaching the operative region adequately and achieving adequate exposure in the presence of an obese colon.
- ❖ Recently, the use of optical port entry for laparoscopic bariatric surgery has revolutionized port entry for morbid obesity cases.

-OPERATIVE PROBLEMS

- ✓ Intraoperative perforation of a viscus more common with the laparoscopic than open (as perforation of the gallbladder)
- ✓ The m.c. arrythmia created by laparoscopy is bradycardia(Rapid stretch >> Vagal response)
- ✓ Decreased renal blood flow which reversible after surgery (Intraoperative oliguria is common But IV fluid intraop. should not linked to urine output)
- ❖ **Antibiotics to prevent infections and sepsis**

A single dose of antibiotics should be administered within 1 hour of skin incision; in contaminated, semi-contaminated or complex procedures, additional doses should be administered, based on local microbiological advice.

- ✓ Bleeding

-Risk factors that predispose to increased bleeding include:

●● cirrhosis

●● inflammatory conditions (acute cholecystitis, diverticulitis)

●● patients on clopidogrel and or dipyridamole

●● coagulation defects: (CI to a laparoscopic procedure)

- ❖ Although most bleeding vessels can be controlled laparoscopically, judgement should be used in convert to an open procedure at an early stage
- ✓ Electrosurgical injuries (2 per 1000)

-POSTOPERATIVE CARE

- ✓ In the absence of problems, patients should be fit for discharge within 24 hours (comfortable,

have passed urine and are eating and drinking satisfactorily)

- ✓ If the patient develops a fever or tachycardia, or complains of severe pain at the operation site, routine investigation should include a CBC , (CRP) measurement, LFT, an amylase test and, probably, an ultrasound scan of the upper abdomen to detect fluid collections. If bile duct leakage is suspected, (ERCP) may be needed

- ❖ The most common routine postoperative symptoms are a dull upper abdominal pain, nausea and pain around the shoulders (referred from the diaphragm)

-Analgesia

A 100-mg diclofenac suppository may be given at the time of the operation

-Oral fluids

There is no significant ileus after laparoscopic surgery, except in resectional procedures, such as colectomy or small bowel resection.

Patients can start taking oral fluids as soon as they are conscious; they usually do so 4–6 hours after the end of the operation.

-Oral feeding

Provided that the patient has an appetite, a light meal can be taken 4–6 hours after the operation

-Drains (depends on the operation performed**)**

THORACOSCOPY

A rigid endoscope is introduced through an incision in the chest to gain access to the thoracic contents. Usually there is no requirement for gas insufflation, as the operating space is held open by the rigidity of the thoracic cavity

Unnessesary to use positive pressure>> less complication (decreased venous return, mediastinal shift)

ENDOLUMINAL ENDOSCOPY

Flexible or rigid endoscopes are introduced into hollow organs or systems, such as the urinary tract, upper or lower gastrointestinal tract, and respiratory and vascular systems.

NOTES : natural orifice transluminal endoscopic surgery is an emergency field within minimally invasive surgery and interventional gastroenterology in which the surgeon accesses the peritoneal cavity via a hollow viscus and performs diagnostic and therapeutic procedures .

PERIVISCERAL ENDOSCOPY

Body planes can be accessed even in the absence of a natural cavity. Examples are mediastinoscopy, retroperitoneoscopic(kidney, aorta and lumbar sympathetic chain), as well as hernia repair.

Other, more recent, examples include subfascial ligation of incompetent perforating veins in varicose vein surgery.

ARTHROSCOPY AND INTRA-ARTICULAR JOINT SURGERY

COMBINED APPROACH

The diseased organ is visualized and treated by an assortment of endoluminal and extraluminal endoscopes and other imaging devices.

Examples include the combined laparoendoscopic approach for the management of biliary lithiasis, colonic polyp excision and several urological procedures,

such as pyeloplasty and donor nephrectomy

-PREOPERATIVE EVALUATION

- ✓ Overall fitness: cardiac arrhythmia(from pneumoperitoneum), Severe chronic obstructive airways disease and ischemic heart disease (as emphysema), (cirrhosis)

- ✓ Medications-allergies
- ✓ Particular attention should be paid to the presence or absence of jaundice, abdominal scars, palpable masses or tenderness
- ✓ Previous surgery: scars, adhesions
- ✓ Body habitus: obesity, skeletal deformity(kyphosis)
- ✓ Normal coagulation
- ✓ Thromboprophylaxis

Venous stasis induced by the reverse Trendelenburg position during laparoscopic surgery may be a particular risk factor for deep vein thrombosis, as is a lengthy operation and the obesity of many patients. Subcutaneous low molecular weight heparin and ant thromboembolic stockings should be used routinely, in addition to pneumatic leggings during the operation.

Patients already taking warfarin for other reasons should have this stopped temporarily or converted to intravenous heparin

- ✓ Informed consent

The nature of the procedure, the risks involved and, when appropriate,

the alternatives that are available and when to convert to open procedure

-postoperative: In the early days of laparoscopic surgery, routine bladder catheterization and nasogastric intubation were advised.

NOTES :

-laparoscopic cholecystectomy is now the 'gold standard' for operative treatment of symptomatic gallstone disease

-see-through (optical) ports that allow the surgeon to cut down through the abdomen while observing the layers through the cameras (Especially in bariatric surgery)
- laparoscopic appendicectomy the most common minimally invasive

emergency procedure

ENDOSCOPY

The ability to take targeted mucosal biopsies remains a unique strength of endoscopy

The introduction of both capsule endoscopy and single/double-balloon enteroscopy allows both diagnostic and therapeutic access to the entire gastrointestinal tract.

Endoscopic ultrasound can examine all layers of the intestinal wall as well as extraintestinal structures

-CONSENT

-The risks of endoscopy

- ✓ Sedation-related cardiorespiratory complications
- ✓ Damage to dentition
- ✓ Aspiration
- ✓ Perforation or haemorrhage after endoscopic dilatation/therapeutic endoscopic ultrasound
- ✓ Perforation, infection and aspiration after percutaneous endoscopic gastrostomy insertion
- ✓ Perforation or haemorrhage after flexible sigmoidoscopy/colonoscopy with polypectomy
- ✓ Pancreatitis, cholangitis, perforation or bleeding after ERCP

-SAFE SEDATION

Conscious sedation (not anesthesia)to decrease endoscopy-related mortality

- ❖ Endoscopy in certain situations (particularly paediatric endoscopy) requires a general anesthetic.

Benzodiazepines (reach their maximum effect 15–20 min after administration – The half-life of benzodiazepines is 4–24 hours)

Coadministration of opiates and benzodiazepines has a synergistic effect

The use of supplementary oxygen is essential in all sedated patients

- Patients must be advised not to drink alcohol or drive for 24 hours

-On Diapitic patient:
A poorly controlled insulin-dependent diabetic undergoing colonoscopy will require more input than a type 2 diabetic on oral hypoglycaemic medication undergoing upper gastrointestinal endoscopy

-The majority of endoscopy can be performed safely without the need for routine antibiotic prophylaxis

Patients with a previous history of endocarditis or a metallic heart valve,

endoscopic percutaneous gastrostomy(stoma infection especially on malignancy) – Coamoxiclave /single dose before procedure

endoscopic manipulation of an obstructed biliary tree in which it is unlikely that complete drainage will be achieved or there is significant comorbidity. – Ciprofloxacine

cystic cavities are aspirated at endoscopic ultrasound, - co-amoxiclav

-Urgent endoscopy for gastrointestinal bleeding in the anticoagulated patient (warfarin)

If (INR) is above the therapeutic range or if they are taking concomitant aspirin/ (NSAIDs) ,if complete reversal is not appropriate, correction of the INR to approximately 1.5 is usually sufficient to allow endoscopic diagnosis and therapy

Anticoagulation can often be resumed 24 hours after successful endoscopic therapy

-Elective endoscopy in patients on anticoagulants and antiplatelet agents

Vary in their potential to produce significant or uncontrolled bleeding.

- Aspirin+NSIDS no need to discontinue therapy before endoscopic procedures.

UPPER GASTROINTESTINAL ENDOSCOPY

- ✓ -(OGD) most commonly performed endoscopic procedure
- ✓ To the ligaments of treitz
- ✓ Traditional forward-viewing endoscopes do not adequately visualise the ampulla, and a side-viewing scope is needed
- ✓ clear mucosal Views **+** mucosal biopsies (histopathology) + brushings (cytology)

-INDICATIONS:

when a patient's symptoms are persistent despite appropriate empirical therapy or are associated with warning signs such as intractable vomiting, anaemia, weight loss, dysphagia or bleeding or symptoms of malabsorption and chronic diarrhea**.**

It is also commonly used in the surveillance of neoplasia development in high-risk patient groups.

-THERAPEUTIC OGD :

- ✓ The most common therapeutic endoscopic procedure performed as an emergency is the control of upper gastrointestinal haemorrhage of any aetiology
- ✓ Oesophageal varices :Band ligation
- ✓ Gastric and duodenal varices : sclerotherapy using thrombin-based glues can be used to control blood loss from it.
- ✓ Peptic ulcer with an active arterial spurt or high-risk stigmata of haemorrhage **:**Injection sclerotherapy with adrenaline coupled with a second haemostatic technique such as heater probe vessel obliteration or haemoclip application, remains the technique of choice for such high-risk bleeds
- ✓ Benign oesophageal and pyloric strictures dilatation : under direct vision with 'through-the-scope' (TTS) balloon dilators or the more traditional guidewire-based systems such as Savary–Guillard bougie dilators or fully-covered removable stent, or with a biodegradeable stent.

- ✓ Achalasia can be treated by pneumatic balloon dilatation with a 30–40 mm balloon or injection of botulinum toxin into the lower oesophageal sphincter(limited duration of benefit (3–6 month))
- ✓ Insertion of a percutaneous endoscopic gastrostomy (PEG) tube in patients unable to maintain oral nutritional intake
- ✓ Dysphagia**:** self-expanding metal stents
- ✓ Benign or malignant tracheo-oesophageal fistulae**:**coverd stent
- ✓ Early oesophageal ,Barrett's high-grade dysplasia, and gastric neoplasia: endoscopic mucosal resection (EMR) or endoscopic submucosal dissection (ESD)

-COMPLICATIONS

The majority of adverse events relate to sedation and patient comorbidity

Perforation is more common in therapeutic endoscopy, particularly oesophageal dilatation and EMR/ESD for early malignancy

Prompt management includes radiological assessment using CT/water-soluble contrast studies, strict nil by mouth, intravenous fluids and antibiotics and early review by an experienced upper gastrointestinal surgeon.

ENDOSCOPIC ASSESSMENT OF THE SMALL BOWEL

- ✓ The most frequent indication is the investigation of gastrointestinal blood loss, which may present with either recurrent iron deficiency anaemia (occult haemorrhage) or recurrent overt blood loss per rectum (cryptic haemorrhage) in a patient with normal OGD

(with duodenal biopsies) and colonoscopy

- ✓ Investigation of malabsorption; the exclusion of cryptic small bowel inflammation such as Crohn's disease in patients with diarrhoea/abdominal pain and evidence of an inflammatory response; targeting lesions seen on radiological investigations
- ✓ Surveillance for neoplasia in patients with inherited polyposis syndromes.

- ❖ Barium follow-through**-** do not give true mucosal views
- ❖ MRI and CT enteroclysis, which demonstrate excellent diagnostic accuracy in this area**-** do not allow direct mucosal views, have no

biopsy capability and have limited scope in terms of therapeutics

-Two major clinical advances small bowel diagnosis and therapeutics:

capsule endoscope allows diagnostic mucosal views of the entire small bowel to be obtained with minimal discomfort in unsedated patients.

Second, the novel technique of single/ double-balloon enteroscopy allows endoscopic access to the entire small bowel for biopsy and therapeutics

-Capsule endoscopy

It compares favourably with the 'gold standard' techniques for the localisation

of cryptic and occult gastrointestinal bleeding and the diagnosis of small bowel Crohn's disease

Use of the capsule endoscope is contraindicated in patients with known small bowel strictures>>obstruction Severe gastroparesis and pseudo-obstruction are also relative contraindications so**.** barium follow-through or small bowel MRI done before to exclude structuring

-Single/double-balloon enteroscopy indications:

- ✓ Bleeding from the gastrointestinal tract of obscure cause
- ✓ Iron deficiency anaemia with normal colonoscopy and gastroscopy
- ✓ Visualization of and therapeutic intervention for abnormalities

seen on traditional small bowel imaging/capsule endoscopy as Polypectomy

Comparison of the advantages and disadvantages of the currently available modalities to endoscope the small intestine.		
Technique	Advantages	Disadvantages
Traditional enteroscopy	Simple technique with wide availability Full range of therapeutics available Performed under sedation	Some discomfort Can only access proximal small bowel
Capsule endoscopy **)**	Able to visualise the entire small bowel Preferable for patients No sedation Painless	No biopsies Not controllable and no accurate localisation Variable transit Incomplete studies due to battery life Not suitable for patients with strictures Large capsule to swallow
Double/ single-balloon enteroscopy	Able to visualise the entire small bowel Full range of therapeutics	Requires sedation/ general anaesthesia Patient discomfort May take 3-4 hours; may require admission Specialist centres only Complications include perforation

ERCP:

-This procedure involves the use of a side-viewing duodenoscope

-Theraputic + cytology/biopsy specimens.

-THERAPEUTIC ERCP

All patients require routine blood screening including a clotting screen

Assessment of respiratory and cardiovascular comorbidity is essential

High level of sedation that is often required during the procedure

-The relief of biliary obstruction (M.C indication)

Contrast injection>> which will clearly differentiate the filling defects associated with gallstones and the luminal narrowing of a stricture
if gallbladder stone>>biliary sphincterotomy (pass spontainously within days to weeks after procdure),another option is balloon catheter or by extraction using a wire basket (to reduce the risk of impaction, cholangitis or pancreatitis**)** or removable plastic stent placement (if adequate stone extraction cannot be achieved at the initial ERCP).

On benign biliary strictures diltation , balloon catheter with temporary plastic stent to maintain drainage

On malignant biliary obstruction >>sphincterotomy + Self-expanding metal stents

- ❖ Stent malfunction, associated with recurrent or persistent biochemical cholestasis, may be due to poor initial stent position, stent migration, blockage with blood clot or debris or tumour ingrowth

-for pancreatic disease(pancreatic stone extraction, the dilatation of pancreatic duct strictures and the transgastric drainage of pancreatic pseudocysts) and the assessment of biliary dysmotility (sphincter of Oddi dysfunction) using manometry

-COMPLICATIONS:

- ✓ Duodenal perforation (1.3%) /Haemorrhage (1.4%) after scope insertion or sphincterotomy
- ✓ Pancreatitis (4.3%)

- ✓ Sepsis (3–30%)
- ✓ The mortality rate approaches 1%.

-POSTPROCEDURAL :

CT scanning is essential in patients with pain, tachycardia or hypotension

All patients undergoing ERCP should be administered per-rectal indomethacin or diclofenac immediately before or after the procedure to reduce the risk of post-ERCP pancreatitis

Risk factors for post-ERCP pancreatitis.	
Definite	Suspected SOD
	Young age
	Normal bilirubin
	Prior ERCP-related pancreatitis
	Difficult cannulation
	Pancreatic duct contrast injection
	Pancreatic sphincterotomy
	Balloon dilatation of biliary sphincter
Possible	Female sex
	Low volume of ERCPs performed
	Absent CBD stone
CBD, common bile duct; ERCP. endoscopic retrograde cholangiopancreatography; SOD, sphincter of Oddi dysfunction.	

COLONOSCOPY

-INDICATIONS FOR COLONOSCOPY

- ✓ Rectal bleeding with looser or more frequent stools +/− abdominal pain related to bowel actions
- ✓ Iron deficiency anaemia (after biochemical confirmation +/− negative coeliac serology): esophagogastroduodenoscopy and colonoscopy together
- ✓ Right iliac fossa mass if ultrasound is suggestive of colonic origin
- ✓ Change in bowel habit associated with fever/elevated inflammatory response
- ✓ Chronic diarrhoea (>6 weeks) after sigmoidoscopy/rectal biopsy and negative coeliac serology
- ✓ Follow-up of colorectal cancer and polyps
- ✓ Screening of patients with a family history of colorectal cancer
- ✓ Assessment/removal of a lesion seen on radiological examination
- ✓ Assessment of ulcerative colitis/Crohn's extent and activity
- ✓ Surveillance of inflammatory bowel disease
- ✓ Surveillance of acromegaly/ureterosigmoidostomy

-THERAPUTIC:

- ✓ The most common therapeutic procedure performed at colonoscopy

is the resection of colonic polyps

- ✓ Retrieved specimens can be assessed for risk factors for neoplastic progression and an appropriate surveillance strategy determined
- ✓ In the treatment of symptomatic angioectasias of the colon
- ✓ Debulk colonic tumours not suitable for resection>>Laser photocoagulation
- ✓ The colonoscopic placement of self-expanding metal stents

-COMPLICATIONS OF COLONOSCOPY.. RARE

- ✓ Perforations (especially on excessive air insufflation in severe diverticular disease)
- ✓ Haemorrhage
- ✓ Postpolypectomy syndrome: a transmural burn with associated localised peritonitis
- ❖ Total colonoscopy is contraindicated in the presence of severe Colitis

ENDOSCOPIC ULTRASOUND.

- combines the traditional mucosal image with a separate ultrasound view that clearly depicts the intestinal layers and proximate extraintestinal structures

-COMPLICATIONS :

- ✓ Over-sedation
- ✓ Oesophageal perforation during diagnostic procedures
- ✓ Haemorrhage/perforation during therapeutic procedures.

-INDICATIONS FOR ENDOSCOPIC ULTRASOUND.

- ✓ Diagnostic: Staging of oesophageal/gastric malignancy
- ✓ Staging of hepatobiliary malignancy
- ✓ Diagnosis of choledoccal microlithiasis
- ✓ Therapeutic Biopsy of paraoesophageal lymph nodes
- ✓ Biopsy of submucosal upper GI lesions
- ✓ Biopsy of pancreaticobiliary mass
- ✓ Biopsy of portal lymphadenopathy
- ✓ Biopsy of left adrenal and left liver masses
- ✓ Transgastric drainage of pancreatic pseudocyst
- ✓ Coeliac plexus block

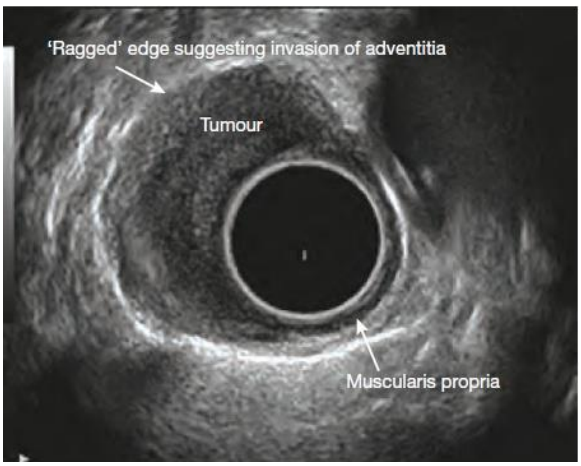


Figure 4 Endoscopic ultrasound image of an esophageal tumour invading into the wall.

IMPROVED ENDOSCOPIC IMAGING (CHROMOENDOSCOPY, NARROW BAND ,IMAGING AND HIGH RESOLUTION MAGNIFICATION ENDOSCOPY)

- ✓ The ability to enhance lesion detection and achieve near-patient discrimination of pathology without the need for histology(give near- histological quality definition to allow near-patient diagnosis)
- ✓ The goal is to allow accurate discrimination of dysplasia grade in areas of Barrett's oesophagus or quiescent ulcerative colitis and to aid polyp detection and the recognition of early gastric and colorectal cancer
- ✓ Chromoendoscopy, which involves the topical application of absorptive stains or pigments(as methylene blue) to improve tissue localization, characterization or diagnosis

Acetic acid >> dysplasia detection in Barrett's esophagus

Lugol's iodine>>early squamous cell carcinoma.

- ✓ Narrow band imaging (NBI) relies on an optical filter technology that radically improves the visibility of capillaries, veins and other subtle tissue structures by optimising the absorbance and scattering characteristics of light.

High-resolution magnifying endoscopy may be used alone or in combination with one of the above techniques to achieve near-cellular definition of the mucosa, and is of particular use in neoplasia surveillance

POST OP PY-REXIA

About 40% of patients develop pyrexia (38.3-39.4) after major surgery however, in most cases no cause is found. the inflammatory response to surgical trauma may manifest itself as fever, and so pyrexia does not necessarily imply sepsis. However, in all patients with a pyrexia, a focus of infection should be sought.

THE CAUSES OF A RAISED TEMPERATURE POSTOPERATIVEL :

- atelectasis of the lung
- superficial and deep wound infection
- chest infection, urinary tract infection and thrombophlebitis
- wound infection, anastomotic leakage, intracavitary collections and abscesses.

THE POSSIBLE CAUSES OF PYREXIA OF A NON-INFECTIVE ORIGIN

- DVT
- transfusion reaction
- wound haematomas
- atelectasis
- drug reactions

ATELECTASIS

commonly occurs in the first 36 hours after operation and typically presents with dyspnea and hypoxia.

Therapy is aimed at reexpanding the collapsed alveoli. For most patients, deep breathing and coughing along with the use of incentive spirometry are adequate.

Postoperative pain should be sufficiently controlled so that pulmonary mechanics are not significantly impaired

In patients with significant atelectasis or lobar collapse, chest physical therapy and nasotracheal suctioning might be used required. In rare cases, bronchoscopy can aid in clearing mucus plugs that cannot be cleared using less invasive measures.

POSTOPERATIVE WOUND INFECTIONS

The majority of wound infections arise from endogenous sources within the patient, but exogenous surgical site infection may also occur from bacteria present in the ward or staff .

gloving before touching patient wounds and hand washing between all patient contacts are important preventive so measures.

Now that patients are discharged more quickly after surgery and many procedures are performed as day cases, many SSIs are missed by the surgical team unless they undertake a prolonged and carefully audited follow-up with primary care doctors.

Suppurative wound infections take **7–10** days to develop, and even cellulitis around wounds caused by invasive organisms (such as b-haemolytic Streptococcus) takes 3–4 days to develop.

Major surgical infections with systemic signs. evidence of spreading infection, cellulitis or bacteraemia need treatment with appropriate antibiotics. The choice may need to be empirical initially but is best based on culture and sensitivities of isolates harvested at surgery or from culture of wound fluids or wound swabs. Although the identification of organisms in surgical infections is necessary for audit and wound surveillance purposes, it is usually 2–3 days before sensitivities are known It is illogical to withhold antibiotics until results are available but, if clinical response is poor by the time sensitivities are known, then antibiotics can be changed Such changes are unusual if the empirical choice of antibiotics is sensible; change of antibiotics promotes resistance and risks complications, such as C. difficile enteritis , evidence of suppuration ,If an infected wound is under tension, or there is clear sutures or clips need to be removed with curettage if necessary, to allow pus to drain adequately

In severely contaminated Delayed primary or secondary closure can be undertaken when the wound is clean and granulating.

Some heavily infected wounds may be left to heal by secondary intention, with no attempt at closure, particularly where there is a loss of skin cover and healthy granulation tissue develops while the end result may be excessive scarring that can always be revised with plastic surgery under clean surgical conditions at a later stage.

If bacteraemia is suspected, but results are negative then repeat specimens for blood culture may need to be taken. A rapid report on infective material can be based on an immediate Gram stain.

Topical antiseptics should only be used on heavily contaminated wounds for a short period to clear infection as they inhibit epithelial ingrowth and so impair wound healing .

ADVANCES IN THE CONTROL OF INFECTION IN SURGERY

●●Aseptic operating theatre techniques have enhanced the use of antiseptics

●●Antibiotics have reduced postoperative infection rates after elective and emergency surgery

●●Delayed primary, or secondary, closure remains useful in heavily contaminated wounds

*Wound infection is diagnosed by local erythema swelling, pain, tenderness and wound drainage.

Fever and leukocytosis are usually present but may be absent in superficial wound infections.

The primary treatment is to open the wound to allow drainage. The wound should be cultured. If the infection is contained in the superficial tissue layers, antibiotics are not required. In the case of a clean procedure that did not enter the bowel, the usual pathogens are: staphylococcal and streptococcal species. If surrounding erythema is extensive parenteral antibiotics should be initiated. Wound infections in the perineum or after bowel surgery are more likely to be caused by enteric pathogens and anaerobes. More aggressive infections with involvement of underlying fascia require emergent operative débridement and broad-spectrum IV antibiotics

INTRAABDOMINAL ABSCESS OR PERITONITIS

abdominal pain, and tenderness. If the patient has generalized peritonitis, present with fever, leukocytosis emergency laparotomy is indicated.

If the inflammation appears to be localized, a CT scan of the patient's abdomen and pelvis should be obtained. The primary management of an intraabdominal abscess is drainage. In many circumstances this can be performed percutaneously with radiologic guidance. In other situations, operative débridement and drainage are required. Empiric antibiotic therapy should cover enteric pathogens and anaerobes.

PNEUMONIA

can cause shortness of breath and is accompanied by fever, elevated white blood cell count, and productive cough. Chest radiograph provides diagnosis and sputum culture can guide antibiotic therapy

Pneumonias that occur in postoperative patients should be treated as nosocomial infections. Patients requiring mechanical ventilation for longer than 48 hours are at risk for ventilator-associated pneumonia (VAP), which may require bronchoscopy for diagnosis

GASTROINTESTINAL INFECTIONS

may present with fever, leukocytosis, and diarrhea Clostridium difficile is a common cause of diarrhea in hospitalized patients, and there should be a low threshold for performing an assay for the C. difficile organism or toxin. Initial therapy includes fluid resuscitation and metronidazole (500 mg orally or IV every 6 to 8 hours) or vancomycin (250 to 500 mg orally every 6 hours)

PROSTHETIC-DEVICE–RELATED INFECTIONS

may present with fever, leukocytosis and systemic bacteremia. Infection of prosthetic valves may present with a new murmur. Management may require removal of the infected device and the use of long-term antibiotics.

CATHETER-RELATED INFECTIONS

also are diagnosed by the presence of feverbleukocytosis, and systemic bacteremia. Local erythema and purulence may be present around central venous catheter insertion sites. Erythema, purulence, a tender thrombosed vein, or lymphangitis may be present near an infected peripheral IV line. Management includes removal of the catheter and IV antibiotic coverage

URINARY INFECTION

Urinary infection is one of the most commonly acquired infections in the postoperative period. Patients may present with dysuria and/or pyrexia. Immunocompromised patients, diabetics and those patients with a history of urinary retention are known to be at higher risk. Most common organisms are: E-coli ,klebsiella , staph .aureus

most important risk factor is duration of catheterization. Treatment involves adequate hydration, proper bladder drainage and antibiotics depending on the sensitivity of the microorganisms Fever.

SUPERFICIAL THROMBOPHLEBITIS

Common causes include external trauma especially to varicose veins, venipunctures and infusions of hyperosmolar solutions and drugs. The presence of an intravenous cannula for longer than 24–48 hours often leads to local thrombosis.

The surface vein feels solid and is tender on palpation, The overlying skin may be attached to the vein and in the early stages may be erythematous before gradually turning brown. A linear segment of vein of variable length can be easily palpated once the inflammation has died down.

A full blood count, coagulation screen and duplex scan of the deep veins should usually be obtained.

Most patients are treated with non-steroidal anti-inflammatory drugs and topical heparinoid preparations and the condition resolves spontaneously. Rarely, infected thrombi require incision or excision.

DEEP VEIN THROMBOSIS

Deep vein thrombosis (DVT) is a well-known and, when complicated by pulmonary embolus, potentially fatal complication of surgery.

Methods of prevention are guided by the risk score and include the use of compression stockings, calf pumps and pharmacological agents, such as low molecular weight heparin.

The symptoms and signs of DVT include calf pain, swelling warmth, redness and engorged veins. However, most will show no physical signs. On palpation the muscle may be tender and there may be a positive Homans' sign (calf pain on dorsiflexion of the foot), but this test is neither sensitive nor specific.

Duplex Doppler ultrasound and venography can be used to assess flow and the presence of a thrombosis. Other investigations include D-dimer.

If a significant DVT is found (one that extends above the knee), treatment with parenteral anticoagulation initially, followed by longer-term warfarin or new oral anticoagulant.

a caval filter may be required to decrease the possibility of pulmonary embolism..

TRANSFUSION REACTIONS.

Febrile transfusion reactions are non-haemolytic and are usually caused by a graft-versus-host response from leukocytes

in transfused components. Such reactions are associated with fever, chills or rigors. The blood transfusion should be stopped immediately. This form of transfusion reaction is rare with leukodepleted blood.

IMPORTANT NOTES

1-causes of post op pyrexia are **5Ws**

wind	**1-2 days**	**Atelectasis , pneumonia**
water	3-5	**UTI**
walking	4-6	**DVT ,PE ,thrombophlebi-tis**
wound	7-10	**SSI and abscess**
Wounder	Any time	**Drug and blood transfu-sion**

2-immidiate pyrexia or within hours after surgery can be caused by **malignant hyperthermia;** de-velop shortly after the onset of anesthetics typically attributed to halothane or succinylcholine. Temper-ature higher than 40, metabolic acidosis, hyper-calcemia, hyperkalemia may occur. a family hx may exist.

Treatment is IV dantroline .100% oxygen ,correc-tion of acidosis and cooling .

RECONSTRUCTION OF THE SKIN BY SOFT TISSUE

SKIN ANATOMY

Skin is thickest on the palms and soles of the feet, while the thinnest skin is found on the eyelids and in the post-auricular region. Skin consists of epidermis and dermis. The epidermis is a layer of keratinized, stratified squamous epithelium (Figure 1) that sends three appendages (hair follicles, sweat glands and sebaceous glands) into the underlying dermis. In superficial skin injuries, appendages are a source of new ceils for reconstitution of the epidermis. The basal germinal layer of the epidermis generates keratin- producing cells (keratinocytes), which become increasingly keratinized and flattened as they migrate to the surface, where they are shed. The basal layer also contains pigment cells (melanocytes) that produce melanin, which is passed to the keratinocytes and protects the basal layer from ultraviolet light.

The dermis is composed of collagen, elastic fibers and fat. It supports blood vessels, lymphatics, nerves and the epidermal appendages. The junction between the epidermis and the dermis is undulating where dermal papillae push up towards the epidermis.

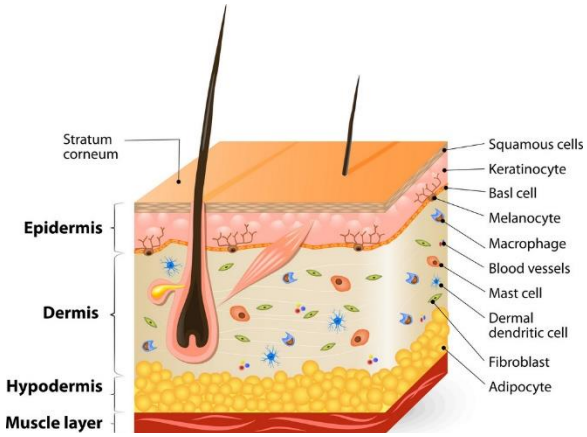


Figure 5 structure of the skin

The three types of epidermal appendage extend into the dermis and, in some places, into the subcutaneous tissues. Hair follicles produce hair, the color of which is determined by melanocytes within the follicle. The sebaceous glands secrete sebum into the hair follicles, which lubricates the skin and hair. The sweat glands are coiled tubular glands lying within the dermis and are of two types; eccrine sweat glands secrete salt and water on to the entire skin surface, while apocrine glands secrete a musty-smelling fluid in the axilla, eyelids, ears, nipple and areola, genital areas and the perianal region.

RECONSTRUCTION OF THE SKIN

if skin has been lost as a direct result of trauma, following the excision of a tumor or necrotic tissue or due to an ulcer, direct suture may not be possible. When the area of skin involved is important, it is often better to speed healing by importing skin to dose the wound. This may be achieved by means of a skin graft, which requires a vascular bed as it has no blood supply of its own, or a flap.

SKIN GRAFTS

A graft refers to total detachment of tissue from one part of the body and transferal to another part, where it must establish its own blood supply. Skin grafts are part of skin that are imported into sites of skin loss with establishment of blood supply. These may be Split-thickness or full-thickness.

SPLIT-THICKNESS SKIN GRAFT

Split-thickness skin graft (STSGs, partial-thickness or Tiersch graft) are cut with a special guarded freehand knife or an electric dermatome (figure 2). Split-thickness skin grafts are harvested by taking all of the epidermis together with some dermis, leaving some skin appendages in the remaining dermis.

The donor site heals by re-epithelialization from epithelial appendages in the remaining dermis (the bases of hair follicles and sweat ducts) within 2-3 weeks, depending on the thickness of the graft (The thicker the graft, the slower the healing). Common donor sites include the thigh and buttocks (in children).

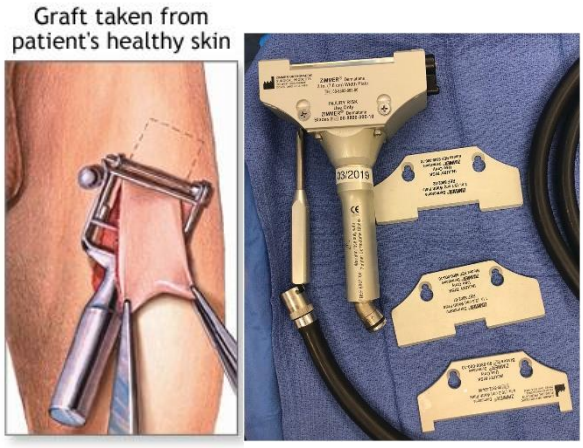


Figure 7 electric dermatome

STSGs are categorized further as thin (0.005-0.012 in), intermediate (0.012-0.018 in), or thick (0.018-0.030 in), based on the thickness of the harvested graft (figure 2). The thinner the graft, the more easily it will take on a bed of imperfect vascularity but the poorer the quality of skin will be (the less the thickness, the less the elements of regeneration, the less it is close to normal) and the more it will shrink. Their thinness, abnormal pigmentation, and frequent lack of smooth texture and hair growth make STSGs more functional than cosmetic.

Because of their thinness, they are fragile and more prone to trauma or disruption.

To cover very large areas, the graft can be expanded by 'meshing'. This is carried out on a device that cuts a series of slits along the skin, allowing it to expand from a ratio of anything from 1:1.5 to about l:6.This also allow exudates to escape and improve take of the graft (see below). STSGs are thus used to cover wounds after acute trauma, granulating areas and burns, or when the defect is large.

FULL-THICKNESS SKIN GRAFT

A full-thickness skin graft (FTSGs) removes the entire thickness of the dermis and leaves a donor defect (which needs to be sutured or grafted) as large as the one to be filled and requires a well-vascularized bed to survive. Features of FTSGs are as follows;

- FTSGs are strong and thus are useful for small defects where strength is needed (e.g. on the palm of the hand).
- In contrast to STSGs, FTSGs do not shrink.
- FTSGs look better than a STSGs as they do not change when they are moved from the donor to the recipient site. Because of these features they are commonly used in reconstructive surgery where a good functional result is important (e.g. on the lower eyelid, Angle of the mouth) or color and texture match (cosmetic results) are important (e.g. the face grafted from behind the ear).
- They tolerate pressure more and thus are perfect for skin loss involving the soles.

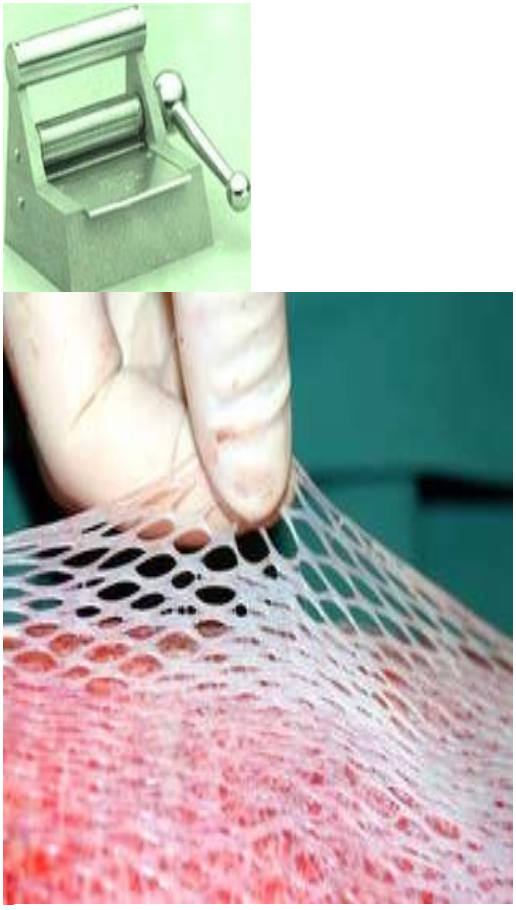


Figure 6 meshing of a STSG

Full thickness grafts should not be used over granulating defects and they are size limited; that is they cannot be applied to large defects. Donor sites for FTSGs are shown in the table below.

Sites of FTSGs donation	
Site of donation	Feature
Postauricular area	limited size
Upper eyelid	Giving to the other eyelid, in old people Because the eyelid is redundant.
Supraclavicular area	Undesirable in Female because of scar. Any area above the clavicle liable to be grafted
Flexural skin	e.g. Antecubital fossa. It is used for joints surfaces as cause no limitation in joint
Groin	Has the disadvantage of being hairy with different color. It is extremely redundant.

TAKE OF THE GRAFT

The process of the attachment of the graft to the recipient site (including the vascularization of the graft) is termed the take of the graft. The following are the steps involved in this process;

- **Adhesion**; In the initial 24 hours, a fibrin bond forms adhering the graft to its bed.
- **Plasmic imbibitions**; the graft swells as interstitial fluid permeates into it from the recipient bed.
- **Inosculation**; ingrowth of vascular tissue starts to occur around day 3 and in thinner grafts revascularization is complete between days 5 and 7. Revascularization starts with capillary beds and then continues with larger arterioles and arteries. Lymphatic circulation is restored after 1 week. Reinnervation occurs over many months.

According to this process, a graft is checked after 7 days. Its success depends on the recipient site which should be capable of producing capillary buds, Approximation of the graft and recipient site (it must be equal in size) and immobilization during the phase of vascularization.

PREPARING THE WOUND FOR GRAFTING

The initial assessment of wounds involves adequate removal of devitalised tissue, assessment of which vital structures will need reconstruction immediately and which might be better reconstructed later, and assessment of the degree of contamination involved, which will require further cleaning.

For skin grafting of the wound, the recipient site should have the following conditions;

- Granulation tissue; Granulation tissue at the base of recipient site should be healthy ; a Healthy granulating tissue is red, flat, vascular, does not bleed easily while an Unsatisfactory granulating tissue is more fibrous and less vascular, bleed easily.

- Type of tissue; Skin grafts will not survive on tissue with limited blood supply (cartilage, tendon, nerve). Skin grafts will survive on periosteum, perichondrium, peritenon, perineurium, dermis, fascia, muscle, and granulation tissue.

GRAFT FAILURE

Graft failure is seen often due to;

- Infection; Regarding STSGs, the group A (ß- haemolytic Streptococcus can destroy split grafts completely (and also convert a donor site to a full-thickness defect) and so the presence of this micro-organism is a contraindication to grafting. Pseudomonas produce so much pus that the graft floats off.

- Bleeding; because blood can separate the donor graft from the recipient site.

- Improper mobilization decreases the chance for fibrin to adhere and capillaries to communicate.

- non-viable grafts; The use of non-viable grafts is also an important and usually a missed cause of graft failure.

SKIN FLAPS

A flap refers to tissue transferred to an adjacent or distal site while retaining a functional vascular

attachment. Whereas skin grafts require a vascular bed to survive, flaps bring their own blood supply to the new site. They can therefore be thicker and stronger than grafts and can be applied to avascular areas such as exposed bone, tendon or joints. In general, they are indicated when skin grafts fail, a skin flap consists of a volume of skin and subcutaneous tissue, while a muscle flap consists of muscle. A myocutaneous flap is composite tissue consisting of skin, subcutaneous tissue, and muscle.

SURGICAL ANATOMY

Direct cutaneous arteries emerge from deeper vessels. Many of these arise in the axilla and groin region (e.g. superficial thoracic artery, superficial epigastric artery) but also at other sites, such as the anterior chest wall. Musculocutaneous perforating arteries from surface of muscles. Generally, these muscles are broad and flat ones that largely exist on the trunk. Fasciocutaneous perforating arteries pass along an intermuscular or intercompartmental fascia septum. Generally, these vessels are found on the limbs (figure 4).

The vessels ascend and anastomose at all levels to form horizontal plexuses that run in the skin subcutaneous tissue and fascia. The three-dimensional area of tissue supplied by a single vessel and its venae commitantes is known as an angiosome (figure 4).

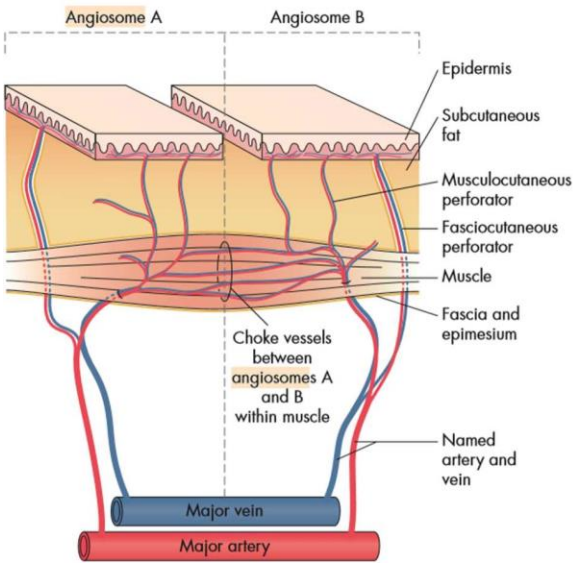


Figure 8 an angiosome

CLASSIFICATION OF FLAPS

LOCAL VS DISTANT FLAPS

The simplest flaps use local skin and fat (local flaps), and are often a good alternative to grafting for small defects such as those left after the excision of facial tumors. If not enough local tissue is available, a flap may have to be brought from a distance (distant flap) and remain attached temporarily to its original blood supply until it has picked up a new one locally. This usually takes 2-3 weeks, after which the pedicle can be divided.

RANDOM FLAPS

These local flaps have no specifically identified vessels and rely on vessels in the subdermal and dermal plexuses (which are smaller and less defined than those of an axial pattern flap (see below)). Because of the random nature of their pedicles, the distal circulation of these skin flaps is unreliable and they can only be safely raised using a length to width ratio of 2 :1 (figure 6-A). These flaps are commonly used for small defects and are categorized according to their direction of movement (advancement, rotation, transposition) (Figure 7). The idea is recruiting tissue from a region of relative excess to a defect, So that the tension (from the tissue lost) can be distributed over larger surface areas (figure 7). If the tension is too great, a back cut, known as a Burow's triangle, can be made to facilitate distal access.

A Z-plasty (figure 5) is an example of a transposition flap and is used to refashion a scar.

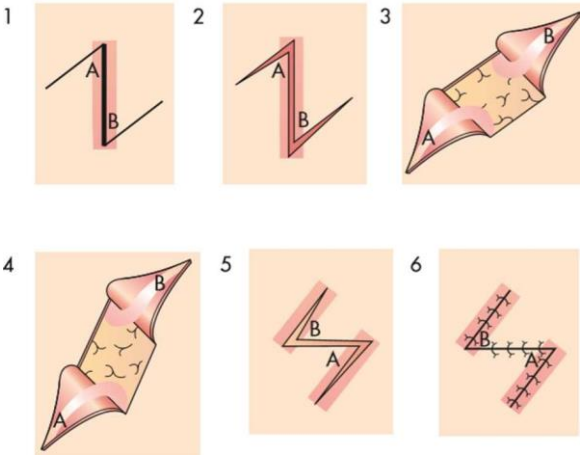


Figure 9 Z-plasty

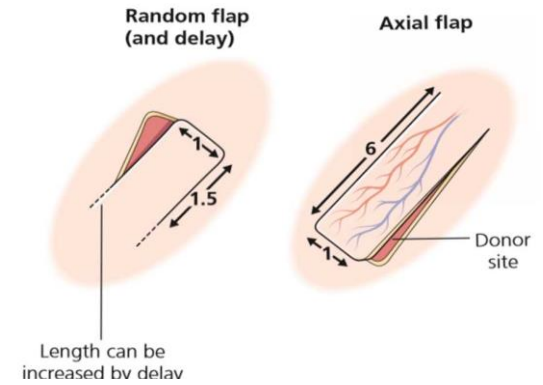


Figure 10 A-random flap B- axial flap

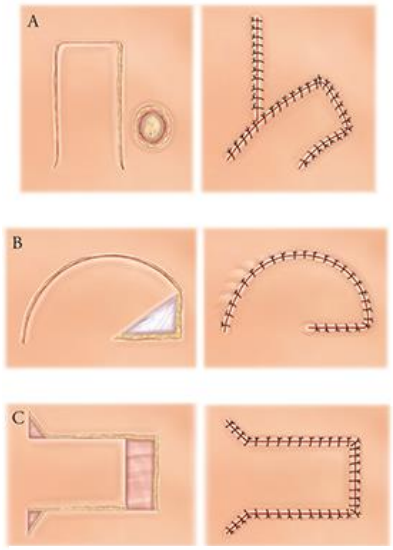


Figure 11 types of flaps

AXIAL PATTERN FLAP

These flaps have a defined single vascular pedicle running longitudinally within them. This axial arrangement allows a flap to be raised with a length to breadth ratio greater than 2:1 (Fig. 6-B). Examples of this type of flap include the groin flap based on the superficial circumflex artery, the deltopectoral flap based on the internal mammary branches, and the median forehead flap based on the supratrochlear artery.

FLAP NECROSIS

The flap has both arterial and venous circulation. To lose flap because of pure arterial insufficiency is rare. Simply, because no one does a flap unless he is sure of the artery. To lose flap due to venous insufficiency is, however common, factors that adversely affect the circulation include mechanical tension, kinking, edema and inflammation.

Flap necrosis is the result of circulatory insufficiency. Signs of flap necrosis include a skin that is acutely congested, cyanosed, with decreased circulation. The development of blisters indicates inevitable necrosis.

APPLICATION OF SKIN RECONSTRUCTION

Skin injury is seen commonly in the context of traumas to skeletal structures, such as bones, joints and cartilages. As a rule; Presence of intact cover of skin prevents infection of bone. Exposed tendons and nerves should be covered by skin to prevent dryness and necrosis, the modality used (graft vs flap) depends on the exposed structure. Periosteum, perichondrium, peritenon, perineurium, dermis, fascia, muscle, and granulation tissue are all favourable for the use of split-thickness skin grafts, when these are injured, it's better to use skin flaps.

BURNS AND INHALATIONAL INJURY

Burns (thermal injury) are a common surgical condition. Over 2 million injuries due to burns require medical attention each year in the United States, with 14,000 deaths resulting.

AETIOLOGY

Most burns follow accidents in the home and could be prevented. In all age groups, Fires in the home are responsible for only 5% of burn injuries but for 50% of burn deaths—most due to inhalational injury with injury to the lungs (this is why it is important to consider this in the management of these patients).

Toddlers (< 3 years) are particularly liable to scalding by hot liquids in kitchen accidents. After 3 years, the incidence of scald injuries starts to decrease, being replaced by injuries due clothes catching fire, contact with hot object, electrocution and chemicals. Teenagers are often injured as a result of illicit activities involving accelerants or electrocution.

The most common bums in adults are suffered by males aged between 17 and 65 years of age. These are mainly due to domestic flame bums but also occur as a result of industrial accidents. The elderly are at risk from scalds.

MORTALITY

Incidence of burns in a specific country can be guessed only, however, mortality is accurately known. Because of enhanced management planes and specialization of centres for burns (burns centre and department), the mortality has been notably declining.

The mortality of burns depends on the site, extent and depth of the burn and on the age and general condition of the patient. The commonest direct causes of death are uncontrolled shock and uncontrolled septicaemia.

PATHOPHYSIOLOGY

LOCAL EFFECTS OF BURNS

There are three zones of a burn area described; At the point of maximum damage there is a zone of coagulation. In this zone there is irreversible tissue loss due to coagulation of the constituent proteins.

This is surrounded by a zone of stasis, characterized by decreased tissue perfusion. The tissue in this zone is potentially salvageable. Additional insults, such as prolonged hypotension, infection or oedema, can convert this zone into an area of complete tissue loss. Finally, there is a zone of hypermedia, where tissue perfusion is increased; this zone will invariably recover.

The local effects result from destruction of the more superficial tissues and the inflammatory response of the deeper tissues. Fluid is lost from the surface or trapped in blisters, the magnitude of loss depending on the extent of injury. With deeper injuries, the epidermis and dermis are converted into a coagulum of dead tissue known as eschar. In its least severe form, the dermal inflammatory response consists of capillary dilatation, as in the erythema of sunburn. With deeper burns, the damaged capillaries become permeable to protein, and an exudate forms with an electrolytic and protein content only slightly less than that of plasma. Lymphatic drainage fails to keep pace with the rate of exudation and interstitial oedema leads to a reduction in circulating fluid volume.

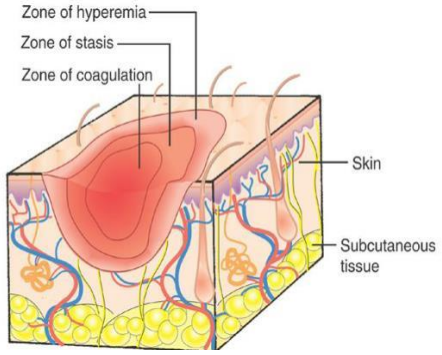


Figure 12 pathophysiology of burns

SYSTEMIC EFFECTS OF BURNS

The systemic effects of a burn depend upon its size. Generally, they are associated with extensive burns. The systemic effects of burn are shown in the table below. The most common causes of mortality are uncontrolled shock (early) and sepsis (late).

Systemic effects of severe burns
• **Hypovolemia**
• **Sepsis**
• **Hyponatremia followed by risk of hypernatremia**
• **Hyperkalaemia followed by hypokalaemia**
• **Haemolysis (anemia)**
• **Hypothermia**
• **Renal failure (acute tubular necrosis due to hypovolemia, haemoglobinuria and myoglobinuria)**
• **Respiratory failure (smoke inhalation, airway obstruction, acute respiratory distress syndrome)**
• **Catabolism and nutritional depletion**
• **Venous thrombosis**
• **Curling's ulcer and erosive gastritis**

CLASSIFICATIO

Burn depth is proportional to the temperature of the causal agent and to the length of contact time. Burns are classified according to depth as either partial- or full-thickness (figure 2). Partial thickness burns are classified according to the depth into superficial and deep. Superficial partial-thickness burns involve only the epidermis and the superficial dermis. In a partial-thickness burn, epithelial cells survive to restore the epidermis; deep partial-thickness (also known as deep-dermal) burns, the epidermis and much of the dermis are destroyed. Restoration of the epidermis then depends on there being intact epithelial cells within the remaining appendages. Full-thickness bums destroy all of the epithelial elements.

In the past, Burns were classified according to increasing depth as epidermal (first-degree), superficial and deep partial-thickness (second-degree), full-thickness (third-degree), and fourth-degree. Further degrees depend on involvement of deep tissues such as muscle and bone.

The table below presents the methods of assessing the depth. The use of laser Doppler can diagnose the different burn depths according to the circulation in the damaged skin; this will be mapped by colours.

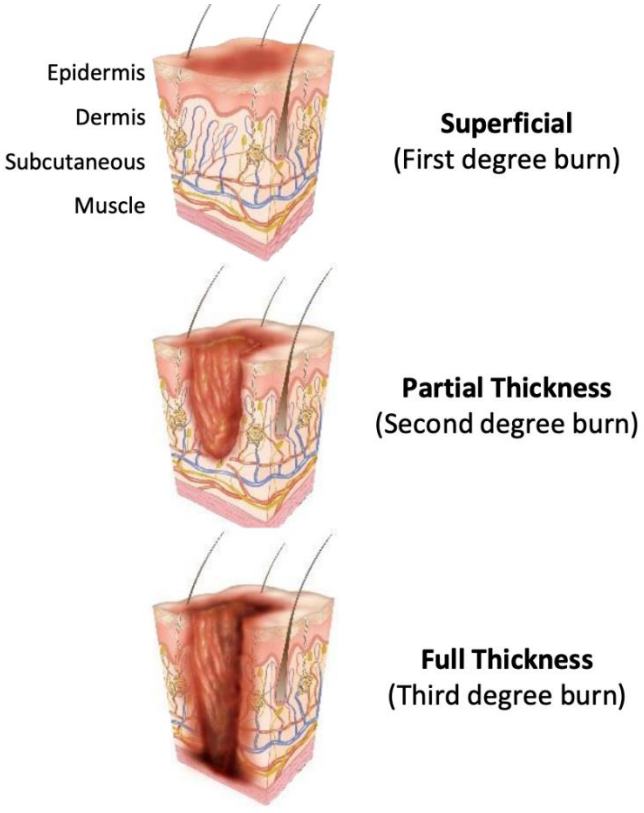


Figure 13 depth of burn injury

Assessment of burn depth			
	Superficial partial	Deep partial	Full thick-ness
Appear-ance	-Pink in colour -Er-ythema -moist -Blisters	-Pink to brown in colour -Ery-thema -moist -Blisters	-Brown (m mahogany-colored) or whit col-oured. -Dry -visible thrombosed veins - Subcutane-ous- pus fat visible in the base
Sensation (pin prick test)	**Positive sensation**	**Posi-tive sensa-tion**	**negative sensation**
Pain	**Very Painful**	**Painful**	**May be pain less**
Blanching test	**positive**	**Posi-tive**	**Negative**
mecha-nism	-Sunburns -Scalds (below boiling point)	-Scalds (above boiling point) -chemi-cals -fires	Scalds (high temperature) -fires -chemi-cals -electro-cution

EXTENT OF BURN

The extent of burn is an important prognostic determinant and affects the management of patients with burns dramatically. It can be calculated according to the affected body surface area (BSA). The approximate extent of the burn can be quickly calculated in adults by using the 'rule of nines'. In this rule, the body is divided into 9%'s, of total body surface area; eleven nines; 2 at the upper limb (4.5 at each side), 1 at the head, 4 at the torso and 4 at the lower limb, (figure 3). The patient's hand and fingers together constitute about 1% of body surface area. The same goes for the genital area.

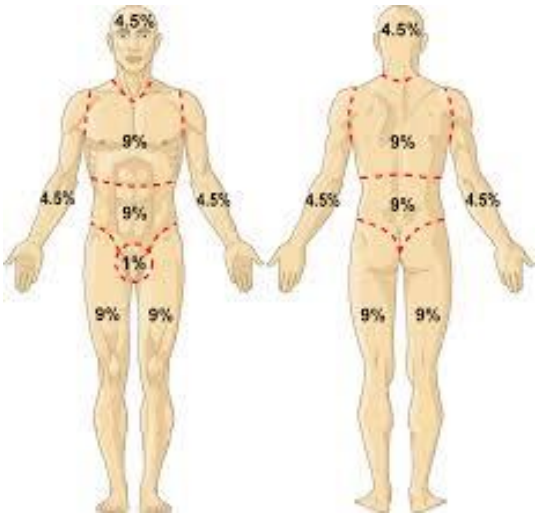
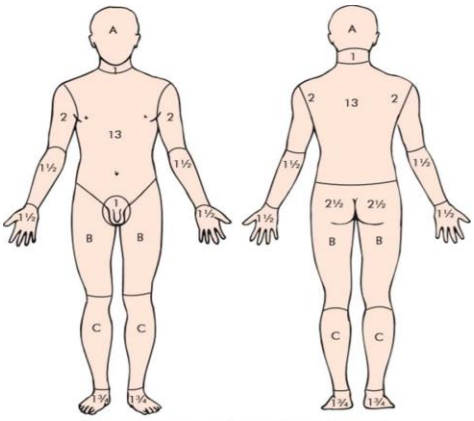


Figure 3 Rule of 9 in adults

In children, because the head constitutes a larger body surface area, it represents two 9%'s instead of one. This is associated with three 9%, divided equally, on the lower limb. The palm method is also useful as a mean of estimation of burns extent in children and small sized burns. It is principled on the fact that a patient palm represents 1% of his/her body surface area among all age groups.

Tables are available for more accurate estimations of burn area. The estimation of burn extent is most accurately determined by using the age-related charts designed by Lund and Browder (lund and browder's charts). These are used in specialized centres and are beyond the scope of this lecture (figure 4).



Relative percentage of area affected by growth

Age in years	0	1	5	10	15	Adult
A Head	9	8	6	5	4	3
B Thigh	2	3	4	4	4	4
C leg	2	2	3	3	3	3

Figure 4 lund and browder's charts

INHALATIONAL INJURY

Inhalation injury (IJ) is a spectrum of respiratory tracts and systemic insults that are caused by the products of combustion and pyrolysis in closed place fires. Inhalational injury may be responsible for up to 50% of burn related deaths. Carbon monoxide (CO) poisoning, thermal injury and smoke inhalation are three distinct aspects of clinical inhalation injury.

partial Carbon monoxide poisoning; The majority of house fire deaths can be attributed to CO poisoning. The affinity of CO for haemoglobin (Hb) approximately 200 times that of oxygen. When inhaled and absorbed, CO binds to Hb to form carboxyhaemoglobin (COHb; The half-life (t1/2) of COHb when breathing room air is approximately 4 hours.). COHb interferes with oxygen delivery to the tissues by preventing reversible displacement of oxygen on the Hb molecule, thus shifting the oxygen-Hb dissociation curve to the left, thereby decreasing oxygen unloading from normal haemoglobin at the tissue level. CO binds to reduced cytochrome a3, resulting in less effective intracellular respiration. Hydrogen cyanide, a common product of the combustion of polyurethane and nitrogen-containing polymers, is an even more effective inhibitor of cellular respiration than CO, and also interferes with oxygen utilization at the tissue level. CO can also bind to cardiac and skeletal muscle, resulting in direct toxicity. CO can act in the central nervous system in a poorly understood fashion, causing demyelination and associated neurologic symptoms.

- Thermal airway injury is caused by the heat carrying capacity of smoke and is most commonly in the upper respiratory tract. True thermal damage to the lower respiratory tract and pulmonary parenchyma is extremely rare, unless live steam or exploding gases.

- Smoke inhalation; Smoke inhalation is the injury that results from the chemical properties of the products of combustion and pyrolysis. Prominent by-products of incomplete combustion are oxides and hydrogenated moieties of sulphur and nitrogen, as well as numerous aldehydes. Epithelial injury can occur at all levels of the respiratory tract, from oropharynx to alveolus.

Patients with inhalational injury may present with symptoms of respiratory distress superimposed on a history of being burned with a fire in a closed place. The table below present clues that raises the suspicion to certainty of inhalational injury.

Clues to the diagnosis of inhalational injury
• **Patient burnt in a closed place**
• **Full-thickness/deep dermal burns to the face neck or upper torso**
• **Singed nasal hair**
• **Carbonaceous sputum**
• **The presence of soot particles on the nose, face, around the mouth and in the oropharynx.**

MANAGEMENT OF BURNS

The initial management of a severely burnt patient is similar to that of any trauma patient. A modified ATLS primary survey is performed, with particular emphasis being placed on assessment of the airway and breathing.

PREHOSPITAL MANAGEMENT

Some measures should be taken immediately;

- extinguishing fire and stopping the bum process (e.g. by rolling on the ground or removing clothes or even dropping in water pools) can change the management dramatically.
- Extensive fires require an immediate supine position so that burns do not reach the face.
- Patients undergoing electrocution should be removed from the power source immediately and cautiously by non-conducting object. Shutting off the electric current is the priority.
- Chemical burns require copious irrigation.
- Cool water is an excellent analgesic and dissipates heat, but common sense must be applied; immersing a child in cold water or covering a patient with cold soaks can cause hypothermia.
- Oxygen is given on the scene when available.

A FOR AIRWAY

An assessment must be made as to whether the airway is compromised or is at risk of compromise. The cervical spine should be protected unless it is definitely not injured. Inhalation of hot gases will result in a burn above the vocal cords; this burn will become oedematous over the course of hours, especially after fluid resuscitation has begun. Consequently, an airway that is patent on arrival may occlude after admission.

Direct inspection of the oropharynx should be performed by a senior anaesthetist. If there is any concern about the patency of the airway, then intubation is the safest policy. Indications for intubation are shown in the table below.

Indications for intubation in burned patients
• **Erythema/swelling of the oropharynx on direct visualization**
• **Change in voice, with hoarseness/harsh cough**
• **Stridor**
• **Dyspnoea**

Tracheostomy is never undertaken lightly in view of the danger of infection of burned tissues around the stoma. However, unfortunately, in many circumstances it is unavoidable.

B FOR BREATHING

Patients burned in an enclosed space or having any signs or symptoms of neurologic impairment should be placed on 100% oxygen via a nonrebreather face mask while waiting for measurement, of COHb levels (On 100% oxygen, the half-life is reduced to 45 to 60 minutes). The diagnosis of CO poisoning can be made using COHb levels. If intubation is required, then hyperventilation with 100% oxygen should be instituted. The use of hyperbaric oxygen (HBO) therapy remains controversial.

the lower chest should be examined for the presence of signs of lower obstruction due to thermal injury (rarely) or smoke inhalation (most common). Respiratory tract injury is suggested by dyspnoea, cough, hoarseness, cyanosis and coarse crepitations or wheezes on auscultation. A chest X-ray and arterial blood sample might be needed. Severe complications such as haemorrhagic tracheobronchitis and ARDS require the attendance of a senior pulmonologist.

Encircling eschar impairing chest or abdominal expansion must be incised (escharotomy) or excised.

C FOR CIRCULATION

the shock in burn patients can be divided into two types;

primary shock; this is a psychogenic shock that passes spontaneously as occurs in most painful injuries. It requires no specific treatment.

Secondary shock; this occurs due to the loss of fluid (hypovolemic in nature) from the skin as explained earlier. The loss becomes significant within hours from the onset of burn and continues until it gradually decreases over 1-2 days as capillaries recover. This is how it differs from traumatic shock, that is; it develops slowly and thus allows more time for the splanchnic circulation to constrict. This however may damage the kidneys on the long run.

It is the duty of the surgeon, thus, to prevent circulator collapse from secondary shock. The focus is on the prevention rather than the repletion of the circulation. In mild burns oral fluid are given for replacement. If the burn is over 20% in extent (15% in children), establishing an intravenous infusion takes priority over a detailed history and physical examination. Once an infusion has been established, the pulse rate, blood pressure along with signs of restlessness and skin colour are monitored. In patients with burns of more than 25%, a catheter is inserted to measure hourly urine output. A nasogastric tube might be inserted.

Various formulae are available to help calculate replacement needs, but all are merely guiding and the amounts of fluid given must be adjusted in the

light of the patient's response to resuscitation. A good formula takes into consideration the following.

A good formula for fluid replacement

- **Rate of loss is maximum over the first few hours.**
- **Gradually declines over the next 1 to 2 days.**
- **In severe burn, the loss continues to 3rd day.**
- **The loss related to size of burned area.**
- **The loss is not related to the depth of burn.**
- **Most formula depends on size and weight.**

After admission, patient body weight is estimated. The extent of burn is then estimated. In the mount-vernon formula commonly used in the UK, the first 36 hours after injury are divided into six successive periods of 4,4,4, 6,6 and 12 hours. The volume of colloid (dextran, dried plasma) to be infused in each period is calculated from the equation:

Volume (ml) = burn area (%) X body weight (kg)/2

Recently, there has been a tendency to resuscitate major burns using crystalloids rather than colloid. The crystalloid solution to be used is ringer lactate as it simulates the plasma more than other crystal-loids. The Parkland formula is now widely used. The fluid volume in millilitres over the first 24 hours;

Volume (ml) = burn area (%) X body weight (kg) X 4

Half of that volume is given in the first 8 hours, the remainder over the next 16 hours.

The circulation can be assessed on hourly basis by checking the pulse, blood pressure, skin colour, signs of restlessness and urine output.

Daily water losses are replaced using 5% dextrose solution, taking care to avoid water intoxication, especially in young children, in the first few days following injury.

Blood should not be given in the first 24 hours but may be needed thereafter in patients with large full thickness bums. This is only needed in burns more than 10% BSA (each 1% deep burn you need 1% of patients blood volume). Haemoglobin concentration and haematocrit should be monitored regularly.

OTHER MEASURES

- Appropriate analgesia should be given. Se-vere pain is relieved by intravenous opiates.
- CVP monitoring when available.
- A metabolic fluid might be needed in adults 60 cc per hour.
- Tetanus can complicate burns, and tetanus toxoid is given if the patient has not received it re-cently.
- The wound provides a reservoir of infect-ing organisms. Catheters, cannula and tracheost-omy wounds are all potential sources of infection, prophylactic antibiotics are indicated to prevent the occurrence of sepsis. Wound decontamination takes priority to antibiotics. Infection often delays heal-ing and can cause further tissue destruction, con-verting the injury to a full-thickness one.

LOCAL TREATMENT OF BURN WOUNDS

Burn wounds differ from other wounds in many as-pects and thus merits special lines of management. The table below presents features which distinct bum wounds from traumatic wounds.

Features of burn wounds

- **It is colonized by potentially pathogenic bacteria.**
- **Frequently contains large amount of non-viable tissues.**
- **Exudes large quantities of water and serum**
- **Remain open for extended periods, not protected from bacteria**
- **It frequently requires viable tissues to be mobilized for the wound closure**

The methods used for treatment of burn wounds are discussed as follows.

EXPOSURE METHOD

The method implies continuous Cleaning with antiseptic agents and removing blisters and dead tissue. The required conditions are dryness, coolness and exposure to light. The principle of this method is drying of the area which might inhibits growth of bacteria as cooler surrounding, and ultraviolet light are hostile to bacterial growth. In addition, Evaporation of the protein-rich exudate leaves a dry, adherent crust that is an effective barrier to bacteria as long as it remains intact. It is used in single surfaces of trunk or limbs, face, perineum when the wound is extensive and complicated. Indications are shown in the table below.

Indications for using the exposure method

- **parts of the body which cannot be sealed off (perineum, face)**
- **only one surface involved**
- **when there is hyperpyrexia (temperature over 40 °C) that cannot be controlled**

DRESSING (CLOSED) METHO

The aim of this method is to provide mechanical barriers that prevent the invasion of bacteria-carrying particles. To absorb fluid exudates and to act as vehicle for antibacterial agent. After initial cleansing (with savlon, cetremide, hibiscrub) and application of an antiseptic agent (e.g. sulphadiazine cream), the wound is covered by a layer of sterile non adherent dressing, e.g. paraffin gauze or Mepotil, a layer of cotton gauze swabs, a bulky layer of cotton wool or Gamgee, and an outer retaining crepe bandage. The process does not require anaesthesia in most cases and sedation is only enough. The principle of this method is the fact that the majority of burn areas are resistant to infection or have no pathogenic organisms in the first few hours, and by providing an artificial barrier, the chance of infection is decreased.

Redressing should be done as less frequent as possible unless indicated and depends on duration of action of the antiseptic applied (e.g. 0.5% silver nitrate has to be wet all the time). The dressing is reviewed daily but left in place for 8-10 days, unless exudate soaks through to the outside or there is pain, feeling of hotness at that site or the wound becomes smelly. Common antiseptics used are shown in the table below.

Antiseptics used in burn wounds

- **NCP cream; Neomycin-Chorhexadine-Polymaxin**
- **Sulphamylon (Mafenide)**
- **Silver Sulphadiazine (Flamazine)**
- **Silver nitrate 0.5% to prevent infection**
- **Soframycin (sofratulle dressings)**

CIRCUMFERENTIAL BUMS.

The danger from encircling eschar (circumferential burns) surrounding the trunks and limbs is Increasing oedema beneath in the limbs which may imperil the circulation and involvement of the chest might impair respiration. The circulation should be monitored regularly with a Doppler ultrasound. Relieving incisions (escharotomy), which run from the top to the bottom of circumferential deep burns, may be needed in the first few hours after injury and are indicated when there is impairment of

circulation evident on doppler, or if the burn involves the neck. They can be done without anaesthesia, but analgesia is needed only in the case of full thickness burns (they are already anesthetized!).

BACTERIOLOGICAL STUDIES AND INFECTION

Bacteriological studies are principled on counting number of bacteria in one gram of tissues which correlates well with the severity of infection. Swab culture required at different times, usually on admission on the 3rd or 4th day, Pre-grafting and In case of sepsis. The Severity of infection decided by the number of bacteria in one gram of tissues. Contamination is defined as the presence of bacteria on the surface.

When Infection occurs, the discharge becomes wet and purulent and the margins become red. Angry and unhealthy-looking wounds suggest haemolytic streptococcal infections. *Pseudomonas aerogenosa* is suggested by the presence of bluish green pus. Large amounts of foul-smelling discharge suggest *proteus vulgaris.* The danger of infection is present virtually over the whole period of illness. If antibiotics are given for long period resistant organisms will appear. *P.aerogenosa* most troublesome invader. In severe burn body's defence greatly disturbed and the incidence of sepsis rises proportionately.

Different burn units have different antibiotics policies. In general, they depend on the status of the patient, the presence of concomitant diseases, the age of the patient, and the sterility of the area to be managed.

SURGICAL MANAGEMENT OF BURN WOUNDS

The aim of burn wounds treatment in general is to restore epidermal cover and get the best cosmetic and functional results. Surgery is indicated in cases of failure of conservative methods to get to this aim, the presence of full thickness burns and in some cases of deep partial thickness burns where healing is unsuccessful.

Surgical wound management is presented by using skin grafts. The use of skin grafts can be early (with the first week) or late (after 3 weeks) depending on estimation of the chance of recovery by conservative approaches. When managed by this method, the dead outer layers of skin are shaved away down to the deep-dermal layer and a split thickness skin graft (or full thickness) is applied immediately. Most commonly the grafts are taken from the trunk, the thighs, the arms and the legs. Meshed skin grafts are used to expand the graft.

Note; more discussion on grafts from this lecture has been transferred to "skin reconstruction by soft tissue lecture".

ELECTRICAL BURN.

An electric burn or shock occurs when a person comes into contact with an electrical energy source. Electrical energy flows through a portion of the body causing a shock. Exposure to electrical energy may result in no injury at all or may result in devastating damage or death.

Many variables determine what injuries may occur, if any. These variables include the type of current (AC or DC), the amount of current (determined by the voltage of the source and the resistance of the tissues involved), and the pathway the electricity takes through the body. Low voltage electricity (less than 500 volts) does not normally cause significant injury to humans. Exposure to high voltage electricity (greater than 500 volts) has the potential to result in serious damage.

Children are not often seriously injured by electricity. They are prone to shock by the low voltage (110-220 volts) found in typical household current. In children aged 12 years and younger, household appliance electrical cords and extension cords caused more than 63% of injuries in one study. Wall outlets were responsible for 15% of injuries.

ELECTRIC SHOCK SYMPTOMS

- Burns are usually most severe at the points of contact with the electrical source and the ground. The hands, heels, and head are common points of contact.

- In addition to burns, other injuries are possible if the person has been thrown clear of the electrical source by forceful muscular contraction. Consideration should be given to the possibility of a spine injury. The person may have internal

injuries especially if he or she is experiencing any shortness of breath, chest pain, or abdominal burn.

- Pain in a hand or foot or a deformity of a part of the body may indicate a possible broken bone resulting from the electric shock.
- In children, the typical electrical mouth burns from biting an electric cord appears as a burn on the lip. The area has a red or dark, charred appearance.

MEDICAL TREATMENT

The doctor's primary concern is to determine if significant unseen injury exists. Injury may occur to muscles, the heart, or the brain from the electricity or to any bones or other organs from being thrown from the electric source. Tests may include any or none of the following:

- ECG to check the heart
- Complete blood count
- Blood or urine test or both for muscle enzymes (would indicate significant muscle injury)
- X-rays to look for fractures or dislocations, both of which may be caused by a near electrocution
- CT scan

Treatment depends on the severity of the burns or the nature of other injuries found:

- Minor burns may be treated with topical antibiotic ointment and dressings.
- More severe burns may require surgery to clean the wounds or even skin grafting.
- Severe burns on the arms, legs, or hands may require surgery to remove damaged muscle or even amputation.
- Other injuries may require treatment:
- eye injuries may require examination and treatment by an ophthalmologist.
- Broken bones require splinting, casting, or surgery to stabilize the bones.
- Internal injuries may require observation or surgery.

PROGNOSIS:

Recovery from electric shock depends on the nature and severity of the injuries. The percentage of the body surface area burned is the most important factor affecting prognosis.

If someone who has received an electric shock does not suffer immediate cardiac arrest and does not have severe burns, he or she is likely to survive.

Infection is the most common cause of death in people hospitalized following electrical injury.

Electrical damage to the brain may result in a permanent seizure disorder, depression, anxiety, or other personality changes.

CHEMICAL BURN.

- chemical burn is irritation and destruction of human tissue caused by exposure to a chemical, usually by direct contact with the chemical or its fumes. Chemical burns can occur in the home (Bleach, Concrete mix, Drain or toilet bowl cleaners, Metal cleaners, Pool chlorinators), at work or school, or as a result of accident or assault.

Most chemical burns are caused by either strong acids or strong bases (for example, hydrochloric acid or sodium hydroxide). Acids damage and kill cells by coagulating cells while bases liquefy cells. Prolonged exposure can severely damage human tissues and, if the patient survives, leads to scarring and disability. Other chemicals like oxidants and certain metals may also produce similar chemical burns. Limiting the time of exposure to any of these chemicals can greatly reduce their damaging effects.

Tissue damage from chemical burns depends on several factors.

- The strength or concentration of the agent
- The site of contact (eye, skin, mucous membrane)
- Whether swallowed or inhaled

- Whether or not skin is intact
- With the quantity of the chemical
- The duration of exposure
- How the chemical works
- The length of time to washing (decontamination)

signs and symptoms of chemical burns include the following:

- Redness, irritation, or burning at the site of contact
- Pain or numbness at the site of contact
- Formation of blisters or black dead skin at the contact site
- Vision changes if the chemical gets into the eyes
- Cough or shortness of breath
- Vomiting.

In severe cases, a person may develop any of the following symptoms:

- Low blood pressure.
- Faintness, weakness, dizziness.
- Shortness of breath or severe cough.
- Headache.
- Muscle twitching or seizures.
- Cardiac arrest or irregular heartbeat.

Chemical Burn Treatment:

Most people with minor chemical burns do not need to be admitted. Most can go home after arranging follow-up care with a doctor. Patients with major chemical burns however, need to be admitted to a hospital. Ingestion or inhalation of chemical burns may need to be admitted for observation, depending on the potential severity of tissue damage.

- IV fluids may be needed to normalize blood pressure and heart rate as any type of burn (fire, chemical, sun exposure) often results in dehydration of the patient.
- The IV access may also be used for any medications needed to treat pain or protect against infection.
- Decontamination will begin (likely water irrigation).
- Some people may be an antidote to counteract the chemical, if appropriate.
- Antibiotics often are not needed for minor chemical burns.
- Wounds will be cleaned and bandaged with medicated creams and sterile wraps as needed.
- Consultation with other medical specialists may be done if indicated.
- Pain in a burn can often be severe. Adequate pain control will be addressed by the doctor.
- If there is any indication of breathing problems, a breathing tube may be placed in the patient's airway to help maintain the airway and provide adequate ventilation.

CAUSTIC IN-JURY

Accidental caustic lesions occur mainly in children, and, in general, rather small quantities of caustics are taken.

In adults or teenagers, the swallowing of caustic liquids is usually deliberate, during suicide attempts, and greater quantities are swallowed. Alkalies are more frequently swallowed accidentally than acids, because strong acids cause an immediate burning pain in the mouth.

PATHOLOGY

The swallowing of caustic substances causes an acute and a chronic injury.

During the acute phase, care focuses on controlling the immediate tissue injury and the potential for perforation.

During the chronic phase, the focus is on treatment of strictures and disturbances in pharyngeal swallowing.

In the acute phase, the degree and extent of the lesion are dependent on several factors: the nature of the caustic substance, its concentration, the quantity swallowed, and the time the substance is in contact with the tissues.

Acids and alkalines affect tissue in different ways. Alkalies dissolve tissue, and therefore penetrate more deeply, while acids cause a coagulative necrosis that limits their penetration.

Animal experiments have shown that there is a correlation between the depth of the lesion and the concentration of sodium hydroxide solution. When a solution of 3.8% comes into contact with the esophagus for 10 seconds, it causes necrosis of the mucosa and the submucosa, but spares the muscular layer. A concentration of 22.5% penetrates the whole esophageal wall and into the periesophageal tissues.

Cleansing products can contain up to 90% sodium hydroxide.

The strength of esophageal contractions varies according to the level of the esophagus, being weakest at the striated muscle-smooth muscle interface. Consequently, clearance from this area may be somewhat slower, allowing caustic substances to remain in contact with the mucosa longer. This explains why the esophagus is preferentially and more severely affected at this level than in the lower portions.

The lesions caused by lye injury occur in three phases.

First is the acute necrotic phase, lasting 1 to 4 days after injury. During this period, coagulation of intracellular proteins results in cell necrosis, and the living tissue surrounding the area of necrosis develops an intense inflammatory reaction.

Second is the ulceration and granulation phase, starting 3 to 5 days after injury. During this period, the superficial necrotic tissue sloughs, leaving an ulcerated, acutely inflamed base, and granulation tissue fills the defect left by the sloughed mucosa. This phase lasts 10 to 12 days, and it is during this periord that the esophagus is the weakest.

Third is the phase of cicatrization and scarring, which begins the third week following injury. During this period, the previously formed connective tissue begins to contract, resulting in narrowing of the esophagus. Adhesions between granulating areas occur, resulting in pockets and bands. It is during this period that efforts must be made to reduce stricture formation.

CLINICAL MANIFESTATIONS

The clinical picture of an esophageal burn is determined by the degree and extent of the lesion.

In the initial phase, complaints consist of pain in the mouth and substernal region, hypersalivation, pain on swallowing, and dysphagia. The presence of fever is strongly correlated with the presence of an esophageal lesion. Bleeding can occur, and frequently, the patient vomits. These initial complaints disappear during the quiescent period of ulceration and granulation.

During the cicatrization and scarring phase, the complaint of dysphagia reappears and is due to fibrosis and retraction, resulting in narrowing of the esophagus.

Of the patients who develop strictures, 60% do so within 1 month, and 80% within 2 months. If dysphagia does not develop within 8 months, it is unlikely that a stricture will occur.

Serious systemic reactions such as hypovolemia and acidosis resulting in renal damage can occur in cases in which the bums have been caused by strong acids. Respiratory complications such as laryngospasm, laryngoedema, and occasionally pulmonary edema can occur, especially when strong acids are aspirated.

Inspection of the oral cavity and pharynx can indicate that caustic substances were swallowed, but does not reveal that the esophagus has been burned.

Conversely, esophageal burns can be present without apparent oral injuries. Because of this poor correlation, early esophagoscopy is advocated to establish the presence of an esophageal injury. To lessen the chance of perforation, the scope should not be introduced beyond the proximal esophageal lesion. The degree of injury can be graded according to the criteria listed in Table 1.

Even if the esophagoscopy is normal, strictures may appear later.

Radiographic examination is not a reliable means to identify the presence of early esophageal injury, but is important in later follow-up to identify strictures. The most common locations of caustic injuries are shown in Table 2.

Table 1 Endoscopic Grading of Corrosive Esophageal and Gastric Burns
First degree: Mucosal hyperemia and edema
Second degree: Limited hemorrhage, exudate ulceration, and pseudomembrane formation
Third degree: Sloughing of mucosa, deep ulcers, massive hemorrhage, complete obstruction of lumen by edema, charring, and perforation

Table 2 Location of Caustic Injury (n=62)	
Pharynx	10%
Esophagus	70%
Upper	15%
Middle	65%
Lower	2%
Whole	18%
Stomach	20%
Antral	91%
Whole	9%
Both stomach and esophagus	14%

TREATMENT

Treatment of a caustic lesion of the esophagus is directed toward management of both the immediate and late consequences of the injury.

The immediate treatment consists of limiting the burn by administering neutralizing agents. To be effective, this must be done within the first hour.

Lye or other alkali can be neutralized with half-strength, vinegar, lemon juice, or orange juice.

Acid can be neutralized with milk, egg white, or antacids.

Sodium bicarbonate is not used because it generates carbon dioxide, which might increase the danger of perforation. Emetics are contraindicated, because vomiting renews the contact of the caustic substance with the esophagus and can contribute to perforation if too forceful.

Hypovolemia is corrected and broad-spectrum antibiotics are administered to lessen the inflammatory reaction and prevent infectious complications. If

necessary, a feeding jejunostomy tube is inserted to provide nutrition. Oral feeding can be started when the dysphagia of the initial phase has regressed.

In the past, surgeons waited until the appearance of a stricture before starting treatment. Currently, dilations are started the first dav after the injury, with the aim of preserving the esophageal lumen by removing the adhesions that occurred in the injured segments. However, this approaches controversial in that dilations can traumatize the esophagus, causing bleeding and perforation, and there are data indicating that excessive dilations cause increased fibrosis secondary to the added trauma.

The use of steroids to limit fibrosis has been shown to be effective in animals, but their effectiveness in human beings is debatable.

Extensive necrosis of the esophagus frequently leads to perforation, and is best managed by resection. When there is extensive gastric involvement, the esophagus is nearly always necrotic of severely burned, and total gastrectomy and near-total esophagectomy are necessary.

The presence of air in the esophageal wall is a sign of muscle necrosis and impending perforation and is a strong indication for esophagectomy.

Management of acute injury is summarized in the algorithm in Fig. 25-80. Some authors have advocated the use of an intraluminal esophageal stent in patients who are operated on and found to have no evidence of extensive esophagogastric necrosis. In these patients, a biopsy of the posterior gastric wall should be performed to exclude occult injury.

If, histologically, there is a question of viability, a second-look operation should be done within 36 hours. If a stent is inserted, it should be kept in position for 21 days, and removed after a satisfactory barium esophagogram. Esophagoscopy should be done, and, if strictures are present, dilations initiated.

Once the acute phase has passed, attention is turned to the prevention and management of strictures. Both antegrade dilation with a Hurst or Maloney bougie and retrograde dilation with a Tucker bougie have been satisfactory. Occasionally, particularly with severe strictures, the patient is instructed to swallow a string, over which metal Sippy dilators are passed until an adequate lumen can be obtained for passage of a mercury bougie.

In a series of 1079 patients, early dilations started during the acute phase gave excellent results in 78%, good results in 13%, and poor results in 2%. Fifty-five patients died during the treatment

In contrast, of 333 patients whose strictures were dilated when they became symptomatic, only 21% had excellent results, 46% good, and 6% poor, with three dying during the process. The length of time the surgeon should persist with dilation before consideration of esophageal resection is problematic.

An adequate lumen should be re-established within 6 months to 1 year, with progressively longer intervals between dilations. If, during the course of treatment, an adequate lumen cannot be established or maintained (i.e., smaller bougies must be used), operative intervention should be considered.

SURGICAL INTERVENTION IS INDICATED WHEN THERE IS

(a) complete stenosis in which all attempts from above and below have failed to establish a lumen,

(b) marked irregularity and pocketing on barium swallow.

(c) the development of a severe periesophageal reaction or mediastinitis with dilatation,

(d) a fistula,

(e) the inability to dilate or maintain the lumen above a 40 F bougie, or

(f) a patient who is unwilling or unable to undergo prolonged periods of dilation.

The variety of abnormalities seen requires that creativity be used when considering esophageal reconstruction. Skin tube esophagoplasties are now used much less frequently than they were in the past, and are mainly of historical interest

Currently, the stomach, jejunum, and colon are the organs used to replace the esophagus, through either the posterior mediastinum or the retrosternal route.

A retrosternal route is chosen when there has been a previous esophagectomy or there is extensive fibrosis in the posterior mediastinum. When all factors are considered, the order of preference for an esophageal substitute is (a) colon, (b) stomach, and (c) jejunum. Free jejunal grafts based on the superior thyroid artery have provided excellent results. Whatever method is selected, it must be emphasized that these procedures be taken

lightly; minor errors of judgment or technique may lead to serious or even fatal complications.

Critical in the planning of the operation is the selection of cervical esophagus, pyriform sinus, or posterior pharynx as the site for proximal anastomosis.

The site of the upper anastomosis depends on the extent of the pharyngeal and cervical esophageal damage encountered. When the cervical esophagus is destroyed and a pyriform sinus remains open the anastomosis can be made to the hypopharynx.

When the pyriform sinuses are completely stenosed, a transglottic approach is used to perform an anastomosis to the posterior oropharyngeal wall This allows excision of supraglottic strictures and elevation and anterior tilting of the larynx.

In both of these situations, the patient must relearn to swallow. Recovery is long and difficult and may require several endoscopic dilations, and often reoperations.

Sleeve resections of short strictures are not successful because the extent of damage to the wall of the esophagus can be greater than realized, and almost invariably the anastomosis is carried out in a diseased area.

The management of a bypassed damaged esophagus after injury is problematic. If the esophagus is left in place, ulceration from gastroesophageal reflux or the development of carcinoma must be considered.

The extensive dissection necessary to remove the esophagus, particularly in the presence of marked periesophagitis, is associated with significant morbidity.

Leaving the esophagus in place preserves the function of the vagus nerves, and, in turn, the function of the stomach.

On the other hand, leaving a damaged esophagus in place can result in multiple blind sacs and subsequent development of mediastinal abscesses years later.

Most experienced surgeons recommend that the esophagus be removed unless the operative risk is unduly high.

CLEFT LIP/PALATE

Cleft lip/palate is a condition where by the lip and/or palate of neonates is incompletely fused. Cleft lip, with or without cleft palate, affects one in 700 babies annually, and is the fourth most common birth defect in the U.S.

EMBRYOLOGY

During early intrauterine life (5th week), two facial clefts are identified. The first cleft is formed between the frontal and maxillary processes and will form the future nasolacrimal duct. The second cleft is formed between the lateral and medial nasal processes and will form the nasal (olfactory) pits (figure 1). The palate develops from the primary and the secondary palate (figure 2). The primary palate develops from the innermost part of the medial nasal process. The secondary palate is formed of two shelf-like outgrowths from the maxillary process. In the seventh week, these shelves fuse with each other to form the secondary palate. Anteriorly, these shelves also fuse with the primary palate and cranially with the nasal septum. The incisive foramen is considered the midline landmark between the primary and secondary palate.

INTRODUCTION

All human beings, at one point, had cleft lip and palate. During normal fetal development, between 6th and 11th (roughly 3 months) week of pregnancy, the clefts fuse together. A cleft lip/palate results from failure of the process of palate and lip fusion beyond this period, leaving a cleft that requires surgical repair.

CLASSIFICATION

A cleft lip range from a tiny notch in upper lip to a split extending into the nose. Cleft Palate on the other hand range from small malformation that result in minimal problem to a large separation that interfere with eating, speaking, and even breathing. Clefts can be referred to as unilateral or bilateral.

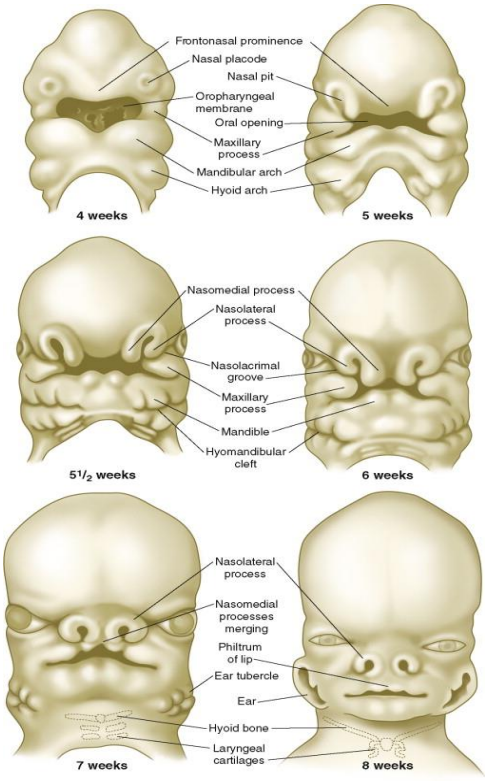


Figure 14 development of the nasal frontal and maxillary processes

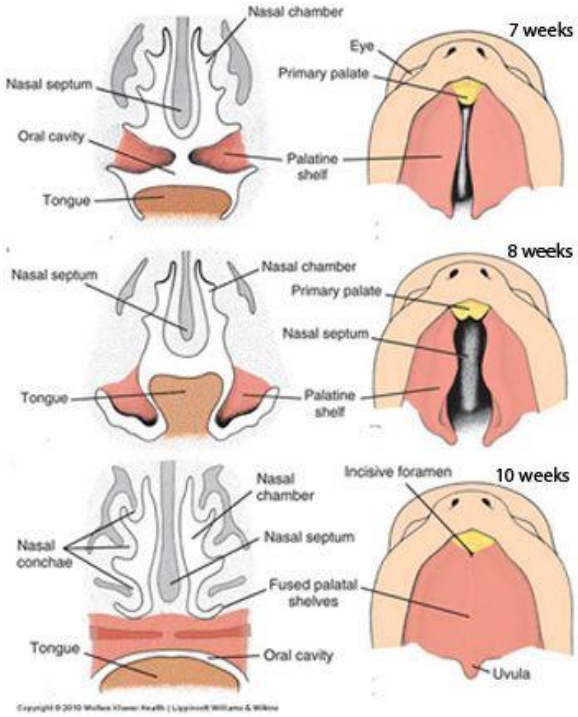


Figure 15 development of the palate

Cleft lip/palate is classified into isolated cleft lip (20%), isolated cleft palate (30%) and cleft lip and palate (50%). About 13% of babies affected with both cleft lip and palate are affected with other anomalies (e.g. cardiac).

Classification of cleft lip/palate			
	Cleft lip	Cleft palate	both
incidence	1:2000	1:1000	1:600
percentage	20%	30%	50%
Sex	females	females	males

Cleft lip can be further classified into complete (where the cleft extends in the nostril floor) or incomplete (where a tissue bridge unites the lateral and central lip). When the cleft of the hard palate remains attached to the nasal septum and vomer, the cleft is termed incomplete. When the nasal septum and vomer are completely separated from the palatine processes, the cleft palate is termed complete.

Victor Veau's classification (1931) of cleft lip :

- Cleft lip Class I : U/L notching of vermillion border, not extending into the lip.
- Class II : cleft extending into the lip, but not including the floor of the nose.
- Class III: extending into the floor of the nose.
- Class IV: any b/l cleft of the lip, whether incomplete or complete.

Cleft palate

- Class I: soft palate
- Class II: soft/hard palate extending no further than incisive foramen.
- Class III: complete unilateral cleft, extending from uvula to incisive foramen, then deviating to one side Class
- IV: two clefts extending forward from the incisive foramen into the alveolus.

ETIOLOGY.

The cause of cleft lip/palate is yet to be identified. Most believe it a genetic predisposition and a contributory environmental component. Genetic predisposition is noted on the high incidence among babies with positive family history of first degree relatives. Genetic predisposition is noted to be strongly affected from the mother side. Environmental factors implicated in clefting include maternal poor health in early pregnancy, exposure to toxins such as cocaine and alcohol, and maternal epilepsy and drugs, e.g. steroids, diazepam and phenytoin.

Cleft lip/palate are an associated feature in over 300 recognized syndromes.

DIAGNOSIS

A cleft lip or palate is usually apparent during the newborn's first examination. Clefts of the lip are also usually obvious. Isolated palate clefts are best diagnosed by direct inspection using a tongue depressor and a small torch. Passing a finger along the palate may miss a minor submucous cleft (palatal mucosa intact but palatal muscles not fused in the midline); a bifid uvula might be seen in this form of cleft.

An antenatal diagnosis of cleft lip, whether unilateral or bilateral, is possible by ultrasound scan after 18 weeks of gestation. Isolated deft palate cannot be diagnosed by antenatal scan.

COMPLICATIONS

- Breathing; when palate and jaw are malformed, breathing becomes difficult. Severe breathing problems and recurrent aspiration are seen in Pierre Robin sequence, in which the palatal cleft is associated with a receding lower jaw and posterior and cephalic displacement of the tongue, obstructing the naso-oropharyngeal airway.

- Feeding; Infant with cleft lip/palate are unable to suck, as a result, the bottle milk needs to be inclined downwards, and the nipple of the bottle dilated so as to allow milk to go down due to gravity. Special feeding devices are present. As a rule, enlarging the openings in an artificial nipple or using a syringe with a soft rubber feeding tube will solve difficulties in sucking.

- Ear infection and hearing loss; Any malformation in the upper airway can affect the function of the Eustachian tubes and increase the possibility of persistent fluid in middle ear. This causes repeat infection and may lead to hearing loss.

- Speech and language delays; normal lip and palate essential for proper sounds, and speech therapy needed after surgery. If not improved, a repeat surgery is needed.

- Dental problems; cleft alveolus affect proper teething and produce non-alignment of the teeth. Early orthodental management is needed.

MANAGEMENT

CLEFT LIP

Surgical repair of cleft lip is not considered an emergency. The optimal time for operation can be described as the widely accepted "rule of 10." This includes body weight of 10 lb (4.5 kg) or more, a haemoglobin of 10 g/dL or more, a white blood cells count less than 10,000, and an age more than 10 weeks

In unilateral cleft lip, closure of the lip will mold distortions of the cleft alveolus into a satisfactory contour. For bilateral cases, closure is done at the same time, or with period of 6 weeks in between.

In occasional cases in which there is marked distortion of the alveolus, such as in severe bilateral clefts with marked protrusion of the premaxilla, preliminary maxillary orthodontic treatment may be indicated. This may involve the use of carefully crafted appliances or simple constant pressure by use of an elastic band.

CLEFT PALATE

Purpose of closure of palate is to enable the child to develop normal speech with swallowing without regurgitation. Requirements (essential) for normal speech are a soft palate that is sufficiently mobile and long enough to close the oronasal nasal sphincter. It is important to perform the surgery Before the child begins learning to speak at age of two as Palate is important for speaking.

This is usually done at 15-18 months of age.

TRAUMA

INTRODUCTION

Trauma is a major public health problem and represents the most common cause of death among patients aged 1-45 years. In the united states, about 60 million people are injured annually with 30 million of those requiring treatment and 3 million requiring hospitalization. The financial costs of injury are staggering and exceed $400 billion annually due to hospitalization expenses and the loss of working young people.

The causes of trauma are shown in the table below.

The most common cause of trauma is road traffic accidents (RTA).

Causes of trauma	
59%	Motor vehicle accidents
22%	Gunshot wounds
9%	Falls
Others	Thermal, chemical, blast, stabs.

Trauma deaths have been classically described as having a trimodal distribution (Figure 1), with peaks that correspond to the types of intervention that would be most effective in reducing mortality. The first peak, the immediate deaths (50%), represents patients who die of their injuries before reaching the hospital. The injuries accounting for these deaths include major brain or spinal cord trauma and those resulting in rapid exsanguination (heart or aortic laceration). Prevention remains the major strategy to reduce these deaths.

The second peaks, the early deaths (30%), are those that occur within the first few hours after injury. Half are caused by internal haemorrhage, and the other half, by central nervous system injuries. Almost all of these injuries are potentially treatable.

The third peak, the late deaths, consists of patients who die days or weeks after injury. Ten percent to 20% of all trauma deaths occur during this period. Mortality for this period has traditionally been attributed to infection, pulmonary embolism and multiple organ failure. Improvements in critical care management continue to be essential in reducing deaths during this phase.

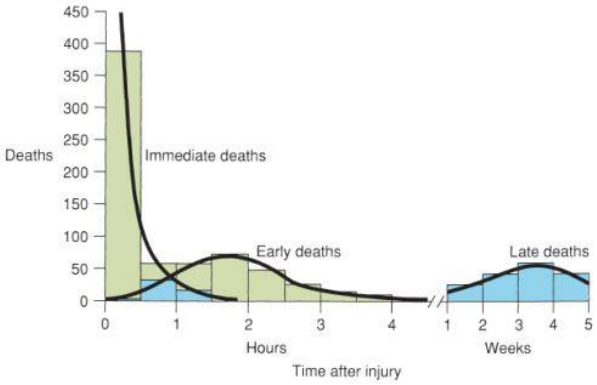


Figure 16 peaks of increased mortality after injury

TRAUMATOLOGY AND TRAUMA SYSTEMS

The practice of preventing and minimizing death (mortality) and disability (morbidity) resulting from trauma is termed traumatology. The challenge is Initial treatment till arrival to a nearby medical centre and detection and evaluation of life saving problems and the quick action. Trauma systems have been proposed to handle both routine events and mass casualty situations. The purpose of a trauma system is to provide timely, organized care in order to minimize preventable morbidity and mortality following injury. a trauma system has the following consideration;

- Access to care
- Pre-hospital care
- Hospital care
- Rehabilitation
- Injury prevention

ACCESS TO CARE

A trauma system should focus on making the process of reaching to a trauma care facility possible. This can be provided through the availability of an emergency room phone number, the presence of emergency phones along major roads, and the availability of safe and rapid transport (e.g. ambulances and helicopters) from accident locus to hospital, with constant communication. This will provide a reasonable access to care.

PRE-HOSPITAL CARE

The presence of well-educated paramedical personnel and even laypersons has a significant effect in preventing mortality and morbidity. Training should be focused on basic life support care (BLSC) and advanced trauma life support (ATLS) care (see later). Pre-hospital care is promoted with the availability of well-equipped helicopters and ambulances.

HOSPITAL CARE

An ideal hospital care is provided at a well-designed trauma centre with available diagnostic services, well trained doctors experienced in dealing with trauma, and the presence of restorative care units (RCU) and critical care beds.

REHABILITATION

The rehabilitation phase focuses on restoring the function of the patient as it was before injury, rehabilitation is both biological and psychological.

PREVENTION

Prevention strategies remain effective in avoiding the occurrence of a trauma. They include education of the public and on an individual level along with strict government regulations (e.g. wearing seatbelts)

BASIC LIFE SUPPORT

Basic life support (BLS) is levels of cares that is taught to medical and paramedical personnel and even the layperson and is employed to patients who has been traumatized and seems to have lost consciousness or are mentally disoriented. It aims to initiate CPR.

An attempt to wake The unconscious patient is made, this is followed by opening the airway through a head tilt, chin left and jaw thrust manoeuvre, checking for breathing (listening to the mouth, looking at the chest and feeling for air), and checking for presence of the carotid pulse (or if the patient is moving or coughing). Patients who do not breath with presence of a carotid pulse, should be artificially ventilated while those who do not breath with absent carotid pulse should undergo chest compressions with artificial breathing in a ratio of 30:1.

ADVANCED TRAUMA LIFE SUPPORT

the Advanced Trauma Life Support (ATLS) is a training program for doctors that focuses on the management of acute trauma cases, and was developed by the American College of Surgeons. This program has been adopted worldwide in over 30 countries. The process begins with the identification and treatment of conditions that constitute an immediate threat to life. The ATLS course refers to this as the primary survey. Any life-threatening problem identified in the initial survey must be treated before advancing to the next step. Once this is achieved, other injuries the patient might have is then identified. This is called the secondary survey.

THE PRIMARY SURVEY

the considerations of the primary survey has been designated as ABCDE, indicating the following;

A FOR ADEQUATE AIRWAY

Ensuring an adequate airway is the first priority in the primary survey. The following is done;

- Any clothes around the neck or the airway should be removed and the mouth is cleaned.
- Jaw thrust manoeuvre; head tilt, chin left is performed.
- Generally, patients do not have a risk on their airway if their voice is normal, if abnormal (hoarseness or stridor) or are unconscious, then they should undergo further evaluation, laryngoscopic view is important for suction of vomit, foreign bodies or blood and if not available, The patient should be intubated (orally, nasally) accordingly, especially those with altered mental states.
- Patients with severe maxillofacial injuries should have an artificial airway surgically formed (i.e. Cricothyroidotomy, tracheostomy).
- all blunt trauma patients require cervical spine immobilization until injury is ruled out. This can be accomplished with a hard collar or the placement of sandbags on both sides of the head taped to the back board. Soft collars do not immobilize the cervical spine.

B FOR BREATHING

Once a secure airway is obtained, adequate oxygenation and ventilation must be assured. The following is considered;

- Oxygen is usually provided by an oxygen mask
- Pulse oximeter is used to monitor oxygen saturation and if the patient is intubated, end tidal CO2 is checked.
- The following conditions may constitute an immediate threat to life due to inadequate ventilation: tension pneumothorax, open pneumothorax, massive hemothorax, or flail chest/pulmonary contusion. All of these diagnoses can be made with a combination of physical examination and chest x-ray. Thus, one must look for asymmetric chest expansion, abnormal chest movement, the presence of apparent wounds and an extra- respiratory effort, the doctor should also see for tenderness, air crepitus (surgical emphysema) or bone crepitus (rib fractures). Auscultation is important to hear for air entry.

C FOR CIRCULATION

With a secure airway and adequate ventilation established, circulatory status is addressed next;

- An external source of bleeding is searched at first. If apparent, an attempt to stop it is taken immediately (pressure by tourniquet, clamping or compressing the bleeder vessel, fluid resuscitation).
- When there is no apparent external bleeding, internal bleeding should be checked. If there is any sign of internal bleeding (absent or rapid pulses, hypotension, pallor, mental deterioration) then site of bleeding is determined and the patient is treated as being shocked.

D FOR DYSFUNCTION OF CNS

After adequate ventilation and tissue perfusion have been restored, immediate attention must be given to the patient's neurologic condition, assessment of neurological injury is done through the following;

- Level of consciousness; wither the patient is alert, confused or unconscious or by using the AVPU method see the table below.
- Pupil size and response to light
- Motor activity and tactile sensation

Method of assessing level of consciousness
AVPU
• **Alert**
• **Responds to Verbal stimuli**
• **Responds to Painful stimuli**
• **Unresponsive**

Patients who are disoriented are treated as having head trauma until proven otherwise. In this case consultation with neurosurgeon is essential.

E FOR EXPOSURE

Undress patient completely and assess the entire body from head to toe for any other unseen injury. Hypothermia can be rapid following trauma, and warming air blankets are vitally important in the resuscitative phase.

SECONDARY SURVEY

After completion of the primary survey, a secondary survey is needed to assure that the patient is needed to check for the presence of injuries that might be life threatening later on. A quick review of the history is made (see the table below) followed by detailed examination of the whole body from head to toe.

Review of patient's history (AMPLE)
• **Allergy**
• **Medication including tetanus status**
• **Past medical history**
• **Last meal**
• **events of the incident**

MASS CASUALTIES

The triage is a system of sorting out (classification) causalities into those that require immediate attention and those that can have their care delayed (see the table below). An effective system of triage is that which puts the patient at the right place, in the right time with the right care provider.

Classification of patients in mass causalities
• **Critical patient; cannot wait**
• **Urgent case; can wait short time**
• **Less serious; not endangered by delay**

The first Field Triage Decision Scheme was published by the American College of Surgeons (ACS), the criteria of decision making are shown in the table below. The decision making is divided into steps and if any criterion is identified at each step, then transport to a trauma centre is immediate.

In any event of mass causalities, pre-event management is important, the establishment of a defined system with central control is critical for the orderly evacuation and transfer of patients. Staff training is an obvious crucial element of disaster planning.

At the event, Anaesthesiologists, general and orthopaedic surgeons are in immediate demand. The Radiology department is the main bottleneck impeding the orderly flow of patients.

ACS criteria for triage decision making scheme
Physiologic response • **Glasgow Coma Scale of <14** • **systolic blood pressure (SBP) of <90 mmHg** • **respiratory rate of <10 or >29 breaths per minute(<20 in infant aged <1 year).**
Anatomic criteria • **flail chest** • **two or more proximal long-bone fractures** • **pelvic fracture** • **crushed, degloved, or mangled extremity** • **amputation proximal to wrist and ankle** • **major burn** • **combination of trauma and bum** • **open or depressed skull fracture** • **paralysis**
Injury mechanism • **Fall >6 meter** • **Rollover** • **ejection from car** • **death in the same compartment** • **extraction time > 20 min** • **major car deformity> 20 inches**
Comorbid factors • **Age <5 or>55** • **Pregnancy** • **insulin dependent** • **bleeding tendency**

HEAD TRAUMA

Head injury comprises a large proportion of emergency neurosurgical practice. Trauma is the leading cause of death among individuals under the age of 45 years and 60% of trauma patients deaths are due to head injury. Every minute there are 4 heads injured and every 12 minutes there is 1 death from head injury. One of the problems facing many hospitals in managing patients with head trauma is the lack of neurosurgeons. Head injuries can involve the scalp, the skull, and intracranial structures and may be associated with direct injury to the face, and may be penetrating (open) or non-penetrating (closed). Head injuries by themselves, are described as 'primary insult' or 'primary head injury' and these may directly cause a 'primary *brain* injury'. When there is enough bleeding to cause shock, the resultant ischemia, and hypoxia taking place in the brain are called 'secondary insult' or 'secondary brain injury*. Secondary brain damage can have a devastating effect on what may initially have been a relatively minor injury and amenable to prevention and treatment , People might die from second insult more than they might die from first insult so the first step in management is hyperventilation and then circulatory problems if found

PATHOPHYSIOLOGY

The level of consciousness is the hallmark of brain injury, deterioration of which can result from injury to the reticular activating system, increased intracranial pressure (which may result in decreased cerebral perfusion pressure), or decreased cerebral blood flow as occurs in secondary insults. When there is brain hypo perfusion (CPP should be more than 50 mmHg), the brain responds by activating the sympathetic system to increase blood pressure, thus increasing cerebral blood flow. This is called 'central nervous ischemic response and is evident in secondary brain injuries and brain edema (decreases CPP). It explains why every patient with closed head - trauma who might have injured one of the arteries supplying the brain, or whom had contusions that increased intracranial pressure and thus decreased there CPP, have markedly increased blood pressure. According to this, it's important to note that blood loss from a head injury is not sufficient to lower the blood pressure and so *you should never presume brain injury is the cause of hypotension unless it is a late stage head injury* (when there is severe blood loss and the central nervous ischemic response is no longer effective), an increase in intracranial pressure secondary to brain edema, hematoma, or hypoxia can be fatal as it decreases the CPP and if severe enough can cause herniation through the foramen magnum, which in turn can result in compression on the brain stem, thus causing immediate death.

FORMS OF HEAD INJURIE

SUBGALIAL HEMATOMA

Injuries that involve the highly vascular loose connective tissue beneath the galial aponuerosis can result in what is called subgalial hematoma, subgalial hematoma manifests as diffuse hematoma in the scalp. The blood may dissect along the loose connective tissue layer towards that around the eyes causing 'raccoon eyes' and behind the ear causing 'battles sign'. The later 2 are typical of basal skull fractures, and thus many subglial hematomas are sometimes misdiagnosed as basal skull fractures, subgalial hematoma is very dangerous in children as their central compartment is not sufficient to withstand the enormous blood loss resulting from such condition. (As little as 100 cc can cause shock).

SUBPERIOSTEAL HEMATOMA

Injury to one of the arteries passing in the external periosteum of the skull can cause subperiosteal hematoma. The hematoma is usually focal, but sometimes may involve one bone of the calvarial skeleton, subperiosteal hematoma is more dangerous than subgalial hematoma as it may compress the periosteal vessels and cause bone necrosis so it needs urgent management

SKULL FRACTURES

These are 5 types;

- **linear skull fractures**; these are breaks in the bone that transverse the full thickness of the skull from the outer to inner table and they are either across the vascular grooves or across suture lines which are more dangerous , are usually fairly straight and involve no displacement of the bone. The common method of injury is blunt force trauma

in which the energy from the blow is transferred over a wide surface area of the skull. Linear fractures of the skull are usually of little clinical significance unless they parallel in close proximity or transverse a suture, or they

Involve a venous sinus groove or vascular channel. The resulting complications may include suture diastasis, venous sinus thrombosis, and epidural hematoma. stellate skull fracture is a type of linear skull fractures with multiple linear fractures radiating from the site of impact and it gives a hint about the magnitude of the trauma.

- **Depressed skull fractures**; is a type of fracture usually resulting from blunt force trauma, such as getting struck with a hammer, rock or getting kicked in the head. These types of fractures, which occur in 11% of severe head injuries, are comminuted fractures in which broken bones are displaced inward. These fractures may become critical if the depression is greater than the full thickness of the skull and requires immediate surgical elevation If less than that then it will cause contusion (no collection of blood)

In children these fractures are flexible and can elevate by themselves (Ping-Pong fracture).

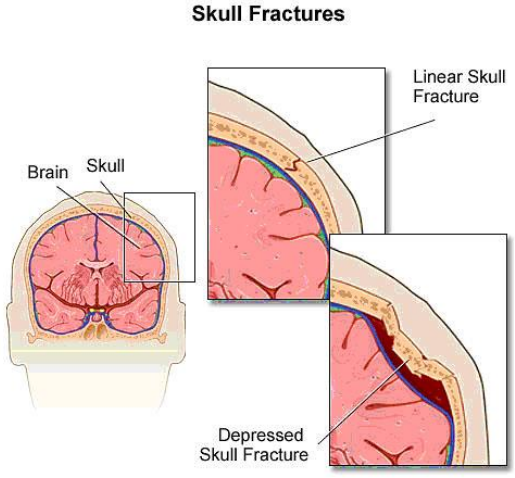


Figure 18 difference between linear and depressed fracture

- **open fractures;** these are compound depressed skull fractures that occur when there is a laceration over the fracture, resulting in the internal cranial cavity be¬ing in contact with the outside environment increas¬ing the risk of contamination and infection. This needs urgent surgery to protect against infection.

- Basal skull fractures; breaks in bones at the base of the skull, require more force to cause than that re¬quired for calvarial skeleton fractures. Thus they are rare, occurring as the only fracture in only 4% of se¬vere head injury patients, basilar fractures have characteristic signs: blood in the sinuses; leaking of cere¬brospinal fluid (CSF) from the nose (rhinorrhoea) or ears (otorrhea), raccoon eyes (bruising of the orbits of the eyes that result from blood collecting there as it leaks from the fracture site), and battle's sign (caused when blood collects behind the ears causing bruising) (figure 2). On examination of the ear, some-times there is accumulation of blood behind the tym¬panic membrane (hemotympanum).

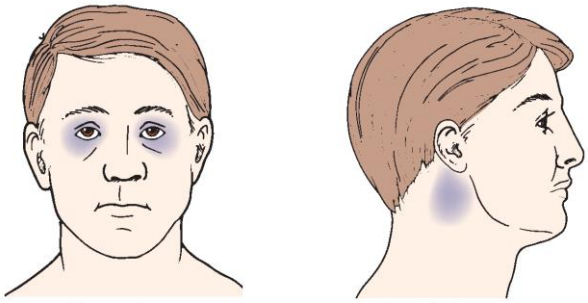


Figure 17 racoon eyes and battle sign

To identify weather the leaking CSF is mixed with blood or not, the discharge is collected on a dressing and checked for the presence of a halo sign (double ring sign), which indicates the presence of blood. A ß-transferrin test is also useful alternatively.

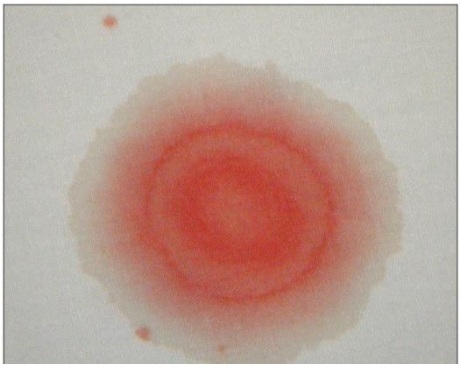


Figure 19 halo sign indicative of fracture of the base of the skull showing mixture of CSF and blood on dressing

During the management of basal skull fractures it's im¬portant to avoid inserting a nasogastric tube or nasotracheal tube as it may penetrate the delicate membrane tissue of the brain in fractures that involve the cribriform plate. An orogastirc tube or orotracheal is used instead. (Figure 4)

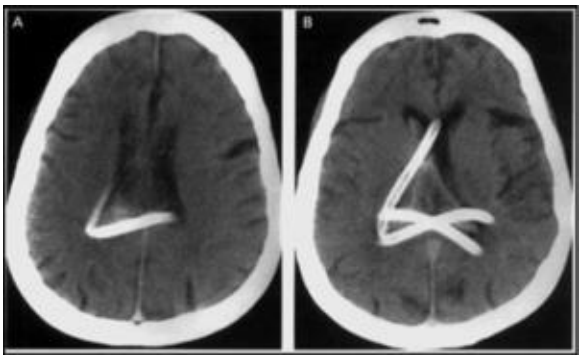


Figure 20 axial CT of a patient with Basel skull fracture show the presence of nasogastric tube in the brain, this was the result of malpractice

BRAIN INJURY

According to the mechanism of injury, brain injury can be:

- Blunt: in which the direct impact on the head may affect the brain causing damage. The injury on the side of the impact is called 'coup'. Sometimes the other side of the brain becomes injured; this is called 'countercoup'.
- Penetrating: the most important example of this type of injury is that resulting from gunshots.
- Shearing injury: it's a decortication problem where the axons are cut between white and gray matter, an important example of this is diffuse axonal injury secondary to sudden acceleration\deceleration. It can result from movements which are either horizontal or rotational.
-

According to the extent, the resultant injury is either focal or diffuse.

FOCAL BRAIN INJURIES

CONTUSION

A contusion is a bruise of the brain tissue. Like bruises in other tissues, cerebral contusion can be associated with multiple microhemorrhages. It can result from sudden deceleration or blunt injury and is not associated with the involvement of the bones and covering meninges. Most commonly it involves the temporal or frontal lobes as illustrated in figure 5. Contusions with this type of injury are coup injuries. Countercoup injury can result in in¬volvement of the occipital lobe. The symptoms and signs depend on the site and weather the lesion is focal or deep. Increased cerebrovascular permeability may result in brain edema with progressive increase in intracranial pressure. This may result in coma and if enough may cause brain herniation and death, thus it requires imme¬diate surgical intervention. Bleeding may occur late in alcoholics, and so does the signs of brain edema.

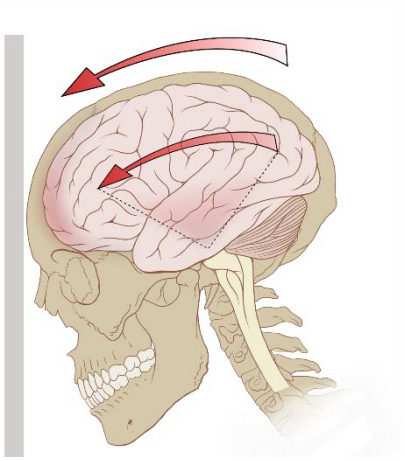


Figure 21 the brain continues to move forward and is contused be the rough surface of the middle fossa and the orbital plate

EXTRADURAL HEMATOMA

This is usually the result of a skull fracture with tearing of a meningeal artery. It is most commonly seen in the middle fossa after a temporal fracture and middle meningeal artery tear. The primary brain injury (loss of consciousness due to concussion, see below) is often minimal, with a typical 'lucid' interval followed by rapid deterioration (reflected by GCS) as the hematoma enlarges (figure 6). This can result in increased ICP and decreased CPP, and can result in brain herniation, which may compress the brain stem causing abnormal posturing, and abnormal pupil

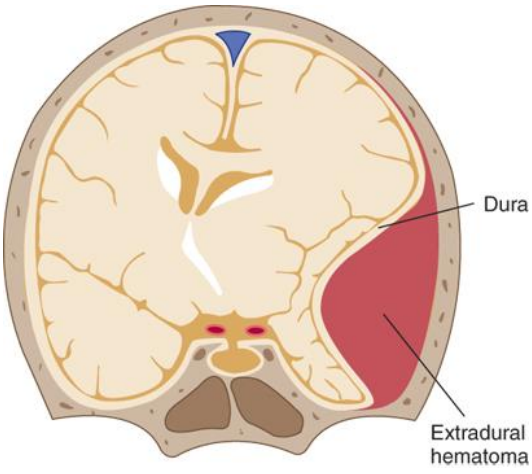


Figure 6 illustration of extradural hematoma

responses to light that may end in loss of con-sciousness. Extradural hematomas are characterized by loss of consciousness, LUCID interval, Coma (due to loss of blood supply to the RAS due to enlargement of the hematoma) , lateralization (ipsilateral dilation and contralateral paresis) , and by CT imaging it looks like a lens

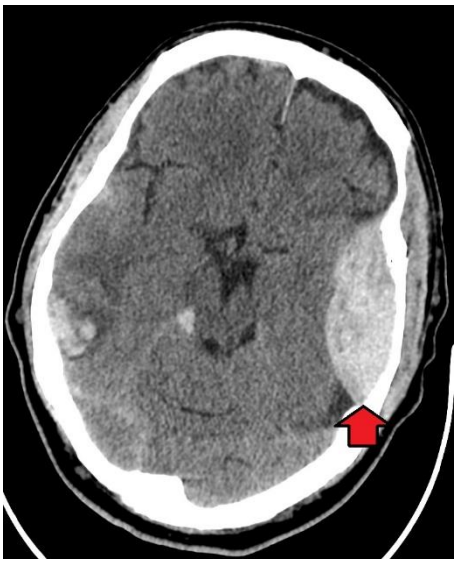


Figure 7 axial CT of an extadural hematoma caused by a fracture

SUBDURAL HEMATOMA

This is more common and more dangerous than extra¬dural hematoma and is due to laceration of vessels (espe¬cially small cerebral veins) on the brain surface, or 'bursting' of the brain. CT shows a hematoma that is con-cave on its inner surface crescent like.

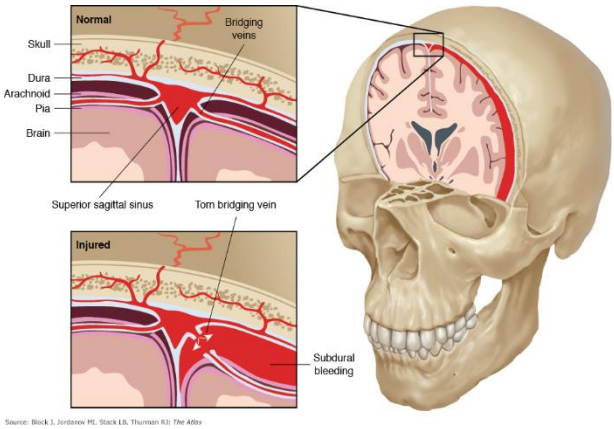


Figure 8 subdural hematoma illustration

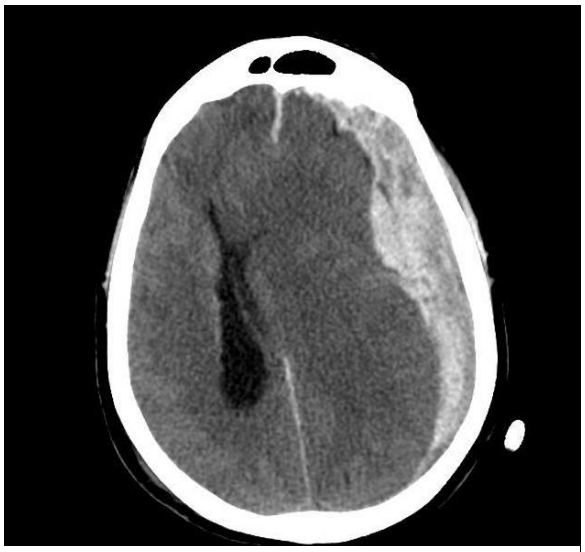


Figure 9 axial CT of a subdural hematoma

This may occur spontaneously, usually from a ruptured cerebral aneurysm, or may result from head injury. The result is hematoma into the subarachnoid space which may cause meningeal irritation with increased intracranial pressure that presents as headache and photophobia.
They are diagnosed by using CT scans (figure 10) or lumber puncture which may show bloody CSF.

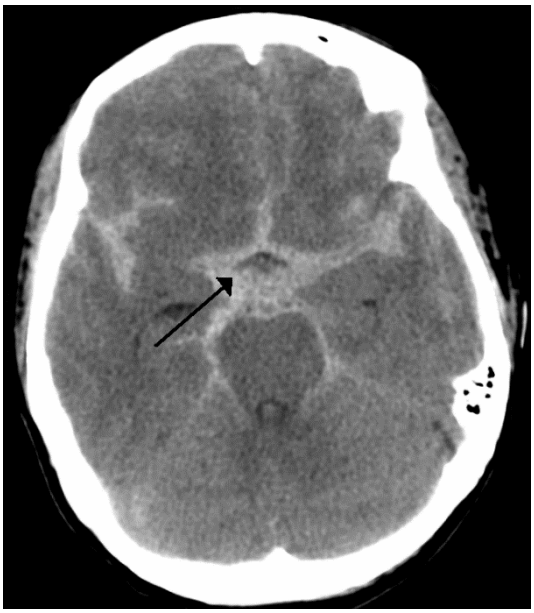


Figure 10 axial CT of subarachnoid hemor-rhage

INTRACEREBRAL HEMATOMA

These occur mainly due to contusions or rupture of a microanuerysm. They present according to the site (e.g., occipital lobe may present with vision abnormalities). Di¬agnosis is by CT scan (figure 11). They have a poor prog¬nosis and a high mortality rate.

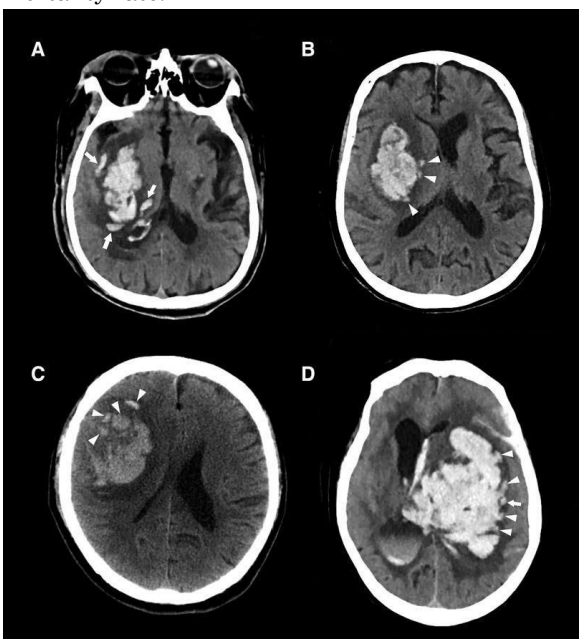


Figure 11 Examples of intracerebral hematoma

PENETRATING INJURIES

Gunshots are common examples of penetrating injuries. Patients who survive penetrating gunshots are lucky, as the survival rate from such accidents is very low. These patients require the use of antiseptics on the entrance and exit of the lesion and in cases of nonpenetrating bul-let injuries, a CT scan is required to assess whether there is a blunt injury or contusion.

DIFFUSE BRAIN INJURIES

These are injuries which can affect wide areas of the brain. 2 common examples are concussions and diffuse axonal injury.

CONCUSSIONS

A concussion is temporary neuronal dysfunction after nonpenetrating head trauma. The head CT is normal, and deficits resolve over minutes to hours. Patients may suf¬fer from amnesia, headache, nausea, vomiting, and may reveal a history of loss of consciousness. Concussion is the mildest injury and it's just depolarization and repolarization of the neurons and according to the severity it's between seconds to minuets and the amnesia is either anterograde or retrograde .
In emergency medicine, a lucid interval is a temporary improvement in a patient's condition after a traumatic brain injury, after which the condition deteriorates.
So the patient must be admitted he could be in the lucid interval

Diffuse axonal injury is caused by damage to axons throughout the brain. The cause of this type of injury is sudden acceleration\deceleration (figure 12) or rota¬tional movement. The brain is a mass that doesn't have a homogenous density, for example when there is a sudden acceleration\deceleration the grey matter may move slower according to its density, while the white matter may move faster, this can result in grey matter- white matter avulsion. Axons may be completely disrupted and then retract, forming axon balls. This form of injury is called

decortication. Small haemorrhages can be seen in more severe cases, especially on MRI (15 mm white grey- white junction).

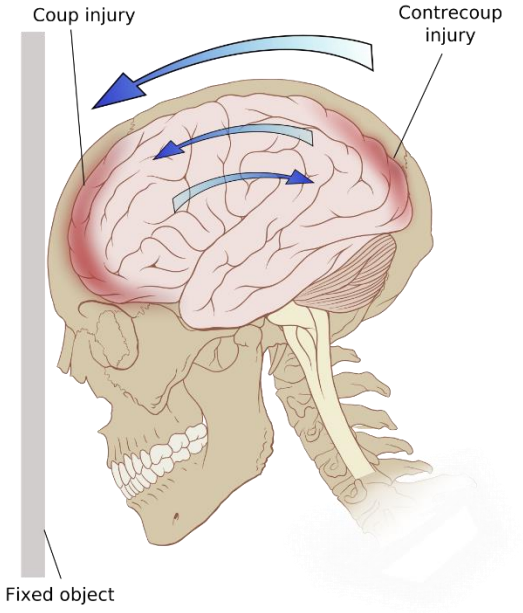


Figure 12 the skull accelerates rapidly, then deceleration due to impact against hard surface

Patients with decortications may have what is referred to as decorticate posturing where by the patient present with the arms flexed, or bent inward on the chest, the: hands are clenched into fists, and the legs extended and feet turned inward.

Another form of diffuse brain injury is called decerebration, caused mainly by rotational movement, and is associated with detachment of the brain stem from cerebrum. Patients may have decerebrate posturing, where by the head is arched back, the arms are extended by the sides, and the legs are extended.

Patients with diffuse axonal injury may suffer from deep coma, hypotension and high fever. This type of injury rarely kills. However, it's the most common cause associated with vegetative death, accounting for 90% of cases.

ASSESSMENT

GLASGOW COMA SCORE

The GCS is a measure of conscious level that has greatly facilitated the classification and objective management of head-injured patients. It is used internationally and records the best verbal response, best motor response and eye opening. The maximum score is 15 and the minimum is 3. Change in GCS over time is much more informative than an absolute reading at any one point in time. According to this, Coma means no eye opening, no response to verbal commands and no word verbalization, and its score is 8 or less. Deterioration in the level of consciousness is reflected by a fall in the points of Glasgow coma score. Generally, one-point fall is considered significant while 3 points fall is considered dangerous.

Glasgow coma score	
Eye opening	
• **Spontaneous**	**4**
• **To verbal command**	**3**
• **To pain**	**2**
• **No response**	**1**
Motor response	
• **To verbal command**	**6**
• **Localizes pain**	**5**
• **Flexion withdrawal**	**4**
• **Abnormal flexion**	**3**
• **Extension**	**2**
• **No response**	**1**
Verbal response	
• **Oriented**	**5**
• **Disoriented**	**4**
• **Inappropriate words**	**3**
• **Incomprehensible sounds**	**2**
• **No response**	**1**
The least equals 3 and the most equals 15	

NEUROLOGICAL EXAMINATION

This should routinely include an assessment of pupil size and reaction (a 1mm difference is significant, and wither it's a risky movement or not); a search for CSF leaks from nose, mouth and ears; a survey of the scalp for penetrating injuries; signs of a basal skull fracture (Battle's sign, raccoon eyes); and an assessment of the maxillofacial skeleton. Peripheral neurological examination will give a guide to focal brain injury, spinal injury or peripheral nerve injury.

MANAGEMENT

As with all injured patients, management commences with airway, breathing and circulation. The management of systemic complications such as severe chest injury, intra-abdominal haemorrhage or major volume loss is a priority in treatment, since these phenomena will lead to secondary cerebral ischemia and hypoxia and thus secondary brain damage. The GCS should be documented on arrival and following resuscitation; and the findings of a neurological survey recorded.

PRIMARY SURVEY

A FOR AIRWAY AND B FOR BREATHING

The neck should be immobilized (use a rigid collar) until a cervical spine injury has been excluded. Airway blockage in patients with head injury is mainly caused by protrusion of the tongue. In general, patients with a GCS of 8 or less are intubated and ventilated. This is to prevent hypoxia and aspiration pneumonitis, and to allow hyperventilation, which reduces the PoCO2 (maintain at 35-40 mmHg) and so lowers ICP through cerebral vasoconstriction. While the patient is on the ventilator, the PaCo2 is monitored by a capnograph. You should monitor Sao2 using a pulse oximetry.

C FOR CIRCULATION

Patients with head injury often have extracranial injury. It is important to remember that head injury alone never causes hypovolemic shock. A bleeding site is searched and stopped immediately. While installing a wide bore cannula (or central line) blood is withdrawn for urea and electrolytes, correction with intravenous fluids should not include hypo-osmolar fluids like dextrose 5%, as these can aggravate brain edema. Colloid fluids should be avoided as any injured brain tissue can accumulate colloid particles and also aggravate brain edema. In the case of head injury ringers lactate is preferable. You should avoid hyperglycaemia and over hydration. Blood pressure and pulse rate along with urine output (after catheterization) should be monitored.

D FOR DISABILITY

Disability in head injury is assessed by Glasgow coma score.

E FOR EXPOSURE

Expose the patient carefully, looking for other injuries. Avoid moving the neck until a cervical spine injury is excluded.

SECONDARY SURVEY

INTRACRANIAL PRESSURE

The most important thing to remember is maintenance of ICP. In many cases, an ICP monitor is inserted for postoperative or elective ICP monitoring (normally less than 25). Causes of increased intracranial pressure in head injuries are masses, brain edema, seizures, or hypoxia.

- **Masses**; Following resuscitation, stabilization and prioritization of injuries, a head CT scan is performed. This will visualize intracranial hematoma, brain contusions, depressed bone fragments, intracranial air and associated maxillofacial fractures. Mass lesions such as extradural hematoma, subdural hematoma and hemorrhagic contusions may cause brain swelling and shift, and are often surgically evacuated. Indications for clot evacuation are > 5 mm midline shift, significant impairment of GCS, or protracted headache or vomiting.
- **Brain edema**; Hyperventilation, mannitol (or frusemide) and barbiturates are used to reduce ICP, and the systemic blood pressure may be raised using fluids and inotropes (to increase arterial blood pressure and thus avoid drops in cerebral perfusion pressure).

- **Seizures**; Seizures can cause episodes of hypoxia that may result in brain edema and increased blood pressure thus must be treated using valium or phenytoin. If seizures persist, general anaesthesia is an option
- **Hypoxia**; hypoxia can cause cerebral vasodilatation and brain edema. The patient is usually restless and is given a sedative (avoid opiates, use chlorpromazine, but be aware of hypotension). Barbiturates are used to decrease brain catabolism and brain oxygen demand. Always avoid hyperthermia, as it may increase the catabolic state of the brain, and thus increase brain oxygen demand. On the other extreme, also avoid hypothermia.

FRACTURES

Compound cranial wounds need to be surgically explored, dead tissue and foreign bodies removed, depressed bone fragments elevated, haemostasis secured and the Dura closed in a watertight fashion. Depending of the age of the wound, bone fragments may be either cleaned and replaced or discard

MAXILLOFACIAL INJURIES

It is the responsibility of the physician to restore appearance and the function to the best of his ability. Facial injury may distort the appearance of the patient which Is an important part of his/her well-being, thus emphasis should be made on preserving the appearance in accordance with maximal function. **maxillofa**cial injuries are the spectrum of injuries affecting the soft tissue and bone of the face.

AETIOLOGY

The most common cause of maxillofacial injuries is road traffic accidents (RTAs). Quarrels represent a significant share of the causes. Other causes include home accidents (most prominently dog bites), industrial accidents and missile injuries in military conflicts.

INITIAL MANAGEMENT

The timing of initiating management is As soon as possible and should be Consistent with the general condition of the patient. Early skilful management decreases the possibility of Permanent disfigurement. However, they are rarely an acute surgical emergency. Bleeding should be controlled in status of Impending shock when the patient is apparently losing a significant amount of blood. Treatment of multiple systemic injuries has a priority over this type of injuries as the later are more life threatening. Unstable patients with fractures in the face can have a period of 1-3 weeks to be treated to have approximately the same outcome.

AIRWAY

The mouth and nasal passages form part of the upper aero digestive tract, and lacerations and fractures of the facial skeleton may give rise to immediate or delayed respiratory obstruction. Immediate obstruction may arise from inhalation of tooth fragments, accumulation of blood and secretions, and loss of control of the tongue in the unconscious or semiconscious patient. To avoid this, the patient should always be nursed in the semi prone position (Figure 1) with the head supported on the bent arm, and never lying on their back. Damaged teeth, blood and secretions can then fall out of the mouth, and gravity pulls the tongue, forward. As the patient is maneuvered into the correct nursing position, the neck should be supported and held in a neutral position - a protective collar is advisable until a fracture of the cervical spine has been excluded. An intracranial injury should always be considered as a possibility, however minor the injury to the face. Because edema is common to all facial injuries occurring within an hour of its onset, its risk on compromising the airway is significant and merits the use of an oropharyngeal airway as a prophylactic measure. If this is not done, an emergency tracheostomy may have to be undertaken later with potential risk to the patient. Other indications for tracheostomy are shown in the table below.

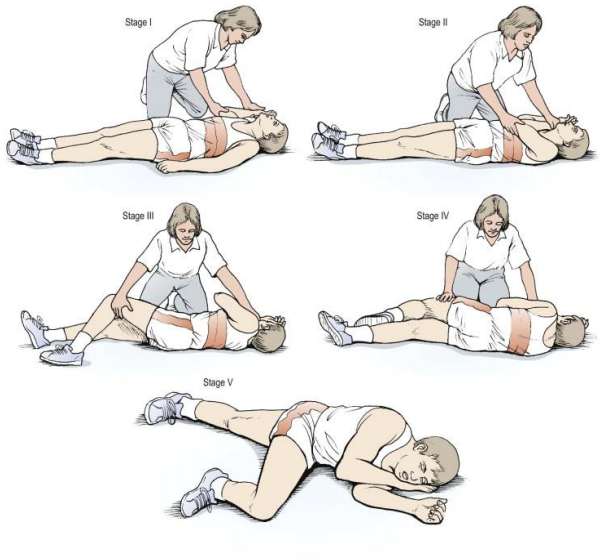


Figure 22 The patient should be nursed in to semiprone postion to allow secreations, blood and foreign bodies to fall from th mouth

Indications for tracheostomy in maxillofacial injuries

- **Unrelievable obstruction in the larynx or pharynx**
- **Intracranial injuries**
- **Chest or high cervical spine injuries**
- **Possibility of prolonged post-operative airway problem**

HEMORRHAGE

Initial hemorrhage after a facial injury can be dramatic. Sustained bleeding is unusual, but

emergency measures to stabilize the facial fractures and control bleeding may be required. This involves local pressure with a dressing, clamping or ligation of the spurting arteries, packing the nasal cavity with gauze or haemostat material and approximation of the wound edges as possible. The need for transfusion is given as indicated.

CLASSIFICATION OF MAXILLOFACIAL INJURIES

According to the type of tissue involved, maxillofacial injuries can be classified into those that affect the soft tissue alone, those that affect both soft tissue and bone and those affecting bone alone.

Identification of the bone affection, namely fracture, can alter the management dramatically and thus is of utmost importance. The examination is an important tool for identification of possible fractures. Any fracture is suggested by the presence of tenderness and edema with loss of function (or painful function) on its site. The presence of a deformity and felt crepitation are also suggestive. Ecchymosis, abrasion, and contusions overlying the affected site are also good indicators. Fractures of the zygomatic bone are suggested by frequent subconjunctival hemorrhage, which will often be found to have no posterior limit when the patient is asked to look to the other side (figure 2).

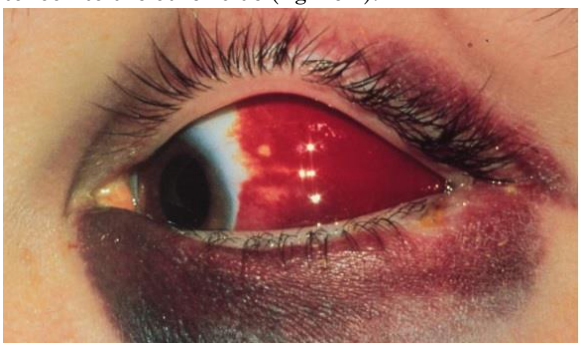


Figure 23 Fractures of the zygoma may often be associated with subconjunctival hemorrhage. This example shows no posterior border to the hemorrhage as the patient looks away from the side of the fracture.

Signs indicating a mandibular fracture include the presence of ecchymosis on the sides of the mandible with loss of function and malocclusion or an open bite deformity.

The use of X-rays is an important diagnostic tool. X- rays are taken fully with every possible position that can give better visualization of each bone. the use of coronal section CT scan with multisite reconstruction determines the course of management.

Other considerations taken into account when initially managing maxillofacial injuries are shown in the table below.

important considerations when managing maxillofacial injuries

- **Prophylaxis against Tetanus**
- **Exploration of the wounds to identify extension to cranium, orbit, salivary glands or ducts, or facial nerve**
- **Good blood supply permits delayed closure**
- **Fixation of bones through Present wounds**
- **Removal of foreign bodies**
- **Photograph for Legal purposes**

FRACTURES OF THE MANDIBLE

The mandible is the most commonly injured facial bone. in general, it is a strong bone, but has certain weak points and is thin at the angles and the mental foramens. The condylar neck is the weakest part of the mandible and is the most frequent site of fracture. Like all fractures. Mandibular fractures are classified as (1) simple (with no break in overlying skin), (2) compound with overlying soft tissue injury and (3) comminuted (multiple fragments of bone).

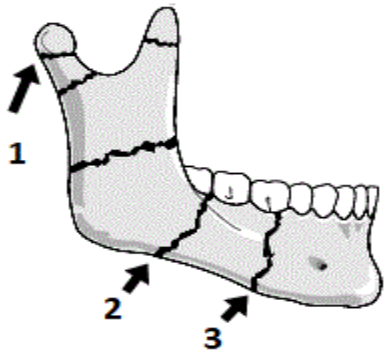


Figure 24 The patterns of fracture of the mandible. (1) The neck of the condyle is the most common site, followed by (2) the angle of the mandible through the last tooth. (3) The third point of weakness is in the region of the canine tooth.

AETIOLOGY

The mandible may fracture directly at the point of the blow, or indirectly where the force from the blow is transmitted and the mandible fractures at a point of weakness distant from the original blow. Mandibular fractures are most commonly encountered in military and gunshot injuries. Multiple fractures are seen in crush injuries.

CLINICAL FEATURES AND DIAGNOSIS

Signs of fracture including tenderness, edema, ecchymosis, loss of function (or painful function), crepitation

and deformity are usually present. Mobility might be felt at the site of fracture. Excessive salivation is seen with involvement of the salivary gland. Malocclusion is seen best in dentulous jaw.

The diagnosis can be made by X-ray findings using panoramic view.

MANAGEMENT

Treatment is aimed at correcting the deranged dental occlusion. fractures can h treated with intermaxillary fixation, where the mandible and maxilla are positioned in appropriate occlusion and are wired together with arch bars or screws. Open reduction is designated for those with condylar dislocation, severe malalignment, or loss of mandible height. Displaced fractures of the angle, body, and symphysis are treated via open reduction.

FRACTURES OF THE MAXILLA

Maxillary fractures involve the maxilla as well as the entire midfacial domain. They are less common than mandibular fractures and are usually caused by direct blow to the head.

CLASSIFICATION

Mild fractures may involve only the alveolar process of the maxilla (alveolar). This is seen due to direct blows or blows to the mandible that are transmitted to the alveolar process of the maxilla. The LeFort classification system is commonly administered to describe and qualify more severe and complex fractures (figure 4). Le Fort I fracture (horizontal) may result from a force of injury directed low on the maxillary alveolar rim in a downward direction. The fracture travels horizontally above the teeth apices, crosses below the zygomaticomaxillary junction. Le Fort II fractures (pyramidal) may result from a blow to the upper maxilla and usually involve the inferior orbital rim. Such a fracture has a pyramidal shape and extends from the nasal bridge at or below the nasofrontal suture and inferiorly through the anterior wall of the maxillary sinus; it then travels under the zygoma, across the pterygomaxillary fissure, and through the pterygoid plates. Le Fort III fractures (transverse) involve the zygomatic arch including the whole maxilla, which is pushed downwards and backwards. These may follow impact to the nasal bridge or upper maxilla. Vertical fractures of the maxilla are rarely seen.

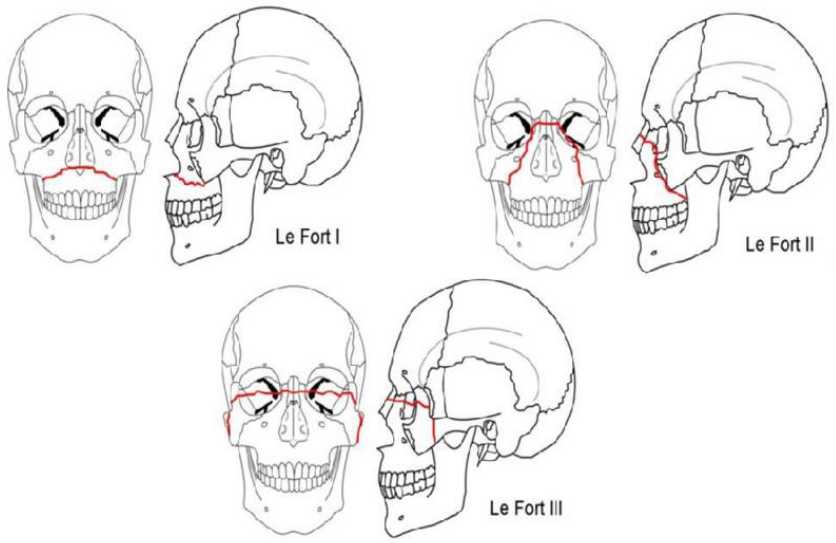


Figure 25 le fort classification of maxillofacial injuries

CLINICAL FEATURES AND DIAGNOSIS

Alveolar fractures may present with the classic signs of fracture. In le fort I fractures, The lower fragment is very mobile and dental occlusion is affected, giving an open bite deformity. Epistaxis and rhinorrhoea may occur in le fort II and periorbital bruising and subconjunctival hemorrhage are also evident. With le fort II malocclusion is present with an anterior open bite. In le fort III fractures, There is extensive facial edema (long donkey appearance) and periorbital ecchymoses with subconjunctival hemorrhage. Involvement of the cribriform frontal sinuses in the line of the fracture gives rise to escape of CSF as rhinorrhea. Tearing of the soft tissues of the mouth is seen due to traction by the dismantled bones.

In le fort II fractures, a Step deformity at infraorbital margin is felt. Mobility of the maxilla can be elicited by palpation in extensive fractures. careful x-ray studies are necessary to determine the extent and complexity of the midfacial fracture. In many instances, a fractured maxilla may be difficult to see due to superimposition of other structures.

MANAGEMENT

Emergency management involves controlling the airway and hemorrhage as explained earlier. definitive treatment depends on the type of fracture. Alveolar fractures are treated by manipulation, reduction and fixation. intermaxillary fixation or open reduction is used for le fort fractures.

Post-operatively, mouth hygiene should be taken care of with frequent mouth washing and proper tooth brushing. Regards to proper nutrition and antibiotics is necessary.

COMPLICATIONS

Hemorrhage and airway obstruction are early complications of maxillary injuries.

Non-union is rarely seen and is mostly due to a comminuted fracture. Malunion is seen due to a delay of diagnosis. Injury to the infraorbital region and nerve can result in its anesthesia. Lacrimal bone injury can result in obstruction of the lacrimal system and epiphora.

Infection is seen due to contamination of the wound, penetrating injury that involves the maxillary sinus, the presence of foreign bodies, loose tooth, presence of rhinorrhoea and otorrhea. Treatment is with antibiotic coverage and avoidance of nose blowing and nasal packing.

CHEST TRAUMA

Thoracic injury (or cardiothoracic trauma) accounts for 25% of all injuries. 20-25% of trauma death are due to the thoracic injuries. Two thirds of those die after reaching the hospital. In most of these patients, the cause of death is hemorrhage (causing hypovolemia and hypoxia). According to the mechanism chest injuries are either blunt or penetrating. The table below presents chest injury according to the thoracic structure involved.

Types of chest injury
Chest wall injury • **rib fractures** • **flail chest (3 ribs or more)** • **sternal fracture** • **clavicular fracture** • **scapular fracture**
Pulmonary injuries • **contusion,** • **pneumothorax,** • **hemothorax**
Tracheobronchial injuries
Associated injuries • **Cardiac and great vessel injuries,** • **Rupture of the diaphragm,** • **Esophageal injury**

BLUNT INJURIES

Blunt injuries can be caused by a direct blow to the chest, sudden deceleration, or compression and is evident after road traffic accidents (accounts for 70-80% of thoracic injuries) and crushing after falling. Rib fracture (especially at the angle) is the most common sign of blunt thoracic trauma; however, injury caused by massive forces can involve the sternum, scapula, or the first rib.

PENETRATING INJURIES

Penetrating injuries are either caused by sharp objects (e.g. stab wounds) or missile injuries (e.g. gunshots). They are very complex and can involve more than one structure, but most commonly, involve the lungs. Involvement of the lung in chest injuries most certainly implies involvement of the pleura and thus is always associated with hemothorax or pneumothorax. Penetrating injuries can involve the heart, great vessels, the diaphragm or/and the esophagus.

ASSESSMENT OF CHEST INJURIES

Resuscitation of all injuries to the chest should follow traditional ATLS principles shown in the table below.

ATLS principles of resuscitation
• **A Airway** • **B Breathing** • **C Circulation** • **D Disability (neurology)** • **E Environment and Exposure**

• A for Airway patency; airways could be occluded by a foreign body or by the tongue in cases there is involvement of the central nervous system (e.g. shock). All airway manipulations must be performed with the control of the cervical spine and ventilation.

• B for Breathing; check for respiratory movements and feel for breathing, administer Oxygen and install a pulse oximeter, if the patient is intubated, end tidal CO2 is checked.

• C for Circulation; An external source of bleeding is searched at first. If apparent, an attempt to stop it is taken immediately. check Patient's pulses, and blood pressure; Radial pulse may be absent if the pressure is less than 70. If there is any sign of internal bleeding (absent or rapid pulses, hypotension, pallor, mental deterioration) then site of bleeding is determined and the patient is treated as being shocked. See whether neck veins are flat (in shock), or distended (in cardiac tamponade).

• D for Disability; you should identify whether the patient has lost consciousness. Loss of consciousness secondary to thoracic injury is indicative of shock and should be treated as so.

• E for Exposure; you should expose the patient to identify whether there are signs of injuries elsewhere.

CLINICAL EXAMINATION

After resuscitation had been instituted, clinical examination should be commenced to assess the injury and involves;

- **look**; Determine the respiratory rate and depth and Look for chest wall asymmetry. Paradoxical chest wall motion may indicate flail chest. Look for bruising, seat belt or steering wheel marks, or penetrating wounds.
- **Feel**; Feel the trachea for deviation, which may indicate mediastinal shift secondary to pneumothorax. Assess whether there is adequate and equal chest wall movements. Feel for chest wall tenderness or rib 'crunching' indicating rib fractures. Feel for subcutaneous emphysema which may give rise to crackling sensation over gas containing tissue.
- **Percuss**; Percuss both sides of the chest looking for dullness or resonance (more difficult to appreciate in the trauma room). Hyper-resonance is heard in pneumothorax while dullness is heard in massive hemothorax.
- **listen**; Listen for normal, equal breath sounds on both sides, especially in the apices and the axillae and at the back of the chest (or as far as you can get while supine).

INVESTIGATIONS

Investigations are more sensitive in the assessment of chest injury in the stable patients. In the unstable patient, chest radiography is the investigation of first choice, provided that it does not interfere with resuscitation. Ultrasound can be used to differentiate between contusion and the actual presence of blood. A chest tube can be a diagnostic procedure as well as a therapeutic one, and the benefits of insertion often outweigh the risks. The CT scan, especially using the newer multislice scanning technology, has become the principal and most reliable investigation for major injury in thoracic trauma. It allows scan of the organs and the skeleton and easy access their involvement by 3-dimentional reconstruction. In blunt chest trauma the CT scan will allow the definition of rib and vertebral fractures, as well as showing hematomas, pneumothoraxes and pulmonary contusion. In penetrating trauma, the scan may show the track of the missile and allow the planning of definitive surgery.

EMERGENCY THORACOTOMY

Emergency thoracic surgery is an essential part of the armamentarium of any surgeon dealing with major trauma. A timely thoracotomy for the correct indications can be the key step in saving an injured patient's life, thoracotomy can be broadly classified into emergency thoracotomy or planned (urgent) thoracotomy. Indications for thoracotomy are shown in the table below.

Indications for thoracotomy
Emergent • **Acute cardiac tamponade unresponsive to cardiac massage** • **Intra thoracic exsanguinating hemorrhage** • **Intra-abdominal hemorrhage requiring aortic cross clamping** • **Need for internal cardiac massage**
Urgent • **Chest drainage more than 250 ml/h (for 2-4 hours)** • **Chest drainage more than 1500cc upon insertion** • **Progressive opacification of chest X-ray** • **unevaluated clotted large hemothorax** • **Chest wall defect** • **Developing cardiac tamponade** • **Massive air leak despite adequate drainage** • **Proven great vessel injury on angiography** • **Proven esophageal injury** • **Traumatic injury of the heart valves or heart septum** • **Proven diaphragmatic laceration**

IMMEDIATE LIFE-THREATENING INJURIES (PRIMARY SURVEY)

Conditions that occur due to chest injury, which are immediately life threatening are shown in the table below.

Primary survey of chest trauma
• **Airway obstruction** • **Open pneumothorax** • **Tension Pneumothorax** • **Massive hemothorax** • **Pericardial tamponade** • **Rib fracture** • **Flail chest**

OPEN PNEUMOTHORAX (SUCKING CHEST WOUND)

This is due to a large open defect in the chest (> 3 cm) following a penetrating injury, leading to equilibration between intra thoracic and atmospheric pressure. Air accumulates in the hemithorax (rather than in the lung) with each inspiration, leading to profound hypoventilation on the affected side and hypoxia. Breathing is also impaired in the other lung, because the pleural pressure on the healthy side falls on inspiration and as a result the mediastinum is displaced to the healthy side. On expiration the pressure rises and the mediastinum moves toward the collapsed side, this is called mediastinal flutter (figure 1). There is usually decreased air entry and hyper-resonance of the affected side on examination.

Initial management consists of promptly closing the defect with a sterile occlusive plastic dressing, taped on three sides to act as a flutter-type valve. A chest tube is inserted as soon as possible in a site remote from the injury site. Normal inflation will take place while the patient breaths spontaneously, if not, intubation and positive pressure ventilation is used.

TENSION PNEUMOTHORAX

A tension pneumothorax develops when a 'one-way valve' air leak occurs either from the lung or through the chest wall. Air is forced into the thoracic cavity without any means of escape, completely collapsing the affected lung. The mediastinum is displaced to the opposite side, decreasing venous return and compressing the opposite lung.

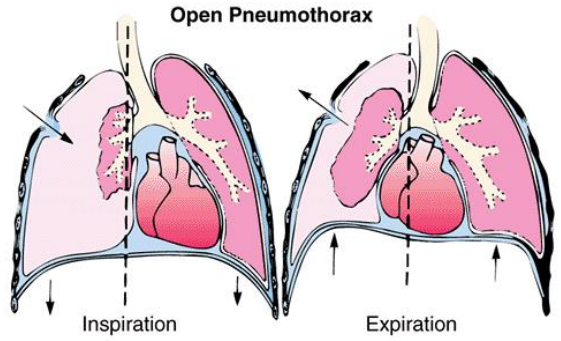


Figure 26 illustration of open pneumothorax

The most common causes are penetrating chest trauma, blunt chest trauma with parenchymal lung injury and air leak that did not spontaneously close. The clinical presentation is acute. The patient have tachypnea, dyspnea and distended neck veins (similar to pericardial tamponade). Clinical examination can reveal widening of the intercostal spaces at the affected side, tracheal deviation (a late finding - not necessary to clinically confirm diagnosis), hyperresonance and absent breath sounds over the affected hemithorax. It is confirmed by a chest x-ray which may show a collapsed lung, depression of the ipsilateral dome of the diaphragm and deviation of the mediastinum to the contralateral side (figure 3).

Treatment consists of immediate decompression and is managed initially by rapid insertion of a large-bore hypodermic needle into the second intercostal space in the mid-clavicular line of the affected hemithorax. This is immediately followed by followed by insertion of a chest tube through the fifth intercostal space (basal drain) in the anterior axillary line thoracostomy.

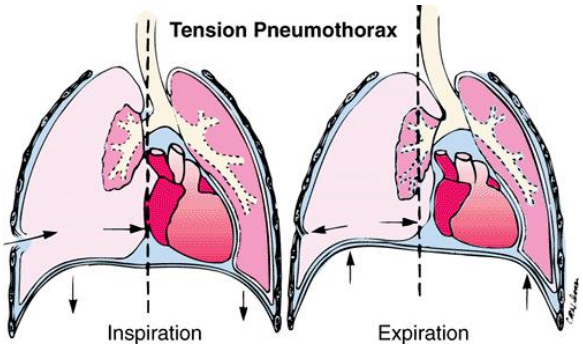


Figure 27 illustration of tension pneumothorax

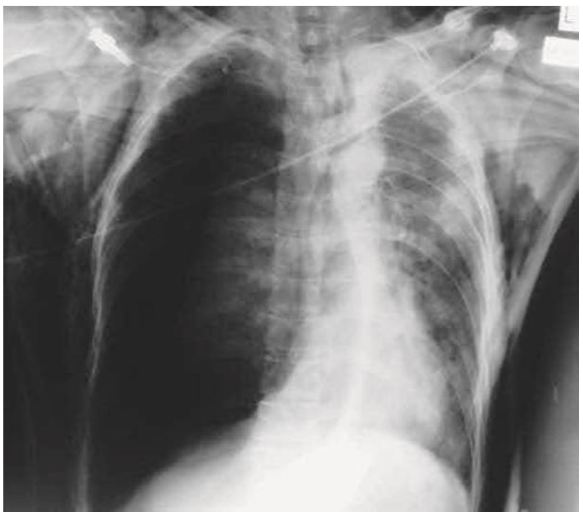


Figure 28 chest x-ray of a man with right tension pneumothorax. It show's depressed diaphragmatic dome with lung collapse and mediastinal shift.

MASSIVE HEMOTHORAX

The most common cause of massive hemothorax in blunt injury is continuing bleeding from intercostal vessels or occasionally the internal mammary artery. other sources of bleeding are shown in the table below.

Sources of bleeding in massive hemothorax
• **Intercostal vessels** • **Internal mammary artery** • **Pulmonary parenchymal injuries** • **Major pulmonary vessels** • **Injury to the heart or great vessels**

Accumulation of blood in a hemothorax can significantly compromise respiratory efforts by compressing the lung and preventing adequate ventilation. Such massive accumulation of blood presents as hemorrhagic shock with flat neck veins, unilateral absence of breath sounds and dullness to percussion. Sometimes the condition is diagnosed clinically but a chest x-ray may be required to confirm the diagnosis. Provisional diagnosis is achieved by CT scan (assessment of size).

The treatment consists of correcting the hypovolaemic shock, insertion of an intercostal drain and in some cases intubation. Blood in the pleural space should be removed completely and rapidly as possible to prevent on-going bleeding. Initial drainage of more than 1500 ml of blood or on-going haemorrhage of more than 200 ml per hour over 2-4 hours is generally considered an indication for urgent thoracotomy, thoracotomy is also indicated when there is progressive opacification on chest X-ray that is consistent with findings of progressive hemothorax on examination.

PERICARDIAL TAMPONADE

Pericardial tamponade most commonly result from penetrating trauma. Accumulation of a relatively small amount of blood into the non-distensible pericardial sac can produce physiological obstruction of the heart.

Classically the presentation consists of venous pressure elevation (as the hemopericardium prevents diastolic filling), decline in arterial pressure (as the preload decreases so does the cardiac output) with tachycardia (reflex tachycardia), and muffled heart sounds. this is known as beck's triad. Pericardial tamponade may be associated with pulsus paradoxus. It is important to note that becks triad is also present in tension pneumothorax and thus must be differentiated from which. Clinical differentiation is possible but investigations are required. Generally, All patients with shock and penetrating injury anywhere near the heart must be considered to have cardiac injury until proven otherwise. A high index of suspicion and further diagnostic investigations (e.g. chest radiography showing an enlarged heart shadow or a cardiac echo showing fluid in the pericardial sac, and insertion of a central line with a rising central venous pressure) are required for the subclinical case. For stable patients, echocardiography and pericardiocentesis are feasible. Needle pericardiocentesis (figure 4) may allow the aspiration of a few millilitres of blood, and this, along with rapid volume resuscitation to increase preload, can buy enough time to move to the operating room. Subxiphoid pericardiotomy means opening a window below the xiphoid process from which blood can drained. Its considered as a mean for diagnosis and management. The correct immediate treatment of tamponade is operative (sternotomy or left thoracotomy), with repair of the heart in the operating theatre if time allows or otherwise in the emergency room.

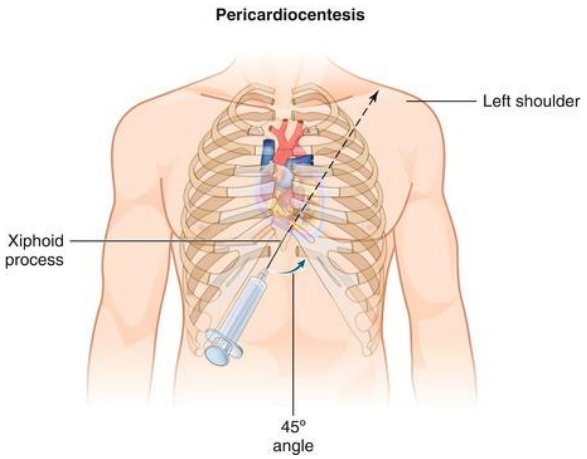


Figure 29 Needle pericardiocentesis

CARDIAC STAB WOUNDS

Penetrating injuries presented by cardiac stab wounds can cause pericardial tamponade. The Right side of the heart is more commonly injured but the left side, Atria, inflow and outflow tracts may also be damaged, generally, Patients with right ventricular wound is more likely to survive than those with left sided injury. Patients commonly present with signs and symptoms pf pericardial tamponade. Initial Treatment consists of resuscitation and pericardiocentesis. Definitive treatment involves accessing the wound through a median sternotomy, where by the wound is fixed directly without the need of a cardiopulmonary bypass using Teflon-pledgeted prolene sutures.

RIB FRACTURES

Isolated rib fractures are the most common form of chest injury (35-40%) and most commonly result from blunt injury to the chest. The site of fracture is usually tender with presence of ecchymosis and crepitus over the fractured site. Patients may present with signs and symptoms of pneumothorax (injury to parietal -pleura) or hemothorax (injury to the internal mammary or the intercostal vessels). Sometimes pain associated with rib fracture can cause respiratory splinting, resulting in atelectasis and pneumonia. The diagnosis can be made on clinical ground; however, a chest x-ray can confirm the diagnosis or detect any underlying pneumothorax, hemothorax, or pneumonia. In elderly with 1st and 2nd or 9-12 fractured ribs should be admitted. 8-12 fractured ribs increase the suspicion of associated abdominal (splenic , hepatica) injuries.

Treatment is conservative and involves rest With pain killers, or the use of nerve block to prevent respiratory splinting caused by pain with adequate ventilation . Epidural analgesia is recently used for this condition.

The prognosis depend on the number of ribs injured, age (below 5 year age have elastic chest wall that may cause lung contusion without being fractured; the wall is "compressible". Elderly with 3 or more fractured ribs tends to have 5 fold increase in mortality rate and a 4 folds increased incidence of pneumonia) and underlying pulmonary status.

FLAIL CHEST

A flail chest occurs when a segment of the chest wall does not have bony continuity with the rest of the thoracic cage. This condition usually results from blunt trauma associated with multiple rib fractures, i.e. three or more ribs fractured in two or more places (figure 5- B). sometimes it may result from a severe blow to the sternum causing multiple

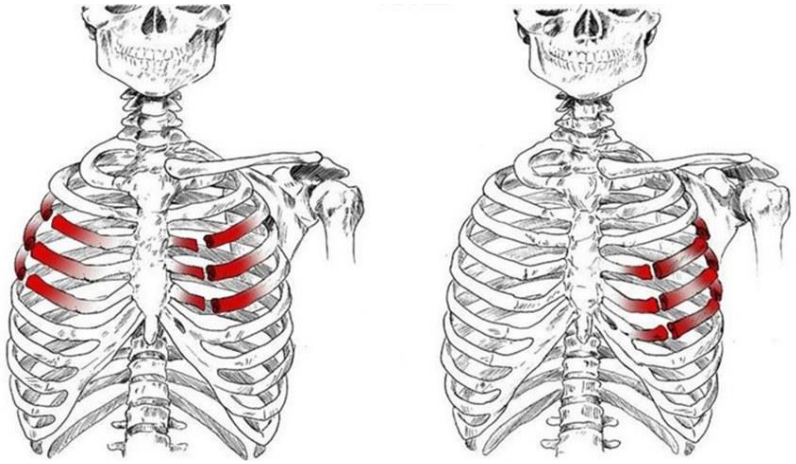


Figure 30Flail chest. (A) A severe blow to the sternum (arrowed) may cause multiple bilateral costochondral fractures, resulting in a flail. (B) chest A direct blow (arrowed) that fractures several ribs at two points will result in a flail segment.

costochondral fractures (figure 5-A). The diagnosis is made clinically, not by radiography. On inspiration the loose segment of the chest

wall is displaced inwards and less air therefore moves into the lungs. To confirm the diagnosis the chest wall can be observed for paradoxical motion of a chest wall segment for several respiratory cycles and during coughing. These patients, along with having lung contusions, are at high risk of developing tension pneumothorax, hemothorax or pneumonia. treatment consists of mechanical ventilation to 'internally splint' the chest until fibrous union of the broken ribs Occurs. Surgery involves internal fixation of the affected segment.

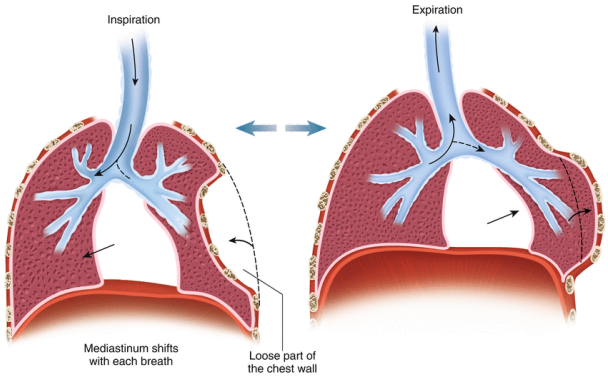


Figure 31 paradoxical movement in flail chest

POTENTIALLY LIFE-THREATENING CONDITIONS (SECONDARY SURVEY)

Conditions associated with chest trauma, which may or may not be life threatening are shown in the table below.

Secondary survey in chest trauma
• **Pulmonary contusion** • **Myocardial contusion** • **Aortic disruption** • **Traumatic diaphragmatic hernia** • **Tracheobronchial disruption** • **esophageal disruption**

ESOPHAGEAL RUPTURE

This is rare and is mainly caused by penetrating injuries. The concern with esophageal rupture is the development of mediastinitis and septic shock secondary to invasion of bacteria from the esophagus into the mediastinum. Dissection of air from the esophagus through the soft tissues can cause subcutaneous (surgical) emphysema, especially when the cervical esophagus is involved. after mediastinitis, pleural effusion and pneumothorax may develop, causing pnuemohydrothorax.

The patients may present with shock, dyspnea, and cyanosis. History may reveal the presence of sudden severe epigastric, chest, or lower back pain. On examination subcutaneous emphysema may be evident which may give rise to crackling sensation over gas containing tissue. Treatment involves resuscitation (the patient should be kept NPO), antibiotics, treatment of pneumothorax and surgical closure.

DIAPHRAGMATIC INJURIES

Any penetrating injury to or below the fifth intercostal space should raise the suspicion of diaphragmatic penetration and, therefore, injury to abdominal contents. It's often unnoticed due to the presence of other abdominal injuries. Herniation of abdominal contents into the diaphragm may cause the patients to present with dyspnea, cyanosis, and chest pain. Chest x-rays may help in the diagnosis by showing herniated viscera in the chest (figure 7). Other means of diagnosis include ultrasound, or CT scan. Treatment involves reduction of herniated viscera and closure of the diaphragmatic defect.

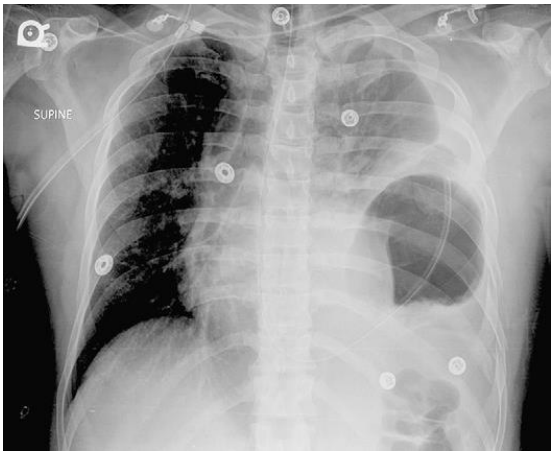


Figure 32 herniation of the stomach through a injured diaphragm

TRACHEOBRONCHIAL INJURIES

These are serious injuries that has a high mortality rate of 30% and involves injury to the trachea and bronchi. 80% of these injuries are within 2.5 cm from the carina, patients may present with

hemoptysis or dyspnea (reduced air entry to the lung). reduced air entry to the lung secondary to injury of one of the bronchi can cause lung collapse. Severe subcutaneous emphysema can suggest tracheobronchial disruption. It can be diagnosed by bronchoscopy. A chest drain placed on the affected side will reveal a large air leak and the collapsed lung may fail to re-expand, If after insertion of two drains the lung fails to re-expand, referral to a trauma center is advised. The condition is sometimes managed conservatively, but if on bronchoscopy, more than 1/3 of the circumference is involved then surgery is commenced. Surgery involves intubation of the unaffected bronchus followed by operative repair.

INJURIES TO THE GREAT VESSELS

Always Suspect the possibility of injury to the great vessels from the mechanism or site of penetrating injury. Injuries to the great vessels are associated with automobile collision or fall from a great height. The patients

Usually presents with shock or pericardial tamponade, features suggesting great vessels injury on Chest x-ray are shown in the table below.

possible chest x-ray findings in a patient with great vessel injury
• **Widening of the mediastinum to greater than 8 cm** • **Depression of the left main bronchus to greater than 140 degrees** • **Hematoma in the left apical area** • **Massive left hemothorax** • **Deviation of esophagus to the right** • **Loss of aortic knob contour** • **Loss of paraspinal pleural stripe**

Angiography and multislice CT scan can also diagnose and plan surgery for major vessel injuries. These injuries require emergency thoracotomy or sternotomy. (Injuries to descending thoracic aorta require left anterior thoracotomy. Injuries to proximal aorta and proximal carotid arteries require median sternotomy).

ABDOMINAL TRAUMA

INTRODUCTION

Abdominal trauma is Injury to abdominal organs, especially those in the retroperitoneal space caused by blunt or penetrating injuries, when bleeding occur the space can hold a great deal of blood, up to four liters. Trauma is the leading cause of death among individuals under the age of 45 years, and after head and thoracic traumas, abdominal trauma is the third ranking cause of death. The leading cause of abdominal trauma in the civilian population is road traffic accidents (RTA). Death from abdominal trauma in the first 48 hours is primarily due hemorrhage but after that, it's mainly due to sepsis. Solid organs, such as the liver and spleen bleed profusely as do the major abdominal blood vessels, the aorta and vena cava, while Injury to hollow organs presents a serious risk of infection, especially if there is a delay in diagnosis

CATEGORIES OF ABDOMINAL TRAUMA

Abdominal trauma can be due to either blunt or penetrating injuries

BLUNT (NOT SHARP) ABDOMINAL TRAUMA

Blunt trauma is the most common form of abdominal trauma and is mainly caused by Road Traffic Accidents (RTA) (accounting for approximately 75%) in the form of auto to auto or in the form of auto to pedestrian collision. Other causes of abdominal trauma are either direct blow to the abdomen (an example is shown in the figure below) or falling down injuries.

The mechanisms that result in injury from a blunt trauma are as follows:

• **Sudden Deceleration**; sudden deceleration can result in differential movement among adjacent structures especially at relatively fixed points of attachment such as the ligament of Treitz (duodenum fixed, jejunum mobile), the ileocecal valve (cecum fixed, ileum mobile), the phrenocolic ligament and Ligamentum teres and can result in traction of the mobile organs on an axis drawn by the fixed ones. This can result in tearing of such organs.

• **Compression with crush**; this can occur when intraabdominal contents are crushed between the anterior abdominal wall and the vertebral column or posterior thoracic cage. They are most commonly seen in injuries that result from auto to pedestrian traumas.

• **Compression with rupture**; external compressive forces that result in a sudden and dramatic rise in intra-abdominal pressure and culminate in rupture of a hollow viscous. For example, blunt trauma to the abdomen while the glottis is closed can cause rupture of the esophagus or compression to the ileum while the ileo cecal valve is closed can culminate in ileal rupture.

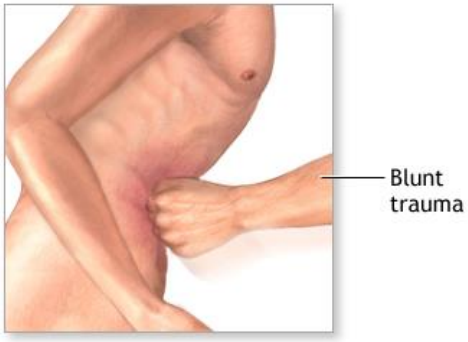


Figure 33 example of blunt trauma

PENETRATING ABDOMINAL TRAUMA

Penetrating abdominal traumas are either caused by missile injuries or stabs. The mechanism of penetrating injuries involves Mechanical disruption of the tissues along the path of stab or bullet passage.

When velocities exceed 500-600 m/s, cavitation injury- a temporary space torn in tissues at right angles to the direction of travel is produced. This process develops in microseconds and, depending upon the body tissues involved and their elasticity, can involve a volume many times the diameter of the bullet itself (see figure 2). Cavitation within a solid organ can result in shattering of that organ (e.g. liver).

Trauma is responsible for approximately 50% of all deaths in pregnant women, most commonly due to motor vehicle collisions, although both falls and

abuse are major causes as well. A gravid uterus displaces the majority of the intra-abdominal organs, and thus relatively protects the mother from penetrating abdominal injury. In general, pregnant patients should be managed similarly, to nonpregnant patients, following the dictum that the best way to take care of the fetus is to take care of the mother

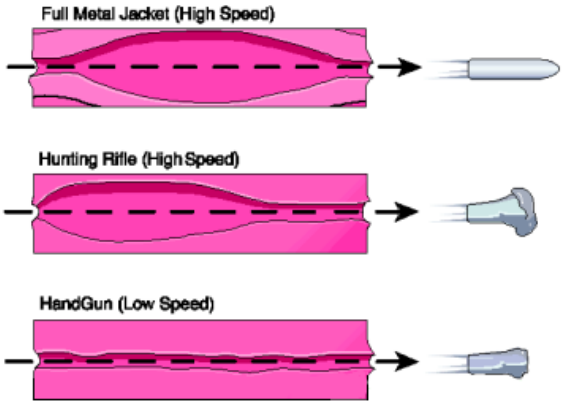


Figure 34 cavitation injury

The table below presents the frequencies of involvement of abdominal organs in both blunt and penetrating injuries.

Frequency of Organ Injury	Blunt.	Penetrating
Liver	15%	22%
Spleen	27%	9%
Pancreas	2%	6%
Kidney	27%	9%
Stomach	1%	10%
Duodenum	3%	4%
Small bowel	6%	18%
Colon	2%	16%
Other	17%	6%

ASSESSMENT OF ABDOMINAL TRAUMA

ASSESSMENT OF BLUNT TRAUMA

Assessment of blunt trauma revolves around one question 's there an intrabdominal injury?'. After the primary survey, assessment of abdominal injury could be done clinically with history and examination. Investigations are indicated when the clinical approach is elusive.

HISTORY FROM RTA PATIENTS SHOULD INVOLVE;

- the type of collision (front to front, front to side, front to pedestrian, crashing...),
- the extent of car damage,
- the patient position in the vehicle (wither he was belted or not),
- the extraction time (the time it took for bystanders or fire fighters to extract the patient out of the car),
- the status of other passengers and wither there was any death.

These may give you a clue about the severity of the underlying injury. You must always obtain history from the pre-hospital team or record as to wither there was hypotension.

The height of falling down is the most important piece of information in falling down injuries. As will be seen later, the site of a direct blow to the abdomen is very important in the assessment of retroperitoneal injuries. Examination of the abdomen may reveal some evidence of intra-abdominal injury. On inspection always look for abdominal distention, marks resulting from seat belts (indicates intra-abdominal injury in about one third of patients) or steering wheel, also, Look for the presence of eccymotic area indicative of retroperitoneal hematoma and for abrasions, or any skin discoloration.

ASSESSMENT OF PENETRATING TRAUMA

History taking in penetrating traumas should be abbreviated to asking about the time between the injury and arrival of pre-hospital team, the type of weapon (knife, handgun, shotgun,... etc) and if it is a knife what is the length and how many times it

stabbed the patient, or if it is a gunshot, what is the type and how many bullets were shot.

EXAMINATION SHOULD FOCUS ON THE FOLLOWING

- **Hemodynamic instability**; this is very important in establishing the plan for management, assessment should be done after stabilization, assessment involves dividing the patients into 3 groups; (1) hemodynamically normal; investigation can be full and treatment planned; (2) hemodynamically stable; investigation is more limited and is aimed at establishing whether the patient can be managed non-operatively, whether surgery is required (3) hemodynamically unstable'; immediate surgical correction of the bleeding is required.
- **Site**; Any wound in the boundaries of the abdomen is considered as a potential abdominal injury (From the 5th intercostal space down to the inferior gluteal fold posteriorly and to the inguinal ligament anteriorly). Any stab wound concerning the retro-peritoneum, consider, the ascending and descending colon.
- **Signs of peritoneal irritation**; guarding, rigidity, tenderness, rebound tenderness
- **Crepitus at lower thoracic cage indicative of rib fracture.**
- **Pelvic instability**
- **Abdominal distension**; this is a late sign (at least 6 hours).
- **Evisceration**; exit of the viscera.
- **Digital rectal examination**; in case of rectal injury as the rectum is extraperitoneal and injury to such organ may not give any sign of abdominal irritation

DIAGNOSTIC AIDS FOR EVALUATION

Clinical examination is never sufficient for assessment and diagnosis of abdominal injuries. An established diagnosis and full assessment is required for. planning management, this is only achieved if further investigations is to be done. Serial physical examinations may help identify wither the patient is improving or is deteriorating.

LOCAL WOUND EXPLORATION

In cases of penetrating traumas, especially stab wounds, an exploration of the wound may help establish the depth; the procedure is this can be done by dilatation at the site of the wound and inserting a probe. The distance of the probe at which it stops is the depth of the wound. In cases of missile injuries, radiological studies have replaced this technique. If the probe by passes the peritoneum, laparotomy is indicated.

FOCUSED ABDOMINAL SONAR FOR TRAUMA

Focused abdominal sonar for trauma (FAST) is a technique whereby ultrasound imaging is used to assess the torso for the presence of blood in the abdominal cavity. The purpose of the ultrasound evaluation of the injured casualty is to determine the presence of free intraabdominal fluid and is not used to identify organs injury. Thus, the study focuses on the presence of blood in Morris pouch, or at the paracolic gutters. This technique can be used in the unstable patient (can be done during resuscitation). Presence of blood in the abdominal cavity is an indication for laparotomy.

CT SCAN

CT of the abdomen is the preferred diagnostic examination for the evaluation of blunt abdominal trauma in the hemodynamically stable patient. CT can identify injuries to all solid abdominal and retroperitoneal organs (liver, spleen, pancreas, kidney), bowel perforations (indirectly by the presence of free gas and fluid), diaphragmatic rupture, retroperitoneal blood and pelvic and spinal fractures. (the most common diagnosis in retroperitoneal organ injury)

DPL (DIAGNOSTIC PERITONEAL LAVAGE)

Diagnostic peritoneal lavage (DPL) is a test used to assess the presence of blood in the abdomen. A gastric tube is placed to empty the stomach and a urinary catheter is inserted to drain the bladder. A cannula is inserted below the umbilicus, directed causally and posteriorly. The cannula is aspirated for blood (> 10 ml is deemed as positive) and, following this, 1000 ml of warmed Ringer's lactate solution is allowed to run into the abdomen and is then drained out. The presence of > 100 000 red cells/μL or > 500 white cells/μL is deemed positive (this is equivalent to 20 ml of free blood in the abdominal cavity). The presence of amylase (>175 units), enteric contents, or bile are also positive

results for diagnostic peritoneal lavage (indicating, bowel, biliary tree and pancreatic injury). Its not sufficient to only withdraw blood from the abdominal cavity and assume that the test is positive, as there might be a leak of blood from the incision that can give false positive results, thus confirmation with ringer lactate irrigation is a must. Laparotomy is indicated in cases of positive peritoneal lavage. The procedure is still performed when alternative diagnostic methods such as computerized tomography (CT) or ultrasound imaging are unavailable.

Indications for diagnostic peritoneal lavage are shown in the table below.

Indication for DPL
1. > 5mls of blood aspirated before fluid is infused.
2. Bloody irrigated fluid
3. the presence of bile,
4. enteric contents.
5. Hematological & biochemical tests for the aspirated fluid:
a. RBC > 100,000/cmm
b. WBC > 500 /cmm
c. Amylase > 175 units

INDIVIDUAL ORGAN INJURY

SPLEEN

Splenic injury occurs from direct blunt trauma; the spleen is often injured by direct energy applied to the overlying ribs (ninth to 11th ribs). Penetrating injuries can also involve the spleen. Injury to the spleen is graded as shown in the table below. Grade 1, 2 and 3 require CT scan observation and Conservative treatment. Conservative treatment should be followed by selective embolization of one of the branches of the splenic artery that supplies the area of injury to stop any expanding hematoma. Surgery should focus on preservation of the spleen to avoid because of the dangers of post-splenectomy sepsis. Operations that aim to preserve the spleen include spleenorraphy, or partial splenectomy (ligation of a segmental splenic artery and removal of the affected segment). Total splenectomy is mainly indicated for hilar injury and total shattering (grade 4 and 5).

Clinical features of splenic rupture:

Symptoms:

- May be painless, or LUQ/diffuse abdominal pain.

- referred left shoulder pain in splenic laceration: Kehr's sign

- Syncope due to hypotension.

Signs

- Physical examination is insensitive and nonspecific.

- Pt may have signs of left upper quadrant tenderness or signs of generalized peritoneal irritation.

- May present with tachycardia, tachypnea, hypotension or shock

Grading of splenic injury	
Grade 1	Minor sub capsular tear (< 1 cm parenchymal depth) or non-expanding sub capsular hematoma
Grade II	sub capsular tear (1-3 cm parenchymal depth) sub capsular hematoma (10-50%) surface area
Grade III	sub capsular tear (>3 cm parenchymal depth), > %50 sub capsular hematoma
Grade IV	laceration >25% of the spleen, ruptured intra- parenchymal hematoma
Grade V	totally shattered spleen or hilar vascular injury

LIVER

Isolated, relatively small liver injuries (grade 1,2 .and 3) may be treated by surgical repair (pregimen manoeuvre, suturing) or local resection but major injuries (grade 4 and 5) are often best treated conservatively until a hepatobiliary unit is to intervene.

CLINICAL FEATURES OF LIVER INJURY:

Symptoms of a liver injury

-right upper quadrant pain, increase with deep breathing.

-nausea or vomiting, tachycardia and fainting,

Physical examination:

-tenderness to palpation in the right upper quadrant of the abdomen. Abnormalities of blood pressure and pulse will be noted (low blood pressure and pulse over 100).

Grading for liver injury is shown in the table below.

Grading for liver injury
Grade I: **Sub capsular hematoma < 10%of surface area, nonexpanding.** **Laceration < 1cm parenchymal depth, nonbleeding.**
Grade II: **Sub capsular hematoma 10-50%** **Parenchymal Laceration 1-3 in depth, <10 cm in length.**
Grade III: **Sub capsular hematoma >50% 3cm parenchymal depth.**
Grade IV: **: Ruptured intra parenchymal hematoma with active bleeding.** **Parenchymal disruption involving 25-50%of hepatic lobe.**
Grade V: **Parenchymal disruption >50%of hepatic lobe Vascular injuries hepatic veins, Inferior Vena cava.**

DISORDER OF THE SALIVARY GLAND

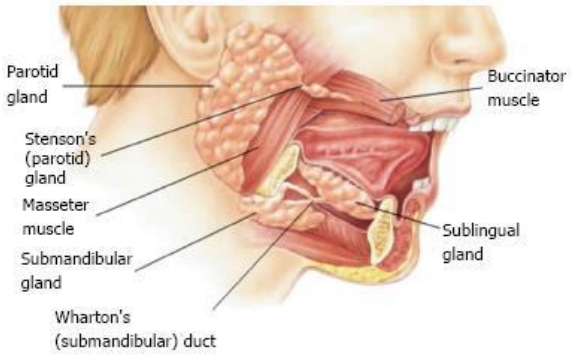


Figure 35 major salivary glands

INTRODUCTION

There are three paired major salivary glands, two parotid glands, two submandibular glands and two sublingual glands. In addition, there are multiple minor salivary glands

MINOR SALIVARY GLANDS

The mucosa of the oral cavity contains approximately 800 minor salivary glands. They are distributed in the mucosa of the lips, cheeks, palate, floor of the mouth and retromolar area. These minor salivary glands also appear in other areas of the upper aerodigestive tract including the oropharynx, larynx and trachea as well as the sinuses. They have a histological structure similar to that of mucus-secreting major salivary glands. Overall, they contribute to 10% of the total salivary volume.

COMMON DISORDERS OF MINOR SALIVARY GLANDS

CYSTS

Cysts are common and arise from trauma to the glandular parenchyma or drainage system. The minor salivary glands have the distinction that they can secrete against pressure and most cysts are of the extravasation rather than retension variety.

They arise in the loose mucosa of the lower lip and the floor of mouth where saliva can easily collect. The swelling is painless and usually, but not always, translucent (Figure 2). Some resolve spontaneously, but most require formal surgical excision +overlying mucosa +underlying minor salivary gland. Recurrence is rare

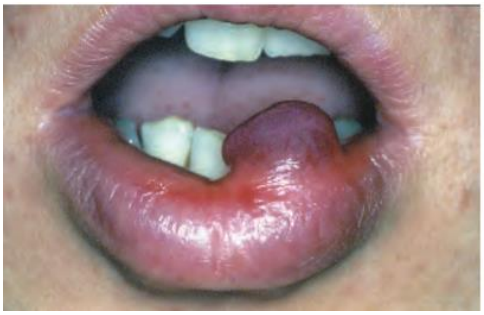


Figure 36 Mucous retention cyst. A translucent swelling on lower lip is typical.

TUMOURS

Few tumours show more diversity in histological appearance and anatomical site than those that arise from mucous glands of the upper aerodigestive tract. Tumours of minor salivary glands are histologically similar to those of major glands; however, more minor salivary gland tumours are malignant and as are almost all tumours in the sublingual glands. Tumours of minor salivary gland origin occur anywhere in the upper aerodigestive tract; however, common sites for tumour formation include the palate, upper lip and retromolar regions. Less common sites for minor salivary gland tumours include the nasal and pharyngeal cavities. These tumours arise in submucosal seromucous glands that are found throughout the upper aerodigestive tract. A well-defined rubbery lump is a salivary gland tumour until proven otherwise.

Benign minor salivary gland tumours present as painless, firm, slow-growing swellings. Overlying ulceration is extremely rare. Minor salivary gland tumours are managed by excision to include the overlying mucosa, with primary closure (Figure 3).

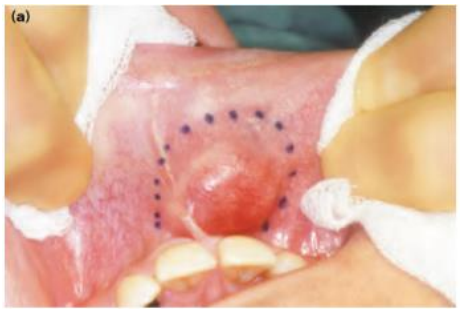


Figure 37

Benign tumours of the palate, less than 1 cm in diameter, can be managed by excisional biopsy, and the defect left to heal by secondary intention (Figure 4). Where the tumours are greater than 1 cm in diameter, a 3 mm punch biopsy (dermatological punch) is recommended to establish a diagnosis prior to formal excision and to avoid the embarrassment of inadvertently encountering a low-grade malignant lesion.

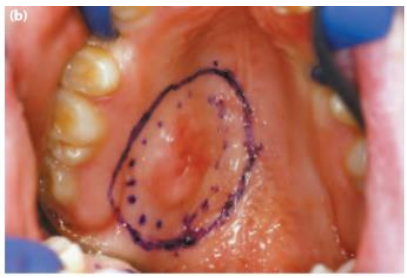


Figure 38

MALIGNANT NEOPLASMS

Malignant minor salivary gland tumours are rare (2 per million population). Most are low grade and present as apparently benign lumps. They have a firm consistency, and the overlying mucosa may have a varied discolouration from pink to blue or black (Figure 5). High-grade lesions usually become necrotic with ulceration as a late presentation

Malignant minor salivary gland tumours of the palate that are low grade and early stage can be managed by wide excision with burring down of the underlying bone and then left to heal by secondary intention. Those that have perforated the palate may require partial or total maxillectomy. The subsequent defect can be managed by either prosthetic obturation or immediate reconstruction. Various microvascular flaps have been designed to reconstruct maxillectomy defects including radial forearm flap, fibular flap, rectus abdominus, latissimus dorsi and vascularised iliac crest graft (Figure).

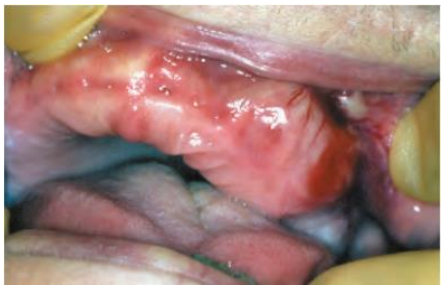


Figure 39 adenoid cystic carcinoma

Malignant minor salivary gland tumours of the palate that are low grade and early stage can be managed by wide excision with burring down of the underlying bone and then left to heal by secondary intention. Those that have perforated the palate may require partial or total maxillectomy. The subsequent defect can be managed by either prosthetic obturation or immediate reconstruction. Various microvascular flaps have been designed to reconstruct maxillectomy defects including radial forearm flap, fibular flap, rectus abdominus, latissimus dorsi and vascularised iliac crest graft (Figure 6).

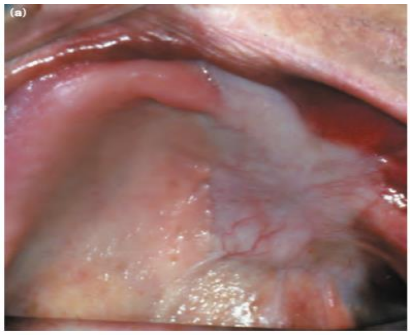


Figure 40

NECROTISING SIALOMETAPLASIA

This is a well-established but rare entity. Typically, it occurs in the palate and mimics an aggressive cancer. It presents as a deep punched out ulcer with an indurated margin. It cannot be distinguished from a neoplastic lesion except by biopsy. The diagnosis is suggested by rapid onset in a young person. The lesion resolves spontaneously with symptomatic treatment.

THE SUBLINGUAL GLANDS

ANATOMY

The sublingual glands are a paired set of salivary glands lying in the anterior part of the floor of the mouth between the mucous membrane, the mylohyoid muscle and the body of the mandible close to the mental symphysis. The gland has a head portion that drains by numerous excretory ducts (Ducts of Rivinus) directly into the oral cavity and the tail that drains into the submandibular duct or directly into the mouth.

COMMON DISORDERS OF THE SUBLINGUAL GLANDS

CYSTS

The term 'ranula' is applied to a mucous extravasation cyst that arises from the sublingual gland. It produces a characteristic translucent swelling that takes on the appearance of a 'frog's belly' (ranula) (Figure 8). A ranula can resolve spontaneously, but many also require active treatment. The traditional and effective way is to remove the sublingual gland, but it is not an easy operation and the morbidity can be significant. New less invasive techniques are now quite effective (85% success) in resolving ranulas while preserving the gland. Incision and drainage, however tempting, usually results in recurrence. Ranula is derived from 'rana', the Latin for frog.

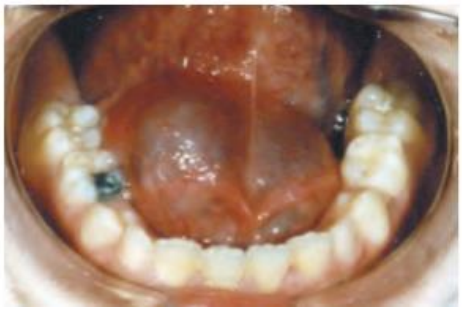


Figure 42 large ranula affecting the floor of the mouth

PLUNGING RANULA

Plunging ranula is a rare form of mucous retention cyst that arises from the sublingual salivary glands. Mucus collects below the gland and perforates through the mylohyoid muscle diaphragm to enter the neck. Patients present with a dumbbell-shaped swelling that is soft, fluctuant and painless in the submandibular or submental region of the neck (Figure 7). Diagnosis is made on ultrasound or magnetic resonance imaging (MRI) examination but clinched by aspirating thick yellow treacly fluid from the cyst. This distinguishes it from a lymphangioma. A cervical approach is now contraindicated. The cyst arises from the sublingual gland. All that is required is to remove the sublingual gland and aspirate the saliva out of the sac. The latter is formed of connective tissue not epithelium so melts away once the leak of saliva is resolved.

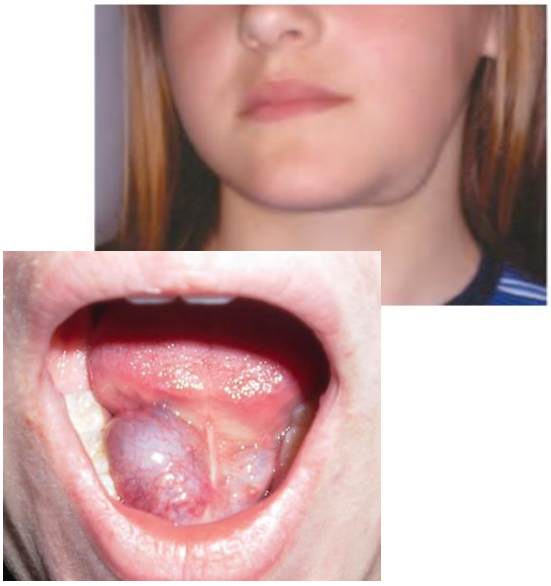


Figure 41 example of Plunging ranula

TUMOURS

Tumours involving the sublingual gland are extremely rare and are usually (90%) malignant. They present as a rubbery painless swelling in the floor of the mouth. Pain or lingual nerve paraesthesia indicate a high-grade tumour. All such lumps must have a formal punch biopsy prior to formulating a treatment plan. Treatment requires a block wide excision involving the overlying mucosa and the adjacent periosteum with simultaneous neck dissection depending on the stage of the disease. Immediate reconstruction of the intraoral defect is recommended, especially when communication with the neck has been established. Radial artery forearm free flap or anterolateral thigh flap is usually

the reconstruction of choice for fit patients, otherwise a pedicled pectoralis major flap can be used. There is normally a low threshold for adjuvant radiotherapy except for low-grade and stage lesions

THE SUBMANDIBULAR GLANDS

ANATOMY

The submandibular glands are paired salivary glands that lie below the mandible on either side. They consist of a larger superficial and a smaller deep lobe that are continuous around the posterior border of the mylohyoid muscle. Important anatomical relations include the anterior facial vein and artery running over the surface of the gland in close association with the ramus mandibularis (marginal mandibular) of the facial nerve. The deep part of the gland lies on the hyoglossus muscle closely related to the lingual nerve and inferior to the hypoglossal nerve. The gland is surrounded by a well- defined capsule that is derived from the deep cervical fascia which splits to enclose it. The gland is drained by a single submandibular duct (Wharton's duct) that emerges from its deep surface and runs in the space between the hyoglossus and mylohyoid muscles. It drains into the anterior floor of the mouth at the sublingual papilla. There are several lymph nodes immediately adjacent and sometimes within the superficial part of the gland

ECTOPIC/ABERRANT SALIVARY GLAND TISSUE

The most common ectopic salivary tissue is a Stafne bone cyst, the origin of which is uncertain. It presents as an asymptomatic, clearly demarcated radiolucency of the angle of the mandible, characteristically below the inferior dental neurovascular bundle (Figure 9). The cavity in the lingual plate houses part of the submandibular gland or ectopic salivary tissue, but the origin of the cavity is unclear and has been attributed to the pulse pressure from the facial artery. No treatment is required

INFLAMMATORY DISORDERS OF THE SUBMANDIBULAR GLAND

Inflammation of the submandibular gland is termed sialadenitis. Submandibular sialadenitis may be acute, chronic or acute on chronic. Common causes are:

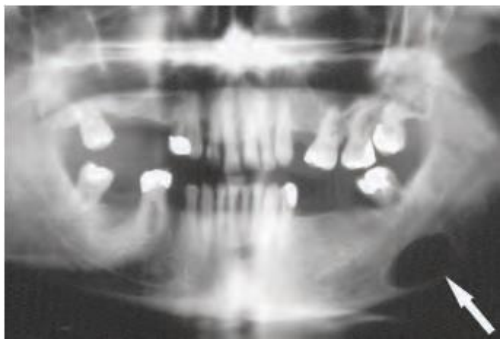


Figure 43 (arrow) Stafne bone cyst

1) Acute submandibular sialadenitis

• Viral. The paramyxovirus (mumps) is a viral illness of the salivary glands that usually produces parotitis. The submandibular glands are occasionally involved, causing painful tender swollen glands. Other viral infections of the submandibular gland are extremely rare.

• Bacterial. Bacterial sialadenitis is more common than viral sialadenitis and occurs secondary to obstruction by stone. These stones can be reliably removed by minimally invasive techniques that preserve the gland.

2) Chronic submandibular sialadenitis.

OBSTRUCTION AND TRAUMA

The most common cause of obstruction within the submandibular gland is stone formation (sialolithiasis) within the gland and its associated duct system. Eighty per cent of all salivary stones occur in the submandibular glands because their secretions are relatively viscous. Eighty per cent of submandibular stones are radio-opaque and can be identified on plain radiography (Figure 10). Stones are mainly composed of phosphate and oxalate salts.

The second most common cause of submandibular duct obstruction is stricture. The remaining 5–10% of cases are secondary to floor of mouth pathology or external pressure, particularly trauma to the floor of the mouth from an overextended flange on a lower denture that impinges on the sublingual papilla, causing inflammation and subsequent stricture.

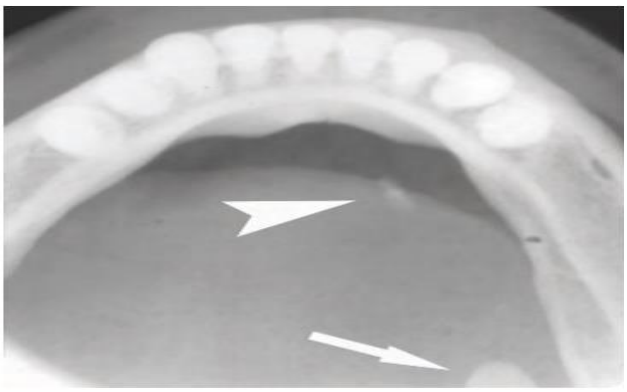


Figure 44 radio-opaque submandibular stones

CLINICAL SYMPTOMS

Patients usually present with acute painful swelling in the region of the submandibular gland, precipitated by eating (Figure 11). The swelling occurs rapidly and often resolves spontaneously over 1–2 hours after the meal is completed (meal-time syndrome). This classical picture occurs when the stone causes complete obstruction as it is washed forward and impacts in the lumen. The two most common sites of impaction are the hilum of the gland as the duct bends over the mylohyoid muscle and near the punctum. The symptoms are frequently intermittent as the stone can dislodge and allow the gush of saliva induced at meal times to pass. In such circumstances, symptoms are moderate with minimal discomfort and swelling. Clinical examination reveals an enlarged firm submandibular gland, tender on bimanual examination. Pus may be visible draining from the sublingual papilla or expressed by bimanual palpation of the gland .

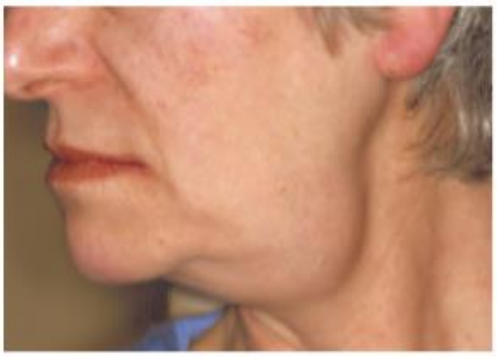


Figure 45 acute painful swelling caused by obstruction

MANAGEMENT

Small (less than 4 mm) mobile stones can be retrieved using a Dormia® basket. This minimally invasive procedure is usually performed under local anaestheisa either endoscopically (sialendoscopy) or under radiological control (ultrasonography). For larger stones, extracorporeal or intracorporeal lithotripsy can be employed to break the stone into smaller pieces using shock waves and the stone fragments can then removed with a basket. Recently, a pneumatic intraductal lithotripter (Stonebreaker™) has been developed that can be advanced into the duct lumen under endoscopic control to fragment the stone. Results are promising, with 70% of stones being cleared in selected cases.

If the stone is lying within the submandibular duct in the floor of the mouth anterior to the point at which the duct crosses the lingual nerve (second molar region), the stone can be removed under local anaesthetic but not by incising directly on to the duct. The latter runs under the sublingual gland and trauma to the head of the sublingual gland induces ranulae. Therefore, the incision has to run along the medial margin of the gland and the latter is then rotated laterally to expose the duct on its deep surface. Once the stone has been delivered via a single longitudinal incision along its surface, the duct should be closed by a resorbable suture and the incision in the floor of mouth closed.

Where the stone is proximal to the lingual nerve (i.e. at the hilum of the gland), stone retrieval via an intraoral approach can be performed quite reliably under general anaesthesia. The key is to choose cases in which the stone is palpable. This approach can be difficult and attendance on a suitable salivary course is recommended. Stone retrieval rates are excellent (95%). The risk to the lingual nerve is greatest with deep stones that are not palpable, as they lie outside the mouth below the mylohyoid. Traction injuries can occur in such circumstances. If stone retrieval fails, submandibular gland excision can be performed.

Indication:

1) Sialadenitis
2) Salivary tumours.

Excision of the submandibular gland involves four distinct phases:

1) **INCISION AND EXPOSURE OF GLAND (figure 12 a and b)**
2) **GLAND MOBILISATION (figure 12 c)**
3) **DISSECTION OF THE DEEP LOBE AND IDENTIFICATION OF THE LINGUAL NERVE (figure 12 d)**
4) **WOUND CLOSURE**

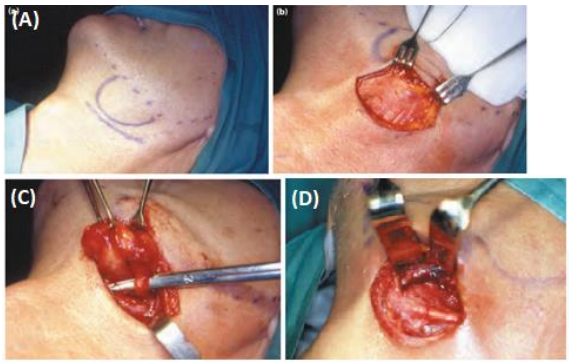


Figure 46 submandibular gland excision

Complications of submandibular gland excision are:

- haematoma;
- wound infection;
- marginal mandibular nerve injury;
- lingual nerve injury;
- hypoglossal nerve injury;
- transection of the nerve to the mylohyoid muscle producing submental skin anaesthesia.

TUMOURS OF THE SUBMANDIBULAR GLAND

Tumours of the submandibular gland are uncommon and usually present as a slow-growing, painless swelling within the submandibular triangle. About 60–70% of submandibular gland tumours are benign, in contrast to 80–90% of parotid gland tumours (Table). In many circumstances, the swelling cannot, on clinical examination be differentiated from submandibular lymphadenopathy. This can be resolved definitively by ultrasound examination. Most salivary neoplasms, even malignant tumours, are often slow-growing, painless swellings. The difficulty is to always distinguish between benign and malignant lesions prior to excision, as in up to 30% referrals of submandibular gland cancers to regional cancer centres, patients have already had surgical excision of the gland, which was subsequently found to be malignant on histological examination. Pain is not a reliable indication of malignancy; however, rapid growth, facial nerve palsy, lymph node enlargement and skin tethering are signs of a high-grade malignant lesion. The most common malignant tumour is an adenocystic carcinoma (40%), which may masquerade as a benign lump.

Salivary gland tumors - frequency and distribution.

Type	Location	Frequency	Malignant (%)
Major	• Parotid • Submandibular • Sublingual	• Common • Uncommon • Very rare	10-20 50 85
Minor	Upper aerodigestive tract	Rare	90

CLINICAL FEATURES OF HIGH-GRADE MALIGNANT SALIVARY TUMOURS

These include:

- facial nerve weakness;
- rapid enlargement of the swelling;
- induration and/or ulceration of the overlying skin;
- cervical node enlargement.

INVESTIGATION

The initial investigation of choice is ultrasound with fine- needle aspiration cytology (FNAC)/True-Cut biopsy. Once it is established that a tumour is present, computed tomography (CT) and MRI scanning are complimentary techniques for imaging tumours arising in the major salivary glands. The tumour is intrinsic to the gland. The scan will highlight the relationship of the tumour to other anatomical structures, which is helpful in planning surgery.

Open surgical biopsy is contraindicated as this may seed the tumour into surrounding tissues, making it impossible to eradicate microscopic deposits of

tumour cells. Fine-needle aspiration or True Cut biopsy is a safe alternative as the risk of seeding tumour is remote. The combination of careful history and examination in conjunction fine-needle aspiration cytology can identify over 95% of malignant cases. The few malignant cases that are indistinguishable from benign disease by these methods and are only identified by histology are usually indolent and act as benign tumours

MANAGEMENT OF SUBMANDIBULAR GLAND TUMOURS

Benign tumours of the submandibular gland can be safely removed by meticulous dissection outside the submandibular capsule. No instrument should be applied to the gland as to do so may crush it. As long as the capsule of the gland is preserved intact, the risk of recurrence is 1–1.5% at 10 years. As with all salivary gland tumours, surgical excision with a cuff of normal tissue is the goal.

The management of malignant salivary gland tumours is governed by the stage and clinical grade of the lesion. The larger and more aggressive the lesion, the more radical is the surgery required. Each case has to be judged on its own merits, but wide clearance of the submandibular triangle with some form of neck dissection is normally the treatment of choice. This may necessitate sacrifice of the lingual and hypoglossal nerves if the tumour is adherent to the deep bed of the gland . Adjuvant radiotherapy is usually dictated by pathological findings such as close margins and high-grade cancers. The prognostic cut off point for salivary cancers is 4 cm and most tumours larger than this need adjuvant therapy as will patients with adenoid cystic carcinoma if optimum results are to be achieved.

THE PAROTID GLAND

ANATOMY

The parotid gland lies in a recess bounded by the ramus of the mandible, the base of the skull and the mastoid process. It lies on the carotid sheath and CNs XI and XII and extends forward over the masseter muscle. The gland is enclosed in a sheath of dense deep cervical fascia. Its upper pole extends just below the zygoma and its lower pole (tail) into the neck. Several important structures run through the parotid gland. These include:

- the facial nerve trunk that divides into its major five branches;
- the terminal branch of the external carotid artery that divides into the maxillary artery and the superficial temporal artery;
- the retromandibular vein;
- intraparotid lymph nodes.

The gland is arbitrarily divided into deep and superficial lobes, separated by the facial nerve. Eighty per cent of the parotid gland lies superficial and 20% deep to the nerve. An accessory lobe is occasionally present lying anterior to the superficial lobe on the masseter muscle.

DEVELOPMENTAL DISORDERS

Developmental disorders such as agenesis, duct atresia and congenital fistula are extremely rare.

INFLAMMATORY DISORDERS

VIRAL INFECTIONS

Mumps is the most common cause of acute painful parotid swelling and predominantly affects children. It is spread via airborne droplets of infected saliva. The disease starts with a prodromal period of 1–2 days, during which the patient experiences fever, nausea and headache. This is followed by pain and swelling in one or both parotid glands. Parotid pain can be very severe and exacerbated by eating and drinking. Symptoms resolve within 5–10 days. The diagnosis is based on history and clinical examination; recent contact with an infected patient with a painful parotid swelling is often sufficient to lead to a diagnosis. Atypical viral parotitis does occur and may present with predominantly unilateral swelling or even submandibular involvement. A single episode of infection confers lifelong immunity. Treatment of mumps is symptomatic with regular paracetamol and adequate oral fluid intake. Complications of orchitis, oophoritis, pancreatitis, sensorineural deafness and meningoencephalitis are rare, but are more likely to occur in adults.

Other viral agents that produce parotitis include Coxsackie A and B, parainfluenza 1 and 3, Echo and lymphocytic choriomeningitis.

BACTERIAL INFECTIONS

Acute ascending bacterial sialadenitis is historically described in dehydrated elderly patients following major surgery. Reduced salivary flow secondary to dehydration results in ascending infection via the parotid duct into the parotid parenchyma. The more common picture today is an acute bacterial parotitis associated with a salivary calculus. The patient presents with a tender, painful parotid swelling that arises over several hours (Figure 13). There is generalised malaise, pyrexia and occasional cervical lymphadenopathy. The pain is exacerbated by eating or drinking. The parotid swelling may be diffuse, but often localises to the lower pole of the gland. Intraoral examination may reveal pus exuding from the parotid gland papilla. The infecting organism is usually Staphylococcus aureus or Streptococcus viridans, and treatment is with appropriate intravenous antibiotics. If the gland becomes fluctuant, ultrasound may identify abscess formation within the gland that may require aspiration with a large-bore needle or formal drainage under general anaesthesia. In the latter procedure, the skin incision should be made low to avoid damage to the lower branch of the facial nerve. Blunt dissection using sinus forceps is preferred, and the cavity is opened to facilitate drainage. A drain is inserted and left in situ for 24–72 hours. Sialography is contraindicated during acute infection. Chronic bacterial sialadenitis is rare in the parotid gland

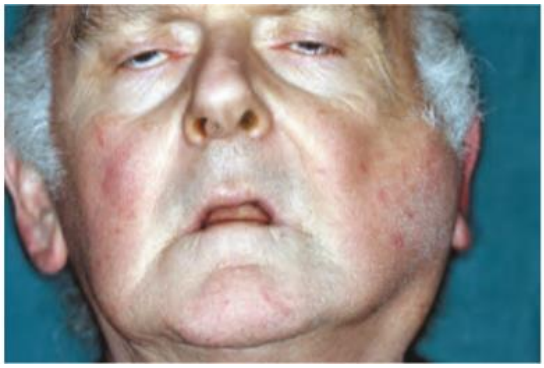


Figure 47

RECURRENT PAROTITIS OF CHILDHOOD

Recurrent parotitis of childhood is a distinct clinical entity of unknown aetiology and variable prognosis. It is characterised by rapid swelling of one or both parotid glands, in which the symptoms are made worse by chewing and eating. Systemic upset with fever and malaise is variable. The symptoms usually last from 3 to 7 days, and are then followed by a quiescent period of weeks to several months. Children usually present between the ages of 3 and 6 years, although symptoms have been reported in infants as young as 4 months. The diagnosis is based on the characteristic history and can be confirmed by sialography. This shows a characteristic punctate sialectasis likened to a 'snowstorm' (Figure 14). The condition is difficult to manage if it becomes established and so the initial treatment is important. The condition responds to regular endoscopic washouts and long courses of antibiotics. The suspicion is that in some cases the condition is caused by an incompetent punctum that leads to soiling of the parotid ducts with contaminated oral fluids

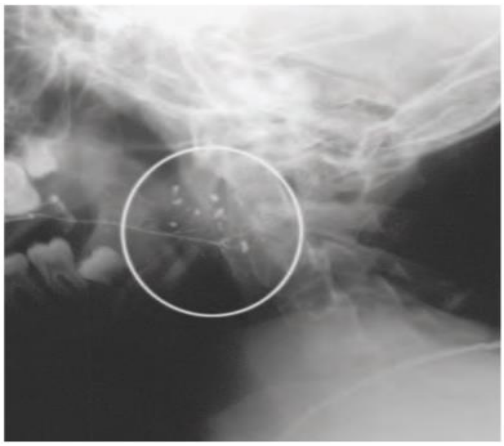


Figure 48 punctate sialectasis, 'snowstorm' apperance

HUMAN IMMUNODEFICIENCY VIRUS-ASSOCIATED SIALADENITIS

Chronic parotitis in children may signify human immunodeficiency virus (HIV) infection. The presentation of HIV- associated sialadenitis is very similar to classical Sjögren's syndrome in adulthood. Although HIV-associated sialadenitis and Sjögren's syndrome are histologically similar, the former condition is usually associated with a negative autoantibody screen. Other presentations of salivary gland disease in HIV-positive patients include multiple parotid cysts, which cause gross parotid swelling and facial disfigurement. CT and MRI demonstrate the characteristic 'Swiss cheese' appearance of multiple large cystic lesions (Figure

15). The swollen glands are usually painless and may regress on the institution of antiviral therapy. Cysts can be aspirated

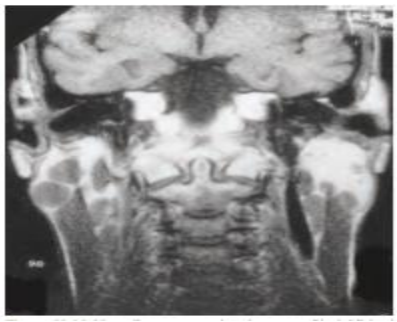


Figure 49

TB PAROTITIS

TB parotitis or tuberculous parotitis is a granulomatous inflammation of the parotid gland that results for *Mycobacterium tuberculosis* infection. It is most common in older children and adults. Primary salivary gland infection by *M. tuberculosis* is believed to evolve from a focus in the tonsils or gingival sulcus ascending to the glands by way of their ducts. Secondary infection of the salivary glands occurs by way of hematogenous or lymphatic spread from the lungs. Mycobacteria are encapsulated in the intraglandular lymph nodes and might be reactivated many years after the primary infection.

CLINICAL FEATURES AND MANAGEMENT

It usually presents as a painless chronic tumorous lesion that is seen as a discrete slow-growing mass that mimics a neoplasm. With Constitutional signs including fever, night sweats, and weight loss might be absent. Involvement of the facial nerve is rare.

Methods of diagnosis such as chest x-ray, purified protein derivative (PPD) skin test and FNA biopsy are hardly sensitive and the diagnosis is usually made intraoperatively.

Once diagnosed, *M. tuberculosis* infection may be treated with triple-drug therapy. In cases in which the diagnosis is uncertain or the lesion is resistant to medical therapy, complete surgical excision is both diagnostic and curative.

CSD

cat-scratch disease (CSD) is a granulomatous lymphadenitis that most commonly results from cutaneous inoculation caused by scratch trauma from a domestic cat. The causative organism is classified as *Bartonella henselae,* a gram-negative bacillus.

The typical history is of a papule or, pustule at a scratch or bite site followed in 1 to 2 weeks by the development of lymphadenopathy in the region of inoculation. The nodes will slowly enlarge over a period of 1 to 2 weeks and might not resolve for 2 to 3 months. Fever and mild systemic symptoms might occur. Parotid enlargement occurs in 3% of cases and is more likely to be a preauricular adenopathy which might be confused with a parotid neoplasm. Other atypical presentations include encephalitis.

Methods of DNA diagnosis and serology are rarely required. In most cases, no active therapy is required. The patient should be reassured that the lymphadenopathy is self-limited and usually will resolve spontaneously in 2 to 4 months.

TOXOPLASMOSIS

Toxoplasmosis is caused by the organism *Toxoplasma gondii.* The usual host for this organism is the domestic cat which are transmitted by their feces in the form of cysts.

Parotid gland disease might involve singular or multiple intraparotid or periparotid lymph nodes and is associated with isolated cervical lymphadenopathy and constitutional symptoms. diagnosis can be made serologically with the **Sabin-Feldman dye test.**

The disease is self-limiting. Chemotherapy is generally reserved for obviously progressive infections or those involving pregnant or immunocompromised individuals.

OBSTRUCTIVE PAROTITIS

There are several causes of obstructive parotitis, which produces intermittent painful swelling of the parotid gland, particularly at mealtimes.

STONE FORMATION AND STRICTURES

Sialolithiasis is less common in the parotid gland (20%) than in the submandibular gland (80%). Parotid duct stones are usually radiolucent and rarely visible on plain radiography. They are frequently located at the confluence of the collecting ducts, at the point the duct courses over the masseter muscle or in the distal aspect of the parotid duct adjacent to the parotid papilla. The stones are easily demonstrated on ultrasound. The same rules for treatment apply to parotid stones as to submandibular duct stones. Small stones (~4 mm) can be retrieved by baskets, slightly larger stones up to 8 mm can be broken with lithotripsy and stones over 8 mm diameter should be removed by endoscopic assisted surgery while preserving the gland. Strictures are common in the parotid gland and are responsible for about 20% of obstructive cases. The symptom complex is a little different as the obstruction is due to mucus plugs. These form after periods of stagnation. Classically, the patient complains of a meal-time syndrome starting at breakfast and the saliva cannot seep past the mucus plug so the swelling persists. Massage eventually releases the plug with a gush of salty saliva. Infection is uncommon unless there is stone formation. Strictures respond to dilatation and endoscopic washouts with steroid solutions.

PAPILLARY OBSTRUCTION

Obstructive parotitis, can be caused by trauma to the parotid papilla. The subsequent inflammation and edema obstruct salivary flow, particularly at mealtimes. This is a rare but real entity. The partial obstruction over a protracted period leads to dilation of the duct and an entity called 'mega-duct'. A large dilated duct is visible coursing over the patient's cheek. Drainage has to be re-established. This can be done by progressive dilatation of the punctum and the insertion of a stent that is kept in position for many weeks. Surgical attempts to refashion the punctum are unlikely to be successful. Papillotomy should not be performed as this often leads to stricture formation and a life time of problems. This is not the case with the submandibular gland

TUMOURS OF THE PAROTID GLAND

Some 80-90% of tumors of the parotid gland are be**nign,** the rest being malignant. Most of benign salivary glands tumors are of epithelial origin. The rest comprises tumors of non-epithelial origin such as hemangioma and lymphangioma. Tumors of epithelial origin are referred to as adenomas. See the table below.

Classification of parotid gland tumors

benign

- **Epithelial (adenomas)**
  - Pleomorphic adenoma
  - Monomorphic adenoma
    - Adenolymphoma (Warthin's tumour)
    - Oncocytoma (oxyphilic adenoma)
- **Non-epithelial**
  - Hemangioma
  - lymphangioma

malignant

Primary

- ***Epithelial (carcinomas)***
  - Mucoepidermoid
  - Adenoid cystic carcinoma
  - Acinic cell carcinoma
  - Adenocarcinoma
  - Squamous cell carcinoma
- ***Non-Epithelial***
  - Non-hodgkin's lymphomas

Secondary malignancy

- **Lymphomas in Sjogren's syndrome**
- **Metastasis**

The number 80% is seen a lot in salivary gland tumors. See the table below.

the rule of 80% in parotid gland tumors

- **80%of the salivary tumors occur in the parotid**
- **80%ofthe parotid tumors are benign**
- **80% of the benign tumors of the parotid are pleomorphic adenoma**
- **80% of the pleomophic adenomas occur in the**
- **superficial lobe**

MALIGNANT TUMOR

The parotid gland is the most common site for salivary tumours. Most tumours arise in the superficial lobe and present as slow-growing, painless swellings below the ear, in front of the ear ,or in the upper aspect of the neck. Less commonly, tumours may arise from the accessory lobe and present as persistent swellings within the cheek. Rarely, tumours may arise from the deep lobe of the gland and present as a parapharyngeal mass. Symptoms include difficulty in swallowing and snoring. Clinical examination reveals a diffuse firm swelling in the soft palate and tonsil.

Some 80–90% of tumours of the parotid gland are benign, the most common being pleomorphic adenoma (Table 49.2).

Malignant salivary gland tumours are divided into two distinct sub-groups:

1- **Low-grade malignant tumours** (e.g. acinic cell carcinoma) are indistinguishable on clinical examination from benign neoplasms

2- **High-grade malignant tumours** usually present as rapidly growing, often painless swellings in and around the parotid gland. The tumour presents as either a discrete mass with infiltration into the overlying skin or a diffuse but hard swelling of the gland with no discrete mass. Presentation with advanced disease is common, and cervical lymph node metastases may be present

Among primary parotid malignant tumours, mucoepidermoid carcinoma is the most common, followed by adenocystic carcinoma. The latter is notorious for its proclivity for perineural invasion and metastatic potential so surgery is normally supported by adjuvant radiotherapy to gain local control of the disease

INVESTIGATIONS

The initial imaging modality of choice is ultrasound as it demonstrates if the lump is intrinsic to the parotid or not. It also facilitates accurate sampling of the lesion by FNAC or True-Cut biopsy. Subsequently, CT and MRI are the most useful imaging techniques . Open surgical biopsy is

Classification of salivary gland tumours (simplified).

Type	Subgroup	Common examples
1 Adenoma	• Pleomorphic • Monomorphic	• Pleomorphic adenoma • Adenolymphoma (Warthin's tumour)
II Carcinoma	low grade	• Acinic cell carcinoma • Adenoid cystic carcinoma • Low-grade mucoepidermoid carcinoma
	High grade	• Adenocarcinoma • Squamous cell carcinoma • High-grade mucoepidermoid carcinoma
III Non epithelial tumours		Haemangioma. lymphangioma
IV lymphomas	• Primary lymphomas • Secondary lymphomas	• Non-Hodgkin's lymphomas • Lymphomas in Sjogren's syndrome
V Secondary tumours	Local Distant	Tumours of the head and neck especially Skin and bronchus
VI Unclassified tumours		
VII Tumour like lesions	Solid lesions	• Benign lymphoepithelial lesion • Adenomatoid hyperplasia
	Cystic lesions	Salivary gland cysts

contraindicated unless evidence of gross malignancy is present, and preoperative histological diagnosis is required as a prelude to radical parotidectomy

no enucleation even if a benign lesion is suspected

TREATMENT OF PAROTID TUMOR

A superficial parotidectomy is when the part of the gland superficial to the facial nerve is removed. A deep lobe parotidectomy is when the part of the gland beneath the nerve is removed and total parotidectomy is when both are dissected and removed.

SUPERFICIAL PAROTIDECTOMY

Superficial parotidectomy is the most common procedure for parotid gland pathology

The aim of superficial parotidectomy is to remove the tumour with a cuff of normal surrounding tissue

Low-grade and low stage malignant tumours treated by superficial parotidectomy

Steps

1) INCISION AND DEVELOPMENT OF A SKIN FLAP (figure 16 a)
2) MOBILISATION OF THE GLAND (figure 16 b)
3) LOCATION OF THE FACIAL NERVE TRUNK (figure 16 c and d)
4) DISSECTION OF THE GLAND OFF THE FACIAL NERVE (figure 16 e)
5) CLOSURE (figure 16 f)

EXTRACAPSULAR DISSECTION

Extracapsular dissection is now an established alternative to parotidectomy for benign parotid gland tumours

RADICAL PAROTIDECTOMY

Radical parotidectomy is performed for patients in whom there is clear histological evidence of a high-grade malignant tumour (e.g. squamous cell carcinoma) with invasion of facial nerve. Radical parotidectomy involves removal of all parotid gland tissue and elective division of the facial nerve, usually through the main trunk (Figure 49.26). The surgery inevitably removes the ipsilateral masseter muscle and may also require simultaneous neck dissection, particularly where there is clinical, radiological and cytological evidence of lymph node metastases in the ipsilateral neck. When indicated, facial nerve can be repaired using cable grafts (interpositional greater auricular or sural nerve grafts).

Complications of parotid gland surgery include:

- haematoma formation;
- infection;
- deformity: unsightly scar and retromandibular hollowing;
- temporary facial nerve weakness;
- transection of the facial nerve and permanent facial weakness;
- sialocele;
- facial numbness;

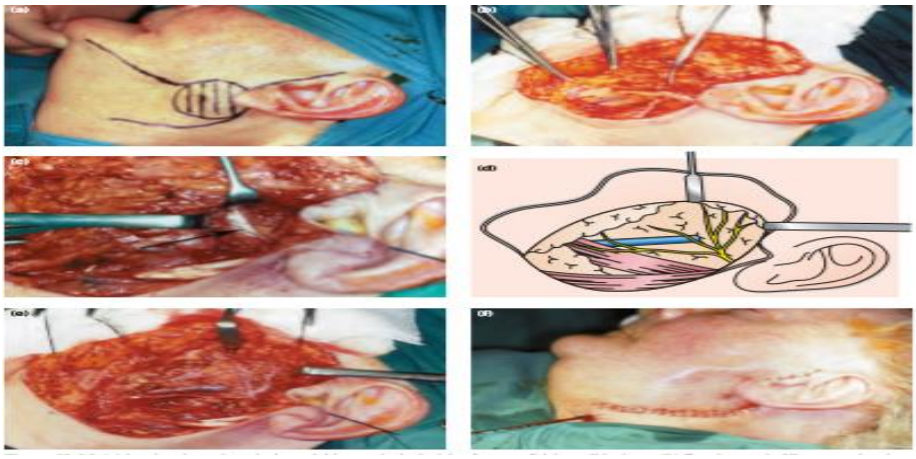


Figure 50

- permanent numbness of the ear lobe associated with great auricular nerve transection;
- Frey's syndrome

FREY'S SYNDROME

Frey's syndrome (gustatory sweating) is now considered an inevitable consequence of parotidectomy unless preventive measures are taken (see below). It results from damage to the autonomic innervation of the salivary gland with inappropriate regeneration of the postganglionic parasympathetic nerve fibres of the auriculotemporal nerve that aberrantly stimulate the sweat glands of the overlying skin. The clinical features include sweating and erythema (flushing) over the region of surgical excision of the parotid gland as a consequence of autonomic stimulation of salivation by the smellor taste of food. The symptoms are entirely variable and are clinically demonstrated by a starch iodine test. This involves painting the affected area with iodine, which is allowed to dry before applying dry starch, which turns blue on exposure to iodine in the presence of sweat. Sweating is stimulated by salivary stimulation. The management of Frey's syndrome involves the prevention as well as the management of established symptoms.

PREVENTION

The incidence of Frey's syndrome is minimal when extracapsular dissection is performed as the parotid fascia is primarily repaired and communication between denuded parotid parenchyma and subcutis sealed off. There are a number of quite invasive techniques described to prevent Frey's syndrome following parotidectomy. These include:

- sternomastoid muscle flap;
- temporalis fascial flap;
- insertion of artificial membranes between the skin and the parotid bed.

All these methods replace the barrier between the skin and the parotid bed to minimise inappropriate regeneration of autonomic nerve fibres.

MANAGEMENT OF ESTABLISHED FREY'S SYNDROME

Methods of managing Frey's syndrome include:

- antiperspirants, usually containing aluminium chloride;
- denervation by tympanic neurectomy;
- the injection of botulinum toxin into the affected skin. The last is the most effective and can be performed as an out-patient.

GRANULOMATOUS SIALADENITIS

This is a group of rare conditions that affect the salivary glands producing a variety of signs and symptoms, particularly painless swellings of the parotid and/or submandibular glands. Systemic upset is variable. These include the following:

- Mycobacterial infection. Tuberculosis and non-tuberculous sialadenitis typically present as a tumour-like swelling of the salivary gland. There is little pain and no fever. Preoperative investigations may be of some help, and the diagnosis is only confirmed when the swelling has been excised by either submandibular gland excision or formal parotidectomy.
- Sarcoidosis. Sarcoidosis can affect the salivary tissue and presents with persistent salivary gland swelling that may be associated with xerostomia. Occasionally, the patient will present with a localised tumour-like swelling in one salivary gland, more commonly the parotid – the so called sarcoid pseudotumour. In such circumstances, the diagnosis is only likely to be made following surgical excision for a presumed neoplasm. Heerfordt's syndrome is sarcoidosis that involves parotid swelling, anterior uveitis, facial palsy and fever
- . Other. These include cat scratch disease, toxoplasmosis, syphilis, deep mycoses and granulomatosis with polyangiitis (previously Wegener's granulomatosis), allergic sialadenitis and sialadenitis associated with radiotherapy of the head and neck

TUMOUR-LIKE LESIONS

There is a group of pathological conditions that affect the salivary glands and which do not fall into any particular classification or category and are often difficult to diagnose. These include such conditions as sialadenosis, adenomatoid hyperplasia and multifocal monomorphic adenomatosis.

SIALADENOSIS

Sialadenosis (sialosis) is used to describe non-inflammatory swelling particularly affecting the parotid gland. It usually occurs in association with a variety of conditions including diabetes mellitus, alcoholism, other endocrine diseases, pregnancy, drugs, bulimia and other eating disorders, and idiopathic diseases.

Most patients present between 40 and 70 years of age, and the salivary swellings are soft and often symmetrical . When the parotid glands are affected, patients may complain of a hamster-like appearance. Drug-induced sialosis is particularly common with sympathomimetic drugs. In many patients, no underlying disorder can be identified. Severe and prolonged malnutrition, as seen in eating disorders, produces sialadenosis by a process of hypertrophy to compensate for swings in acid balance. The pathological mechanism of sialadenosis can be associated with a process of neuropathy, which interferes with salivary gland function and subsequent acinar cell atrophy. This may be the case in diabetes mellitus, where autonomic neuropathy is a recognised complication as well as drug-induced sialosis.

The treatment of sialosis is unsatisfactory, but treatment is aimed at correction of the underlying disorder. Drug- associated sialadenosis may regress when the drug responsible is withdrawn..

ADENOMATOID HYPERPLASIA

It involves idiopathic hyperplasia of the acinar cells. It is extremely rare condition .

DEGENERATIVE CONDITIONS

SJÖGREN'S SYNDROME

Sjögren's syndrome is an autoimmune condition causing progressive destruction of salivary and lacrimal glands. Primary Sjögren's syndrome differs from secondary Sjögren's syndrome in that xerostomia and keratoconjunctivitis sicca occur without an associated connective tissue disorder or other autoimmune condition (rhematoid arthritis, systemic lupous erythematosus, scleroderma, polymyositis, thyroiditis). However, the symptoms are often more severe, and the incidence of lymphomatous transformation (see below) in the primary group is higher than that in the secondary group (Table).

Degenerative disorders.	
Primary Sjdgren's syndrome	More severe xerostomia
	Widespread exocrine gland dysfunction
	No connective tissue disorder
Secondary Sjogren's syndrome	M:F: 1:10
	Middle age
	Underlying connective tissue disorder
Benign lymphoepithelial lesion	20% develop lymphoma
	Diffuse parotid swelling 20% bilateral

The incidence of Sjogren's syndrome is 0.5–2% of the population. Females are affected more than males in the ratio 10:1. Occasionally, there is enlargement of the salivary glands, more commonly the parotid rather than the submandibular glands. The glands are occasionally painful, and the patient rarely develops a bacterial sialadenitis due to ascending infection from the associated xerostomia.

The characteristic pathological feature of Sjögren's syndrome is progressive lymphocytic infiltration, acinar cell destruction and proliferation of duct epithelium in all salivary and lacrimal gland tissue. The diagnosis is based on the history as no single laboratory investigation is pathognomonic of either primary or secondary Sjögren's syndrome .

MANAGEMENT

Management of Sjögren's syndrome remains symptomatic. No known treatment modifies or improves the xerostomia or keratoconjunctivitis sicca. An ophthalmological assessment is important, and artificial tears are essential to preserve corneal function. For dry mouth, various artificial salivary substitutes are available, but patients often consume large volumes of water, carrying a bottle of water with them at all times. In the dentate patient, the use of salivary substitutes with fluoride is important to counter the risk of accelerating dental caries. Other oral complications include oral candidosis and accelerated periodontal disease

COMPLICATIONS OF SJÖGREN'S SYNDROME

There is an increased incidence of developing lymphoma (most commonly non-Hodgkin's B-cell lymphoma) in patients with Sjögren's syndrome. The risk is highest within the primary group, and the onset of lymphoma is heralded by immunological change within the blood. The incidence of lymphoma in patients with Sjogren's syndrome is 4.3% (18.9 times higher than in the general population). Enlarged and painful parotid glands raise the prospect of MALT (mucosa associated lymphoid tissue) lymphoma .

XEROSTOMIA

Xerostomia is a common symptom in many aspects of medical practice. Normal salivary flows decrease with age in both men and women, although many patients with xerostomia are postmenopausal women who also complain of a burning tongue or mouth. Common causes of xerostomia are:

1- chronic anxiety states and depression;
2- dehydration
3- anticholinergic drugs, especially antidepressants;
4- salivary gland disorders – Sjögren's syndrome. Ascending parotitis is an occasional complication of xerostomia and is managed with antibiotics and increased fluid intake;
5- radiotherapy to the head and neck.

SIALORRHOEA

Certain drugs and oral infection produce a transient increase in salivary flow rates. In healthy individuals, excess salivation is rarely symptomatic as excess saliva is swallowed spontaneously. Uncontrolled drooling is usually seen in the presence of normal salivary production in children with mental and physical handicap, most notably cerebral palsy.

MANAGEMENT

Sialorrhoea can be managed medically with antisialogogues or with intraparenchymal botulinum toxin injection Uncontrollable drooling is managed surgically, and many operations are available. Surgical options include:

● bilateral submandibular duct repositioning and simultaneous sublingual gland excision;

● bilateral submandibular gland excision;

● transposition of the parotid ducts and simultaneous submandibular gland excision.

Most resting salivary gland flow arises from the submandibular glands, and surgery should be focused on this gland to control uncontrolled sialorrhoea

DISEASES OF THE LUNG AND PLEURA

ANATOMY OF THE THORAX

The bony thoracic cage is formed by the 12 thoracic vertebrae at the back, the sternum in front and 12 pairs of ribs in between. The upper seven pairs of ribs articulate anteriorly direct with the sternum through their respective costal cartilages. The costal cartilage of ribs 8, 9 and 10 articulates with that of the rib above. These ribs with the xiphisternum form the lower costal margin. The lowermost point of the thoracic cage is the 10th costal cartilage. The space between two adjacent ribs is known as the intercostal space. Thus, there are 11 intercostal spaces on each side. The junction between the manubrium and the body of the sternum is the sternal angle. The second costal cartilage articulates at the sternal angle (Figure 1).

The thoracic cavity contains on either side the right and left lungs surrounded by the pleural cavities and the mediastinum in between. The right lung is subdivided into superior, middle and inferior lobes by an oblique fissure and a horizontal fissure.

The left lung usually has only two lobes, a superior and an inferior with an oblique fissure in between. The root of the lung connects the lung to the mediastinum and consists of, anterior to posterior, two pulmonary veins, the pulmonary artery and the bronchus. The area where these structures enter the lung is the hilum of the lung. These structures are enclosed in a sleeve of pleura which loosely hangs down in its lower part as the pulmonary ligament.

The lung is surrounded by the pleural cavity, the potential space between the two layers of pleura. The outer parietal layer of pleura lines the thoracic cavity and the inner visceral layer closely fits on to the surface of the lung. The two layers become continuous with each other at the root of the lung.

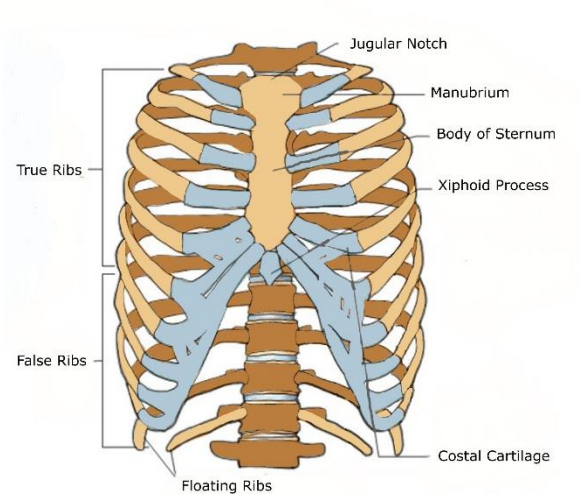


Figure 51 anatomy of the thoracic cage

The parietal pleura lining the diaphragm is known as the diaphragmatic pleura and that lining the mediastinum as the mediastinal pleura.

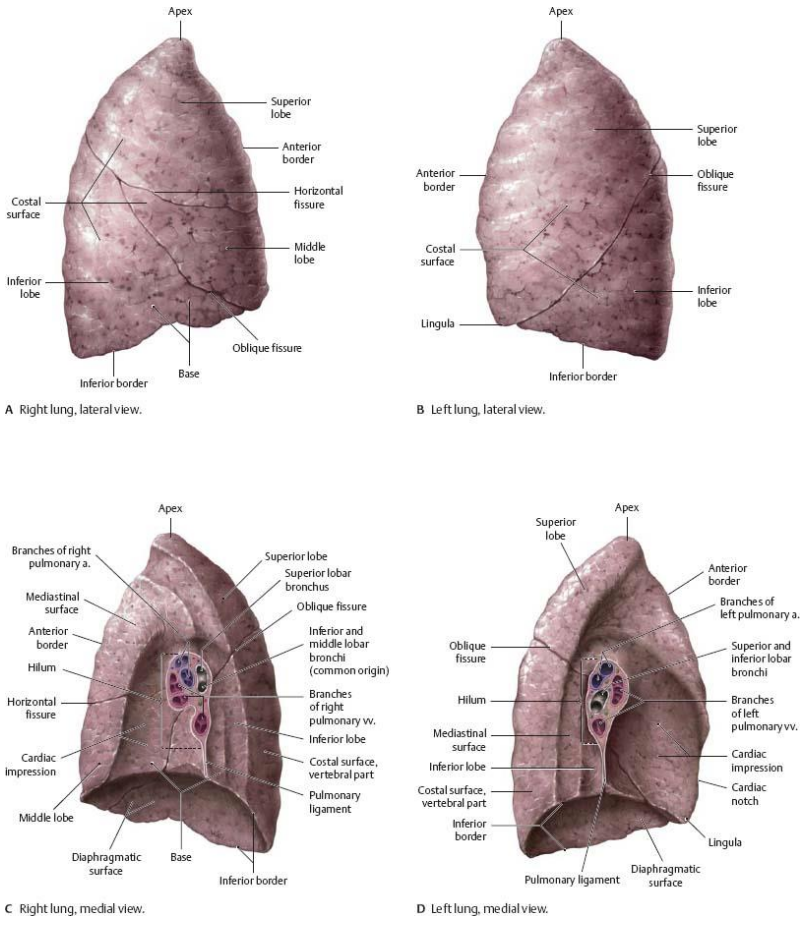


Figure 52 anatomy of the lung

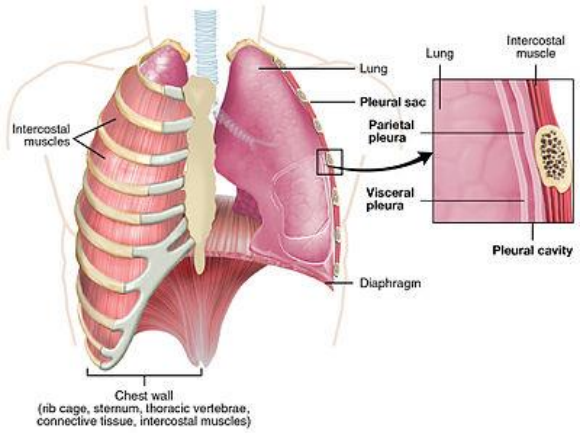


Figure 53 pleura

CONGENITAL CHEST WALL DEFORMITIES

Pectus excavatum (sternal retraction or funnel shaped chest) is due to abnormal growth of the sternum and ribs (figure 1) and is the most common type of congenital chest wall abnormality (90%). It represents inward growth of the sternum and surrounding ribs. Pectus excavatum occurs in an estimated 1 in 300-400 births, with male predominance (male-to-female ratio of 3:1). There is often a mild degree of scoliosis present and patients characteristically stand with a hunched posture. Pectus excavatum is often present in Marfan's syndrome Often, however, "patients with these deformities present in their early teenage years. Symptoms range from exercise intolerance to atypical chest pain (musculoskeletal in origin).

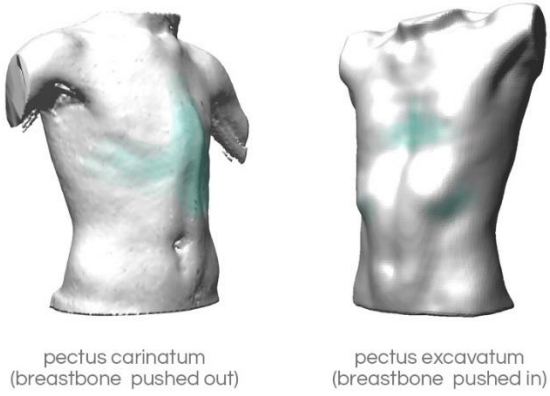


Figure 54

At this time, the deformity is exacerbated by accelerated growth and the individual becomes extremely sensitive about his or her appearance.

Correction is only indicated when the patient's quality of life is clearly impaired because of appearance. Ravitch operation is the most commonly performed procedure (its details are unnecessary).

Pectus carniatum (also known as pigeon chest) is the opposite of pectus excavatum and represents forward protrusion of the chest. It is less common and ma¬naged surgically when needed.

EMPYEMA

Empyema is defined as a suppurative infection within the pleural cavity.

ETIOLOGY

Empyema commonly follows pneumonia due to secondary infection of a reactive parapneumonic effusion. Other causes of empyema are shown in the table below. Commonly implicated organisms include streptococci, pneumococci, staphylococci, gram negative anaerobes.

Common causes of empyema

Pulmonary infections

- **Pneumonia**
- **Lung abscess**
- **Bronchiectasis**
- **Tuberculosis**

Trauma and postoperative

- **Chest injuries**
- **Esophageal perforations**
- **Leaking intrathoracic anastomosis**

Subdiaphragmatic infections

- **Subphrenic abscess**
- **Hepatic abscess**

PATHOGENESIS

In the initial phase (acute or exudative phase), the infected fluid is thin and may be completely evacuated by a low intercostal drain (antibiotics may be all that is required but additional drain is preferable). The empyema quickly becomes thick and loculated as a result of the deposition of fibrin (transitional or fibrinopurulent phase), and at this stage formal surgical drainage is required. The organising (chronic) phase causes the lung to be trapped by

a thick peel or 'cortex' for which surgical management may be required.

CLINICAL FEATURES

Typical symptoms of pleural include fever, chest pain, sweating and dyspnea. Clubbing may be present in cases of a (chronic phase). There is a dull percussion note and reduced breath sounds on the affected side of the chest.

DIAGNOSIS

Diagnosis is confirmed by thoracentesis; frank pus or merely cloudy fluid may be aspirated from the pleural space. The pleural fluid typically has a leucocytosis, low pH (<7.20), low glucose (<40 mg/dL), a high LDH (lactate dehydrogenase < 1000 IU/L), elevated protein and may contain infectious organisms.

Air-fluid levels may appear on supine and erect chest films and CT scan. The collection is typically placed posteriorly towards the base of the pleural cavity and causes a D-shaped shadow on lateral chest films (figure 5).

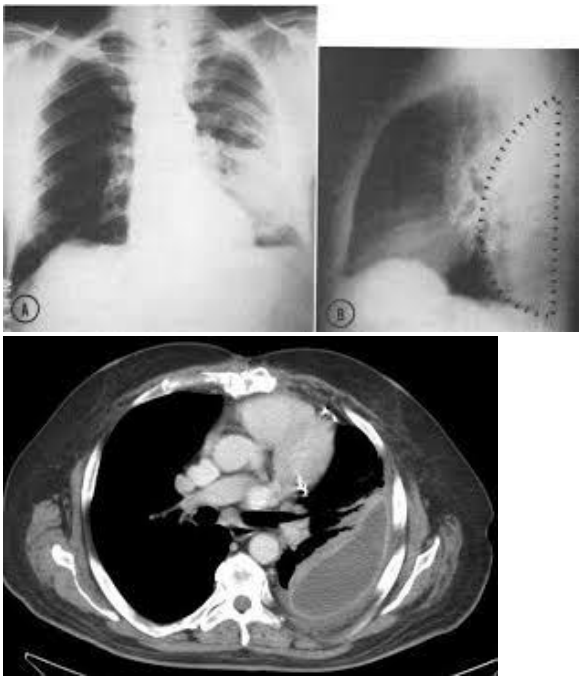


Figure 55 radiological studies A- Lateral X-ray of empyema. This film shows the D-shaped outline of a typical empyema. Infected fluid collections can, however, occur anywhere within the chest. B-PA chest X-ray of a patient with empyema, showing opacity in the lower left zone. C- CT scan of a patient showing a posterior collection of pus in the left lung.

MANAGEMENT

An acute empyema is treated with appropriate antibiotics along with repeated drainage. A chest tube may be inserted. Large bore chest tubes are preferred so that clogging with thick pus is minimized. To improve the chest tube drainage, fibrinolytics (streptokinase and urokinase) and DNA enzyme can be given intrapleural through the chest tube to break, the fibrinous septation and to reduce the pus viscosity.

Open surgical drainage is achieved by excising a 2 cm segment of rib over the lowest part of the empyema and suctioning and curetting the cavity clean, Open thoracotomy allows the fibrous cavity to be excised and any cortex over the lung removed (decortications). This returns more lung function to the patient and avoids open drainage, so that recovery is more rapid.

A postpneumonectomy empyema can result from a bronchial stump dehiscence with contamination of the pneumonectomy space. Increasing air in the pneumonectomy space on CXR is often diagnostic. Bronchopleural fistula has a high mortality rate and is managed via drainage of the space, antibiotics, and surgical repair of the fistula.

LUNG ABSCESS

A lung abscess is a Focus of infection within the lung associated with parenchymal necrosis usually with capitations. showing a posterior collection of pus in the left lung. B-PA chest X-ray of a patient with empyema, showing opacity in the lower left zone. C- Lateral X-ray of empyema. This film shows the D-shaped outline of a typical empyema. Infected fluid collections can, however, occur anywhere within the chest.

ETIOLOGY

The causes of lung abscess are shown in the table below.

causes of a lung abscess
Primary necrotizing pneumonia **Aerobic infection** • *Staphylococcus aureus* • *Klebsiella Pneumonia* and other gram negatives. • Mycobacterium *(M. tuberculosis* and atymical myocobacteria) **Anaerobic infection** • Bacteroides *(B.fragilis, B.melaninogenicus)* • Fusobacterium species. • Actinomyses. **Parasitic infection** • *Entamoeba histolytica* • Echinococcus (*E. granulosus, E. multilocularis)*
Aspiration pneumonia
Bronchial obstruction • Neoplasm • Foreign body
Complication of systemic sepsis • Septic pulmonary emboli • Seeding of pulmonary infarct**.**
Complication of pulmonary trauma • Infection if hematoma or contusion. • contaminated foreign body or penetrating injury.
Direct extension from extra parenchymal infection • Pleural empyema. • mediastinal, hepatic, subphrenic abscess.

Lung abscesses occur more commonly in the elderly as they are more likely to develop pneumonia or suffer from dysphagia and aspiration, and to have poor oral health. The onset may be gradual in anaerobic infections but can be quite rapid with aerobic bacteria

CLINICAL FEATURES

The manifestations of a lung abscess include a promi¬nent cough that usually yields copious amounts of foul-smelling, purulent, or sanguineous sputum; occa¬sionally, hemoptysis occurs. Patients usually have high fever, malaise, and sweating and weight loss. The fever is often swinging and associated with tachycardia

Clubbing of the fingers is seen occasionally. On examination of chest there will be features of con¬solidation such as localized dullness on percussion and bronchial breath sound

DIAGNOSIS

The diagnosis is usually established by chest X-ray, which shows air fluid level (figure 6). CT scan is rarely needed. Fiber optic bronchoscopy is often performed to exclude obstructive lesion; it also helps in bronchial drainage of pus. Bronchoscopy aspirates can also be cultured.

MANAGEMENT

Lung abscesses are usually treated with antibiotics alone (at least 8 weeks until symptoms improve) but occasionally a cavity requires drainage. Drains are usually placed percutaneous under ultrasound or CT guidance. Drainage by bronchoscopy can also be achieved.

Most acute abscesses resolve with appropriate antibiotic therapy and postural drainage; thus, surgery is avoided. Indications for surgery are shown in the table below.

Indications for surgery in lung abscesses

- **Failed medical treatment.**
- **Abscess > 5 cm in diameter.**
- **Serious hemorrhage.**
- **Malignancy.**
- **Foreign body.**

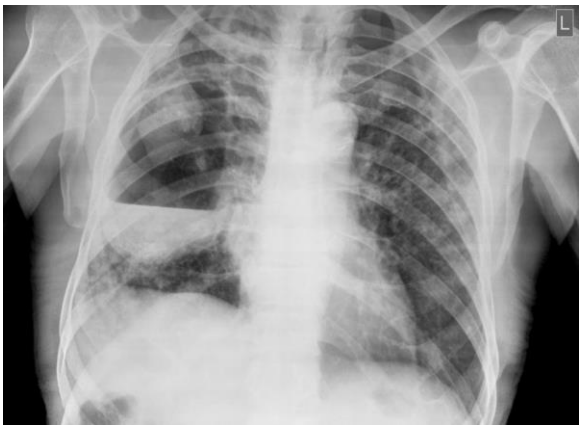


Figure 56 lung abscess in the right, note the air-fluid level

LUNG CANCER

Lung cancer is the 2nd most common cancer among male and female (it come after colorectal ca in male , breast ca in female). From the time of diagnosis with lung ca, 80% of patients are dead within 1 year and only 5% survive 5 years, **making lung cancer the most common cause of cancer death.**

Surgical resection has a limited role in curative treatment because at the time of presentation many cancers are locally advanced or widely disseminated and are beyond surgical cure (20–30% of cases are potentially surgically resectable).

Male to female ratio 2:1

Risk factors:

1. Cigarette smoking is undoubtedly the major risk factor for developing bronchial carcinoma and accounts for 85–95% of all cases. It increased risk 4-8x to have lung ca.
2. Urban air pollution.
3. Industrial exposure (workers in phosphate mines and petroleum industries, patients who exposed to asbestos fiber because they were think that asbestosis cause mesothelioma which is cancer of pleural cavity of lung).
4. Family component (breast , colon , lung , bladder) .

Types:

lung cancers are divided into small cell lung cancer and non-small cell lung cancer (NSCLC), which are seen in a ratio of about 1:4.

I. **Small cell lung cancers:** highly malignant, were known as **oat cell cancers** because of the packed nature of small dense cells. They are a type of neuroendocrine tumor (NET) because it's production of peptides (like Dopa decarboxylase, Adrenocorticotropic hormone ACTH, Gastrin releasing peptide, Creatinine kinase) go to target cells and bind to receptors to give effect of this hormones, it represents about 15-35% (median20%) of all lung cancers. They tend to metastasize early to lymph nodes and by blood-borne spread. The median survival is measured in months. The tumors are very responsive to chemotherapy but they are rarely, if ever, cured. Surgery is rarely offered unless in very limited stage disease (doctor Ali said that no role for surgery even stage 1 , it doesn't improve survival).

II. **Non-small cell lung cancer (N.S.C.L)**

1. Squamous cell carcinoma 35% (not highly malignant, closely related to cigarette smoking, affect base of lung, lymphatic spread more than hematogenous spread, sensitive to chemotherapy).

2. Adenocarcinoma 30-50% (it's the most common subtypes of all lung ca, hematogenous spread, it's chemo and radio resistant, best treatment by surgical excision).

a. Bronchioalveolar CA. (bilateral with mucus secretion, very aggressive)

b. Acinar adenocarcinoma.

c. Papillary adenocarcinoma.

d. Solid carcinoma with mucus formation.

3. Undifferentiated large cell carcinoma 2%.

4. Adenosquamous carcinoma 1%.

CLINICAL PRESENTATION

Asymptomatic 5%: silent because lung very elastic tissue, so if the tumor in the peripheral side of the lung it grow in size without affect function of lung ,in this cases tumor discover incidentally .

I. BRONCHOPULMONARY SYMPTOMS:

- dry cough 75%: most common symptom of lung ca due irritation of wall of bronchi and bronchioles this led to narrowing of lumen followed by cough. the cough will persist for 2-3 months and it refractory to antibiotics.
- hemoptysis 57%: bright fresh blood without clot, due to either erosion of small arterioles or venules in bronchial wall or due to sloughing of bronchi mucosa or if tumor present within

bronchi or bronchioles or main bronchus this lead to necrosis and bleeding .

- chest pain: if patient have only lung ca without METS, he will complain of mild chest pain dull and aching in nature or he feel like heaviness upon the chest relief by simple pain killer. If its become severe it's alarm >> indicate bone metastasis (ribs , thoracic vertebrae) .
- dyspnea
- febrile respiratory symptoms: if tumor present within bronchus, the lung parenchyma distal to narrowing will be ischemic and collapse follow by consolidation and mucous secretion and accumulation all of this lead to bacterial over growth >>>pneumonia >>fever, wheezing, stridor.

II. EXTRAPULMONARY INTRATHORACIC SYMPTOMS 15%.

- hoarseness of voice: Due to involve of left recurrent laryngeal nerve.
- superior vena cava syndrome: flushing face, congestion, engorged neck vein.
- sever chest pain: METS due to periosteum erosion.
- pain in the upper extremity (because of brachial plexus invasion).
- Horner's syndrome: miosis, ptosis, anhidrosis.
- dysphagia.
- pleural effusion: transudate from lung cancer, its bad sign mean malignancy >>stage 3=distant METS.
- phrenic nerve paralysis.

III. EXTRATHORACIC NONMETASTATIC SYMPTOMS 2%.

- Paraneoplastic manifestations. (B symptoms)
- Metabolic manifestations.
- Cushing's syndrome.
- ACTH production.
- Excessive Anti-diuretic hormone production.
- Hypercalcemia (indicate bone metastasis)
- Hypertrophic pulmonary osteoarthropathy.
- Clubbing of fingers.
- Thrombophlebitis: if the patient has migrate recurrent thrombophlebitis think about hidden malignancy (pancreas, breast) .

Recent National Institute for Health and Care Excellence (NICE) guidance on referral in patients with suspected lung cancer.

Symptoms and signs indicating urgent chest x-ray	Offer urgent chest x-ray to patients presenting with haemoptysis, or any of the following rt unexplained or present for more than 3 weeks:	Cough Chest/shoulder pain Dyspnoea Finger clubbing Signs suggesting metastases (for example, in brain, bone, liver or skin) Weight loss Chest signs Hoarseness Cervicai/supraciavicular lymphadenopathy
Symptoms and signs indicating urgent referral	Offer urgent referral to lung cancer MOT (usually the chest physician) while waiting for chest x-ray results If any of the following are present:	Persistent haemoptysis In a smoker or ex-smoker older than 40 years Signs of superior vena cava obstruction (swelling of the face and/or neck with fixed elevation of Jugular venous pressure) Stridor
	Offer urgent referral to lung cancer MDT (usually the chest physician) if:	A chest x-ray or CT scan suggests lung cancer (including pleural effusion and slowly resolving consolidation) or Chest x-ray is normal, but there is a high suspicion of lung cancer

IV. EXTRATHORACIC METASTATIC SYMPTOMS.

- Neurologic symptoms: brain MET: hemiplegia, hemiparesis or symptom of increase intracranial pressure as persistent vomiting, severe headache, diplopia. etc.
- Bone pain, pathological fracture: due to MET to ribs, thoracic and lumber vertebral body, femur, pelvis, humerus.
- Jaundice, ascites, abdominal mass: liver MET
- Non-specific symptoms.
- Wt. loss, weakness, anorexia, malaise.

METASTASIS

- Direct extension.
- Lymphatic metastasis.
- Hematogenous spread.

brain, liver, lungs, bone, kidney, pancreas, skin, subcutaneous tissue, bone, adrenal glands (50% of lung ca patients who die from lung ca have adrenal gland Met).

DIAGNOSIS

- CXR: the smallest mas can we see on CXR it's about 1 cm mean it was being there for 6 months and this mean that tumor has complete 3/4 of it's life cycle.

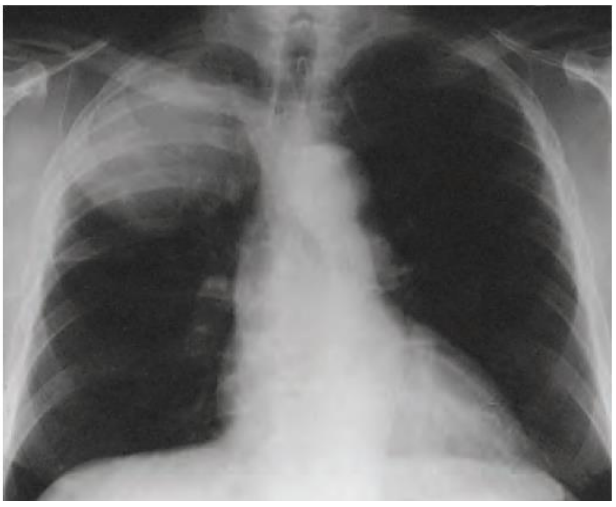


Figure 57 Chest x-ray of carcinoma of the lung. This patient has a large mass in the right upper lobe, causing Horner's syndrome.

- Sputum cytology +ve 45-90 %. (3 times –ve >> no tumor)
- Bronchial brushing & wash cytology.
- Pulmonary angiography- CT: for staging process
- Chest CT scan.
- MRI.
- Ultrasound.

- liver, adrenals, abdomen.

- EUC (electrolyte, urea, creatine) OR EUS (endoscopic US).

- EBUS (endobronchial US).

- PET scan: to stage and follow up.
- Invasive diagnostic procedures: bronchoscopy, percutaneous transthoracic needle aspiration: if tumor peripheral, video assessment thoracoscopy, mediastinoscopy, supraclavicular lymph node biopsy.

P.S: the diagnosis confirms by tissue biopsy.

Staging:

Summary: LUNG	
IX	Positive cytologx only
TI	≤3 cm
T1a	≤2 cm
T2b	>2-3 cm
T2	Main bronchus ^2 cm from carina, invades visceral pleura, partial atelectasis
T2a	>3- 5 cm
T2b	>5 cm-7 cm.
T3	>7 cm; chest wall, diaphragm, pericardium, mediastinal pleura, main bronchus <2 cm Irtiin carina, total atelectasis, separate nodule(s) in same lobe
T4	Mediastinum, heart, great vessels, carina, trachea, oesophagus, vertebra; separate tumour nodule(s) in a different ipsilateral lobe
N 1	Ipsilateral peribronchial, ipsilateral hilar
N2	Subcarinal, ipsilateral mediastinal
N3	Contralateral mediastinal or hilar, scalene or supraclavicular
M 1	Distant metastasis
M la	Separate tumour nodulc(s) in a contralateral lobe; pleural nodules or malignant pleural or pericardial effusion
M lb	Distant metastasis

TREATMENT:

-surgery

Stage 1,2 >>surgery indicate

Stage 3a >>operable but not curable

Stage 3b,4>> non operable, incurable

-chemotherapy

-radiothearpy

Survival table following operation for carcinoma of the bronchus.	
	(%)
Five-year survival according to presurgical staging	
Stage 1	56-67
Stage II	39-55
Stage Illa	23
Stage lllb	<10
Five-year survival according to cell type	
Squamous cell carcinoma	35-50
Adenocarcinoma	25-45
Adenosquamous carcinoma	20-35
Undifferentiated carcinoma	15-25
Small cell carcinoma	0-5

TUBE THORACOSTOMY

Tube thoracostomy (Intercostal intubation) is the insertion of a tube (chest tube, intercostal drain) into the pleural cavity to drain air, blood, bile, pus, or other fluids. Placement of a chest tube allows for continuous, large volume drainage until the underlying pathology can be more formally addressed.

INDICATIONS AND CONTRAINDICATIONS

The indications for tube thoracostomy are shown in the table below

Indications for tube thoracostomy
• **Pneumothorax**
Open or closed
Simple or tension
• **Hemothorax**
• **Hemopneumothorax**
• **Hydrothorax**
• **Chylothorax**
• **Empyema**
• **Pleural effusion**
• **Patients with penetrating chest wall injury who are intubated or about to be intubated**
• **Considered for those about to undergo air transport who are at risk for pneumothorax**

The need for emergent thoracotomy is an absolute contraindication to tube thoracostomy. Relative contraindications are shown in the table below.

Relative contraindications for tube thoracotomy
• **Coagulopathy** • **Pulmonary bullae** • **Pulmonary, pleural, thoracic or diaphragmatic adhesions** • **Loculated pleural effusion or empyema (loculation: fibrotic scar tissue may form in the pleural cavity)** • **Skin infection over the chest tube insertion site**

PROCEDURE

- Obtain informed consent from the patient or patients' representative.
- Before inserting a chest tube, the patient should be positioned supine or at a 45° angle. (Elevating the patient lessens the risk of diaphragm elevation and consequent misplacement of the chest tube into the abdominal space.) The arm on the affected side should be abducted and externally rotated, simulating a position in which the palm of the hand is behind the patient's head.
- To drain a pneumothorax, a size 8-14 Fr catheter is inserted. Drainage of an effusion, empyema or hemothorax requires a larger drain.
- Identify the fifth intercostal and the midaxillary line. The skin incision is made in between the midaxillary and anterior axillary lines over a rib that is below the intercostal level selected for chest tube insertion (usually between the 4^{th} and 5^{th} ribs). A "safe triangle". (figure 2) has been described as the preferred site of insertion. This is the triangle bordered by the anterior border of the latissimus dorsi (anterior to the mid-axillary line), the lateral border of the pectoralis major muscle, and a line superior to the horizontal level of the nipple. A surgical marker can be used to better delineate the anatomy.
- Meticulous attention to sterility throughout.
- Adequate local anaesthesia to include the pleura.
- Sharp dissection to cut only the skin.
- Blunt dissection with artery forceps (Kelly clamp) down through the muscle layers; these should only be the serratus anterior and the intercostals.

- A gloved finger is inserted into the pleural cavity (Upon entry into the pleural space, a rush of air or fluid should occur). This ensures the incision is correctly placed, prevents injury to other organs, and permits any adhesions or clots to be cleared (by 360 rotation inside the chest).

- insert the tube obliqly (posteriorly and superiorly) into the pelural space to prevent kinking of the tube.

- The drain should pass over the upper edge of the rib to avoid the neurovascular bundle that lies beneath the rib.

- An oblique tract, so that the skin incision and the hole in the parietal pleura do not overlie each other and the

- drain is in a short tunnel, which reduces the chance of entraining air.

- A drain for pneumothorax and haemothorax should aim towards the apex of the lung. A drain for pleural effusion or empyema should be nearer the base

- The retaining stitch should be secure but should not obliterate the drain.

- A vertical mattress suture is inserted for later wound closure. This is vital for pneumothorax management but should be omitted if the drain is for empyema (provided there is adherence of the pleura) because that tract should lie open.

- Connect the drain to an underwater seal device which functions as a one-way valve.

- After completion, check that the drain has achieved its objective and inserted in correct position by taking a chest radiograph

- It is preferable not to apply suction to the drain or clamp it. The danger is that the clamp may be applied for transport and forgotten. A bubbling drain should (almost) never be clamped. Remove the drain when it no longer has a function.

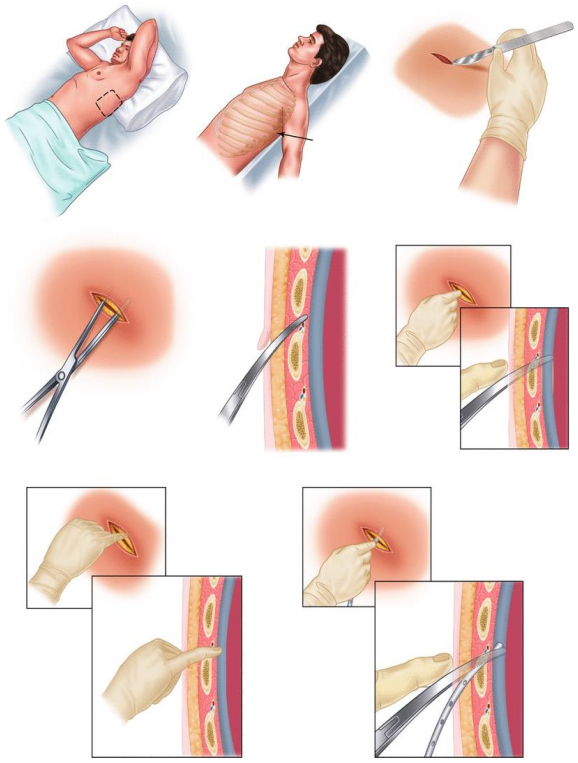


Figure 58 chest tube insertion

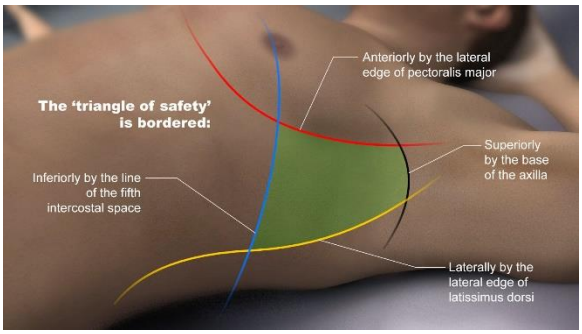


Figure 59 the safety triangle

COMPLICATIONS

Minor complications of thoracostomy tube placement such as unresolved/reaccumulating of pneumothorax or misplacement of the tube (too deep/kinked) are common and approach approximately 30%.

- **Improper placement;** Horizontal (over the diaphragm) placement is acceptable for hemothorax as most fluid accumulates at the lower zones; however, this should be repositioned for pneumothorax (should be more superior). Subcutaneous placement must be repositioned. When the tube

is placed too far into the chest (against the apical pleura), it should be retracted. Tubes placed into the abdominal space, should be removed.

- **Tube dislodgement**
- Bleeding; local bleeding usually responds to direct pressure. Hemothorax (lung vs intercostal artery injury), might require thoracotomy if it does not resolve spontaneously. Hemoperitoneum (liver or spleen injury) Requires emergent laparotomy.
- **Organ penetration;** (usually requires surgical repair). Stomach, colon, or diaphragm penetration
- occurs as a result of unrecognized diaphragmatic hernia .Lung penetration occurs as a result of pleural adhesions or use of a thoracostomy tube trocar.
- Empyema; Chest tube (foreign object) could introduce bacteria into the pleural space
- Retained pneumothorax or hemothorax; Might require insertion of a second chest tube.
- infection.
- subcutaneous emphysema.
- nerve injury.

PNEUMOTHO-RAX

Pneumothorax occurs when air enters the potential space between the visceral and parietal pleura through either an external chest wound (open) or an internal air leak (closed).

ETIOLOGY

Spontaneous pneumothorax arises in the absence of trauma. It is described as primary or secondary. Primary pneumothorax typically occurs in young (15-35 years) individuals with essentially normal lungs apart from a few apical bullae or sub-pleural blebs. Secondary pneumothorax develops in elderly patients (55-75 years) with a background of emphysema and chronic obstructive pulmonary disease. It is caused by rupture of a large bulla. The causes are shown in the table below.

Causes of secondary pneumothorax

- **Bullous disease - including COPD**
- **Cystic fibrosis**
- **Spontaneous rupture of the esophagus**
- **Marfan's syndrome**
- ***Pneumocystis carinii*- especially in AIDS patients**
- **Metastatic cancer - especially sarcoma**
- **Pneumonia with lung abscess**
- **Catamenial**
- **Asthma - secondary to mucous plugging**
- **Lung cancer**

Acquired pneumothroax is the result of traumatic rupture to the lung and/or chest wall. The causes are classified and shown in the table below.

Causes of acquired pneumothorax

iatrogenic

- **Subclavian- percutaneous- catheterization (Central lines, Pacemaker insertion)**
- **Transthoracic needle biopsy**
- **Transbronchial lung biopsy**
- **Thoracocentesis**
- **Chest tube malfunction**
- **Following laparascopic surgery**

Barotrauma

Traumatic

Blunt trauma
- **Motor**
- **vehicle accidents**
- **Falls**

Penetrating trauma
- **Gun shot wounds**
- **Stab wounds**

PATHOPHYSIOLOGY

When the pressure inside a pneumothorax is static, the condition is called a simple pneumothorax and although this condition requires treatment, it is not usually serious. In contrast, a tension pneumothorax is always life-threatening. In these patients, the laceration (wither from the chest or the lung) acts as a one-way valve, admitting inspired air into the pleural cavity with each inspiration but closing during expiration. The consequence is a progressive build-up of air with rising pleural pressure on the affected side. The result is total lung collapse and a shift of the mediastinum, including the heart and great vessels, to the contralateral side. Thus, in addition to respiratory distress and hypoxemia, the patient may develop cardiovascular collapse (obstructive shock).

CLINICAL FEATURES

The symptoms of pneumothorax are pleuritic chest pain, anxiety, dyspnea and cyanosis. On examination, there is usually an apparent chest wound in acquired pneumothorax. The trachea may be deviated (especially in tension pneumothorax). Percussion of the chest wall reveals hyperresonance and on auscultation air entry is diminished or absent.

DIAGNOSIS

Along with clinical features, the diagnosis is confirmed by a chest X-ray. Sharp delineation of visceral pleural by dense pleural space, mediastinal shift to opposite side (especially in tension pneumothorax) and air-fluid level in pleural space on erect chest radiograph (in cases of associated blood or fluid).

MANAGEMENT

The treatment of a patient with tension pneumothorax must be immediate. If the patient is very distressed, a large-bore hypodermic needle is inserted into the second or third interspace anteriorly to decompress the pleural cavity until the equipment for underwater seal drainage becomes available.

The principle of underwater seal drainage (figure 1) is that a liquid trap is interposed between the tube exiting the pleural cavity and the atmosphere. If the pressure in the pleural cavity is greater than atmospheric pressure, air, fluid or blood will drain out through the water-immersed tube. On the other hand, when pleural pressure becomes negative, atmospheric air is prevented from being sucked into the pleural space by the water level. The underwater seal bottle or disposable equivalent (such as Pleurivac) must always be kept at a lower level than the chest, and during changes or emptying of the bottle the exit tubing from the chest cavity must be clamped.

For treatment of a pneumothorax, the chest drain is inserted into the apex of the pleural cavity (apical drain). A basal drain is necessary for drainage of blood or fluid. This is inserted posterolaterally through the fifth interspace. Patients with hemopneumothorax require both apical and basal chest drains.

The chest drain is maintained until there is full lung expansion and any blood or fluid has drained away. A persistent air leak is easily recognized as bubbling through the water of the underwater seal drain. Lung expansion is signified by cessation of movement of the fluid column but this must be confirmed by chest X-ray. The tube is then clamped for several hours and if repeat radiography shows that the lung remains expanded, the drain is removed and the chest wound sutured and dressed.

In case of spontaneous pneumothorax. If the lung does not expand or the pneumothorax is recurrent, surgical treatment designed to obliterate the bullae and excise or abrade the parietal pleura is required. Pleurectomy or pleural abrasion is undertaken to encourage the formation of adhesions between the lung surface and the chest wall. This is now performed via the thoracoscopic approach.

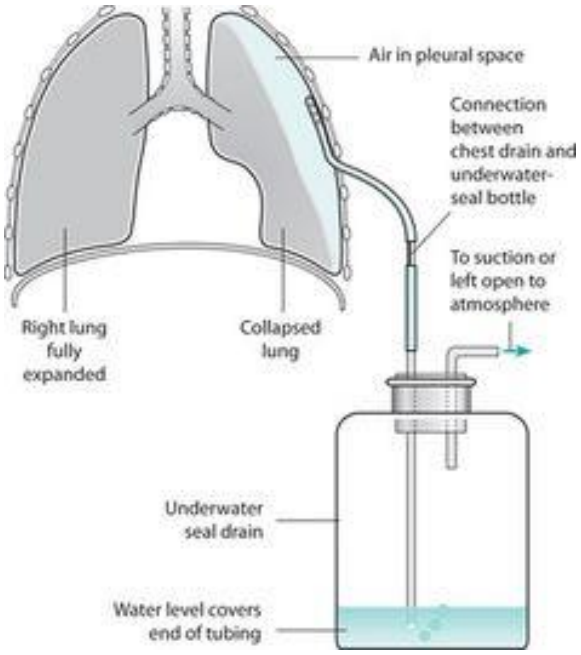


Figure 60 underwater seal

AORTIC DISSECTION

PATHOLOGY AND CLASSIFICATION

Aortic dissection, the most common catastrophic event involving the aorta, is a progressive separation of the aortic wall layers that usually occurs after a tear forms in the intima and inner media. As the separation of the layers of the media propagates, at least two channels form (Fig. 22-17): the original lumen, which remains lined by the intima and which is called the true lumen, and the newly formed channel within the layers of the media, which is called the false lumen. The dissecting membrane separates the true and false lumens. Additional tears in the dissecting membrane that allow communication between the two channels are called re-entry sites. Although the separation of layers primarily progresses distally along the length of the aorta, it can also proceed in a proximal direction; this process often is referred to as proximal extension or retrograde dissection

CAUSES AND RISK FACTORS

any condition that weakens the aortic wall increases the risk of aortic dissection. Common general cardiovascular risk factors, such as smoking, hypertension, atherosclerosis, and hypercholesterolemia.

connective tissue disorders, aortitis, bicuspid aortic valve, or preexisting medial degenerative disease

Aortic injury during cardiac catheterization, surgery, or endovascular aortic repair is a common cause of iatrogenic dissection.

cocaine and amphetamine abuse.

emotional stress or extreme physical exertion such as during weightlifting

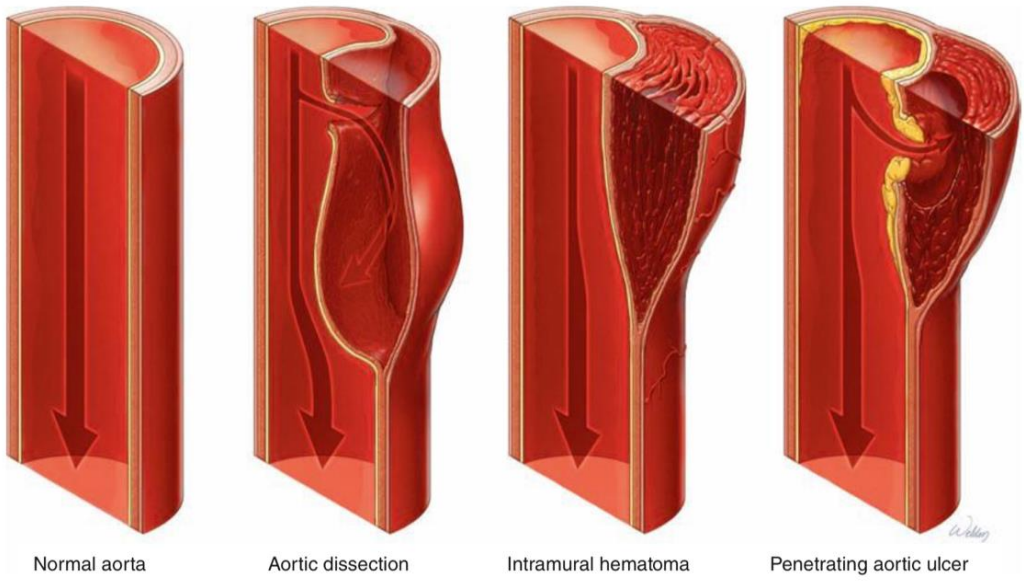


Figure 61 Illustration of longitudinal sections of the aortic wall and lumen. Blood flows freely downstream in normal aortic tissue. In classic aortic dissection, blood entering the media through a tear creates a false channel in the wall. Intramural hematomas arise when hemorrhage from the vasa vasorum causes blood to collect within the media; the intima is intact. Penetrating aortic ulcers are deep atherosclerotic lesions that burrow into the aortic wall and allow blood to enter the media. In each of these conditions, the outer aortic wall is severely weakened and prone to rupture.

CLASSIFICATIONS

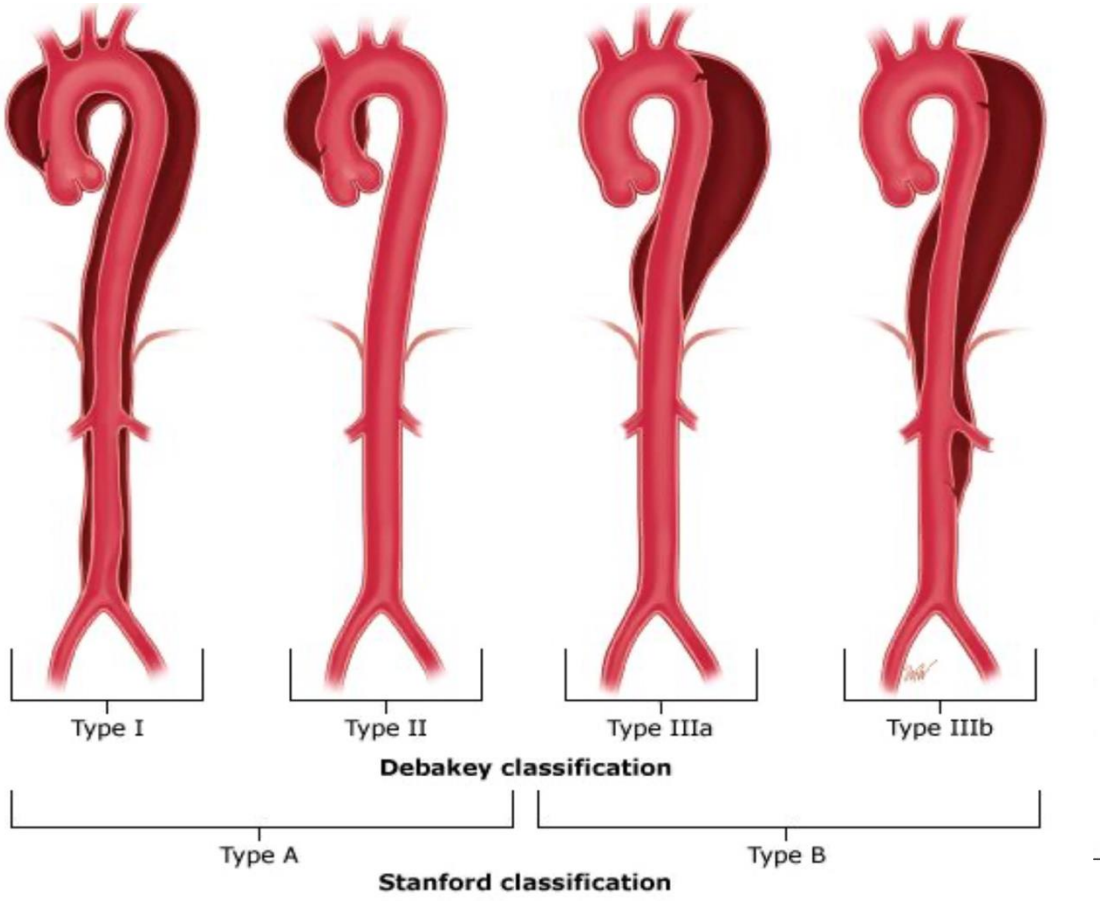


Figure 62

CLINICAL MANIFESTATIONS

The onset of dissection often is associated with severe chest or back pain, classically described as "tearing," that migrates distally as the dissection progresses along the length of the aorta. The location of the pain often indicates which aortic segments are involved. Pain in the anterior chest suggests involvement of the ascending aorta, whereas pain in the back and abdomen generally indicates involvement of the descending and thoracoabdominal aorta.

Discrepancy between the extremities in pulse, blood pressure, or both is the classic physical finding in patients with aortic dissection. Involvement of the aortic arch often creates differences between the right and left arms, whereas descending aortic dissection often causes differences between the upper and lower extremities.

Potential complications of dissection of the aorta (and involved secondary arteries) may include cardiac ischemia (coronary artery) or tamponade, stroke (brachiocephalic arteries), paraplegia or paraparesis (intercostal arteries), mesenteric ischemia (superior mesenteric artery), kidney failure (renal arteries), and limb ischemia or loss of motor function (brachial or femoral arteries).

Ascending aortic dissection can directly injure the aortic valve, causing regurgitation

DIAGNOSTIC EVALUATION

laboratory studies are of little help in diagnosing acute aortic dissection. There has been continued interest in using D-dimer level to aid in making this diagnosis.144 Several reports indicate that D-dimer is an extremely sensitive indicator of acute

aortic dissection; elevated levels are found in approximately 97% of affected patients.

Normal ECGs and serum marker levels in patients with acute chest pain should raise suspicion about the possibility of aortic dissection.

CXRs may show a widened mediastinum or abnormal aortic contour, up to 16% of patients with dissection have a normal-appearing CXR

Once the diagnosis of dissection is considered, the thoracic aorta should be imaged with CT, MRA, or echocardiography.

CT SCAN FINDING

The classic diagnostic feature is a double-lumen aorta

In addition, CT scans provide essential information about the segments of the aorta involved; the acuity of the dissection; aortic dilatation, including the presence of preexisting degenerative aneurysms; and the development of threatening sequelae, including pericardial effusion, early aortic rupture, and branch vessel compromise. Although MRA also provides excellent imaging (with both a sensitivity and specificity of 98%), the MR suite is not well suited for critically ill patients. In patients who cannot undergo contrast- enhanced CT or MRA, transthoracic echocardiography can be used to establish the diagnosis.

Transesophageal echocardiography (TEE) is excellent for detecting dissection, aneurysm, and IMH in the ascending aorta. In appropriate hands, TEE has a demonstrated sensitivity and specificity as high as 98% and 95%, respectively.149 Furthermore, TEE offers important information about ventricular function and aortic valve competency. Finally, TEE is the diagnostic modality of choice for hemodynamically unstable patients in whom the diagnosis of ascending dissection is suspected; ideally, these patients should be taken to the operating room, where the TEE can be performed and, if the TEE is confirmatory, surgery can be started immediately.

TREATMENT

The initial management strategy, commonly described as anti-impulse therapy or blood pressure control, focuses on reducing aortic wall stress, the force of left ventricular ejection, chronotropy, and the rate of change in blood pressure

The drugs initially used to accomplish these goals include IV beta-adrenergic blockers, direct vasodilators, calcium channel blockers, and angiotensin-converting enzyme inhibitors. These agents are used to achieve a heart rate between 60 and 80 bpm, a systolic blood pressure between 100 and 110

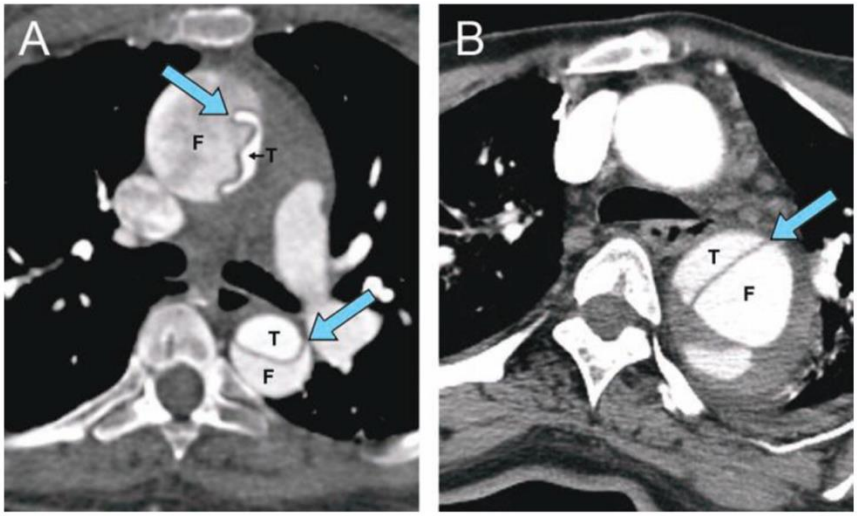


Figure 63 Computed tomographic scans showing that the aorta has been separated into two channels—the true (T) and false (F) lumens— in two patients with different phases of aortic dissection. A. An acute DeBakey type I aortic dissection. The dissecting membrane appears wavy (arrows) in the early phase of dissection. Here, the true lumen of the proximal aorta can be seen to be extensively compressed. This may lead to malperfusion of the heart. B. A chronic DeBakey type III aortic dissection. In the chronic phase, the membrane appears straighter and less mobile (arrow) because it has stabilized over time.

mmHg, and a mean arterial blood pres- sure between 60 and 75 mmHg.

adequate pain control with IV opiates, such as morphine and fentanyl

INITIAL MANAGEMENT

- Admition to ICU
- Intraarterial BP monitoring
- Urine cath. Insertion
- Blood group and cross match for 4 -6 units when surgery is likely
- If hemodynamically unstable>>intubation

1- Place two large bore IVs.

2- Control heart rate and blood pressure.

- -Maintain heart rate <60 BPM
- -systolic blood pressure between 100 and 120 mmHg.

3- Administer IV esmolol or labetolol .>> to control heart rate

- If beta blockers are not tolerated, alternatives are verapamil, diltiazem, or nicardipine.

4- give IV vasodilator therapy. (nitroprusside, nicardipine)

- IF the systolic blood pressure remains above 120 mmHg,
- should not be used without first controlling heart rate with beta blockade.

5- Give IV opioids for analgesia (eg, fentanyl).

Place Foley catheter for assessment of urine output and kidney perfusion

TYPE A:

• The main objectives of operation are to remove the intimal tear site, to replace diseased or dilated aorta as necessary, to obliterate the false lumen and redirect blood flow into the true lumen, and to correct associated valvular insufficiency or coronary ischemia.

•The technique includes internal replacement of the dissected aortic segment with a Dacron graft.

- OPEN REPAIR

TYPE B:

• Admit to the ICU for medical management of hypertension.

•Surgery is indicated if complication occur(malperfusion ,persistent HTN or pain ,rapidly enlarging aortic diameter ,extension of the dissection and rupture)

- ENDOVASCULER REPAIR

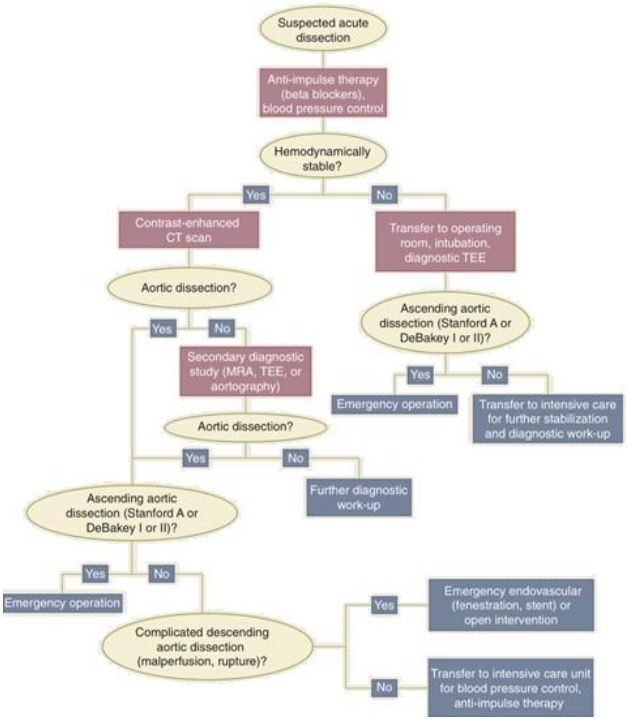


Figure 64

ACUTE LOWER LIMB ISCHEMIA

DEFINITION:

sudden occlusion of a previously patent artery supplying the lower limbs which posses a potential threat to the viability of the limbs.

Causes: The most common cause is embolization.

•The heart is the source of embolus in

80-90% of episodes.

•5-10% of emboli of unknown origin called cryptogenic emboli.

if adequate collateral circulation not present, irreversible changes may appear as early as 4-6hs after onset.

So, priority must be given to restore the blood flow within this period.

Once the occlusive process has begun, vasospasm & propagation of thrombus distal to the site of initial occlusion can contribute to further ischemia.

THE CHARACTERISTIC S&S ARE THE 6PS:

1.Pain.

2.Pallor.

3.Pulselessness.

4.Poikilothermia.

5.Paresthesia.

6.Paralysis.

Paresthesia: is an essential finding. The earliest sign of tissue loss:

• is the loss of:

light touch, 2 point discrimination, vibratory perception, & propreoception esp. in the 1st web space of the foot (not pain, pressure or temperature BCZ:- the larger fibers serving these functions are relatively less susceptible to hypoxia).

Paralysis: the onset of motor paralysis is an indication of advanced limb threatening ischemia & impending gangrene If changes persist beyond 12h. Tense swelling with acute tenderness of a muscle belly occur.

(common in gastrocnemious muscle after superficial femoral artery occlusion).

The extent of paralysis must be determined.

•Skin & subcutaneous tissue have greater resistance to hypoxia than nerves & muscles

•Peripheral pulses: in earlier stages the pulse at site of occlusion can be palpable but absent distally,

because the fresh clot, is soft, semiliquid consistency & allow the pulse transmission to the site of occlusion, when becomes organized & densely compacted the pulse lost at site of occlusion.

DIAGNOSTIC TESTING

- CBC, platelets count, Blood chemistry, coagulation profile.
- CXR, AXR (look for calcifications), ECG, transesophageal echocardiogram.
- Doppler segmental pressure is useful to localize the involved arterial segment and as a quantitative index to gauge the severity of the problem.
- Arteriography

ARTERIOGRAPHY

the gold standard for diagnosis.

* it should not be performed if doing so would keep a critically ischemic limb from receiving prompt surgical therapy.

* should be reserved for patients with viable limbs who can tolerate the additional delay before revascularization.

MANAGEMENT

- If physical examination demonstrates clear evidence of embolization, the definitive therapy should not be delayed.

Immediate heparin i.v bolus of 80units/kg, followed by i.v. infusion of 18units/kg/h, & PTT should be maintained between 60-80sec.

Heparin prevents proximal and distal propagation of thrombus, maintains patency of collateral vessels, and in addition can have a beneficial effect by reducing the extent of ischemic injury

Surgical therapy

- Embolectomy:
  - under local or general anesthesia.

- Fogarty catheter used.
- intraoperative thrombolysis When residual thrombus exists.
- intraoperative arteriogram necessary to define the position of the clots so that the catheter can be placed as close to the clot as possible.and When the viability of the extremity is threatened or when there are poor Doppler signals in the distal extremity .

catheter embolectomy is done through the common femoral artery. Most aortic, iliac, superficial femoral, and popliteal artery occlusions can be managed successfully through this vessel.

- Bypass grafting.
    - Heparin is reinstituted 6 to 12 hours after surgery because of a significant incidence of recurrent embolism.
    - Amputation (usually after 12h the limb is paralyzed due to irreversible damage).
- Thrombolytic therapy

Useful in patients with clearly viable extremity in whom thrombosis is the likely underlying cause of their acute ischemia.

In general, the fresher the thrombus the more successful the thrombolysis.

Urokinase is the agent of choice

- Percutaneous aspiration thromboembolectomy

Useful as an adjunct to thrombolysis to reduce the clot volume

COMPLICATIONS

1.REPERFUSION INJURY:

reestablishment of blood flow leads to further tissue death.

- results from formation of oxygen-free radicals.
- cause direct tissue damage & accumulation of WBCs &
- sequestration in the microvascular system,
- it prolongs the ischemia despite restoration of axial blood flow.
- currently no proved therapy limits the injury.

2. COMPARTMENT SYNDROME:

- prolonged ischemia cause cell membrane damage & leak of fluid to interstitium.
- edema increased intracompartmental pressure, when exceeds CPP, further muscle & nerves necrosis occur.
- Fasciotomy should be done if ischemia>6h
- 2 incisions, one antrolateral, one posteromedial.
- skin lift open, to be closed either secondarily or by graft later on.

INDICATIONS FOR FASCIOTOMY:

- 4–6 hour delay after vessel injury.
- Combined vein and artery injury.
- Concomitant fracture/crush, severe soft-tissue injury,muscle edema or patchy necrosis.
- Tense compartment/compartment pressures exceeding 40 mm Hg.
- Prophylactic for patients with prolonged transport times or long periods without observation (no surgical care available).

3.MYO-NEPHROPATHIC SYNDROME:

- products of ischemic muscles as K, lactic acid, myoglobin, Cr phosphokinase released to the circulation after reperfusion leads to arrhythmias & renal failure.
- aggressive hydration, diuresis as mannitol, intravenous sodium bicarbonate sufficient to alkalinize the urine.

Alkalization of the urine reduces the extent of myoglobin precipitation in the renal tubules.

Insulin and glucose given intravenously may be necessary for extreme or sudden elevations in serum potassium levels

4.Catheter related complications:

- early: from arterial wall trauma includes perforation, rupture, intimal dissection, pseudo-aneurysm formation.
- late: development of accelerated atherosclerosis in-the embolectomized vessel.

ACUTE VENOUS DISEASES

LOWER EXTREMITY VEINS ANATOMY

Lower extremity veins are divided into superficial, deep, and perforating veins. The superficial venous system lies above the uppermost fascial layer of the leg and thigh and consists of the great saphenous vein (GSV) and small saphenous vein (SSV) and their tributaries. The GSV originates from the dorsal pedal venous arch and courses cephalad, anterior to the medial malleolus, entering the common femoral vein approximately 4 cm inferior and lateral to the pubic tubercle. The saphenous nerve accompanies the GSV medially from the ankle to the level of the knee and supplies cutaneous sensation to the medial leg and ankle. The SSV originates laterally from the dorsal pedal venous arch and courses cephalad in the posterior calf. Most often, it penetrates the popliteal fossa, between the medial and lateral heads of the gastrocnemius muscle, to join the popliteal vein. The termination of the SSV may be quite variable, however, with a proximal extension of the SSV (the vein of Giacomini) frequently connecting with the deep femoral vein or GSV. The sural nerve accompanies the SSV laterally along its course and supplies cutaneous sensation to the lateral malleolar region.

DEEP VENOUS THROMBOSIS

RISK FACTOR

- Mutation prothrombin 20210A gene variant; antithrombin, protein C, and protein S deficiencies; and dysfibrinogenemias. In some patients, the cause of the thrombophilia may have both a heritable and an acquired component. These mixed causes include homocysteinemia; factor VII, VIII, IX, and XI elevation; hyperfibrinogenemia; and activated protein C resistance in the absence of factor V Leiden.11 There may be a synergistic effect when particular multiple inherited and acquired risk factors are present in the same patient.
- Other patient-specific factors associated with venous thrombosis include the traditional cardiovascular risk factors of obesity, hypertension, and diabetes. VTE is more com- mon in whites and African American
- Orthopedic surgical patients are generally excluded from risk assessment scores because of the disproportionately increased risk of VTE in orthopedic surgery compared with the general and abdominopelvic surgery population.

Risk factoirs for venous thromboembolism.	
Patient factors	• Age • Obesity • Varicose veins • Immobility • Pregnancy • Puerperium • Hlgh-dose oestrogen therapy • Previous deep vein thrombosis or pulmonary embolism Thrombophilia
Disease or surgical procedure	• Trauma or surgery, especially of pelvis, hip and bwer limb • Malignancy, especially pelvic, and abdominal metastatic • Heart failure • Recent myocardial infarction • Paralysis of lower llmb(s) • infection Inflammatory bowel disease • Nephrotic syndrome • Polycythaemia • Paraproteinaemia • Paroxysmal nocturnal haemogloblnuria • antibody or lupus anticoagulant • Behcet's disease • Homocystlnaemla

Abnormalities of thrombosis and fibrinolysis (thrombophylia) that lead to an increased risk of venous thrombosis.	
Congenital	Deficiency of antithrombin III, protein C or protein S Anti phospholipid antibody or lupus anticoagulant Factor V Leiden gene defect or activated protein C resistance Dysflbrlnogenaemlas
Acquired	Anti phospholipid antibody or lupus anticoagulant

CLINICAL EVALUATION.

Early in the course of DVT development, venous thrombosis is thought to begin in an area of relative stasis, such as a soleal sinus vein or immediately downstream of the cusps of a venous valve in the axial calf veins. Isolated proximal DVT without tibial vein thrombosis is unusual. Early in the course of a DVT, there may be no or few clinical findings such as pain or swelling. Even extensive DVT may sometimes be present without signs or symptoms. History and physical examination are therefore unreliable in the diagnosis of DVT. In addition, symptoms and signs generally associated with DVT, such as extremity pain and/or swelling, are nonspecific. In large studies, DVT has been found by venography or DUS in ≤50% of patients in whom it was clinically suspected. Objective studies are therefore required to confirm a diagnosis of VTE or to exclude the presence of VTE.

Clinical symptoms may worsen as DVT propagates and involves the major proximal deep veins. Extensive DVT of the major axial deep venous channels of the lower extremity with relative sparing of collateral veins causes a condition called phlegmasia cerulea dolens This condition is characterized by pain and pitting edema with associated cyanosis.

When the thrombosis extends to the collateral veins, massive fluid sequestration and more significant edema ensue, resulting in a condition known as phlegmasia alba dolens. The affected extremity in phlegmasia alba dolens is extremely painful and edematous and pale secondary to arterial insufficiency from dramatically elevated below lower knee compartment pressures. Both phlegmasia cerulean dolens and phlegmasia alba dolens can be complicated by venous gangrene and the need for amputation.

VASCULAR LAB AND RADIOLOGIC EVALUATION

Duplex Ultrasound DUS is now the most commonly performed test for the detection of infrainguinal DVT, both above and below the knee, and has a sensitivity and specificity of >95% in symptomatic patients.

DUS provides the ability to noninvasively visualize venous anatomy, detect occluded and partially occluded venous segments, and demonstrate physiologic flow characteristics using a mobile self-contained device.

From the common femoral through the popliteal vein, the primary method of detecting DVT with ultrasound is demonstration of the lack of compressibility of the vein with probe pressure

Lack of coaptation indicates thrombus. Calf vein thrombi are often best detected by abnormalities in color flow imaging.

Lower extremity DVT can be diagnosed by any of the following DUS findings: lack of spontaneous flow (Fig. 24-6), inability to compress the vein (Fig. 24-7), absence of color filling of the lumen by color flow DUS, loss of respiratory flow variation, and venous distention.

Venography is the gold standard to which other diagnostic modalities are compared. A small catheter is placed in a dorsal foot vein with injection of a radiopaque contrast agent. Radiographs are obtained in at least two projections. A positive study result is failure to fill the deep system with passage of the contrast medium into the superficial system or demonstration of discrete filling defect

Magnetic resonance venography is now as accurate as contrast venography.

Pulmonary embolism is diagnosed definitively by computed tomography (CT) scanning of the pulmonary arteries, which can show filling defects in the pulmonary arteries.

Pulmonary angiography is rarely required

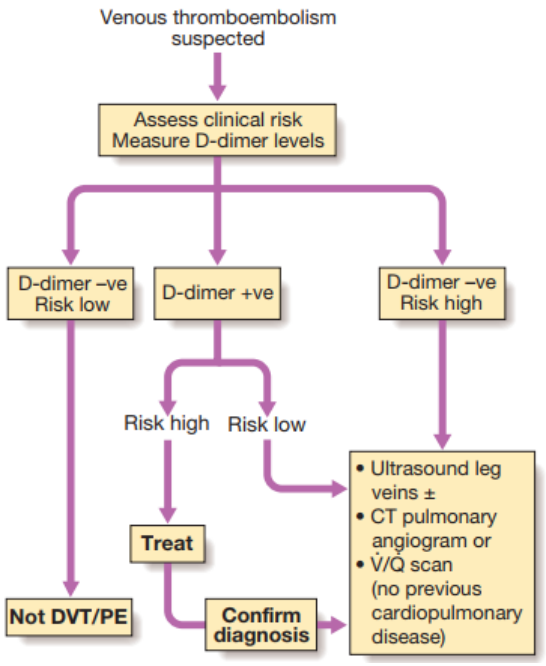


Figure 65 Algorithm for the investigation of patients with suspected pulmonary thromboembolism. Clinical risk is based on the presence of risk factors for venous thromboembolism and the probability of another diagnosis.

DIFFERENTIAL DIAGNOSIS :

The differential diagnosis of a deep vein thrombosis includes a ruptured Baker's cyst, a calf muscle haematoma, a ruptured plantaris muscle, a thrombosed popliteal aneurysm and arterial ischemia.

Duplex scanning will detect many of these conditions but often patients present with non-specific pain in the calf that resolves with no firm diagnosis being made.

TREATMENT

Once the diagnosis of VTE has been made, antithrombotic therapy should be initiated promptly. If clinical suspicion for VTE is high, it may be prudent to start treatment while the diagnosis is objectively confirmed. The goals of VTE treatment are the prevention of mortality and morbidity associated with PE and the prevention of the postthrombotic syndrome (PTS)

Treatment regimens may include **antithrombotic therapy,** temporary or permanent vena cava filter placement, catheter-directed or systemic thrombolytic therapy, and operative thrombectomy.

Antithrombotic Therapy. Mostoften,antithrombotic-therapy for VTE is initiated with IV or subcutaneous (SC) unfractionated heparin or SC low molecular weight heparin. Fondaparinux, a synthetic pentasaccharide, is sometimes also used as an alternative to heparin to initiate therapy. An oral vitamin K antagonist, usually sodium warfarin, is begun shortly after initiation of IV or SC therapy. Either SC or IV therapy is continued until effective oral anticoagulation with warfarin is achieved as indicated by an international normalized ratio (INR) ≥2 for 24 hours. A minimum of 5 days of heparin or fondaparinux therapy is recommended. Recently, the U.S. Food and Drug Administration(FDA) has also approved alternative oral anticoagulants for both treatment and prophylaxis for VTE.

Systemic and Catheter-Directed Thrombolysis. Patients with extensive proximal, iliofemoral DVT may benefit from systemic thrombolysis or catheter-directed thrombolysis (CDT). CDT appears to be more effective (see later in chapter) and potentially reduces acute congestive lower extremity symptoms more rapidly than anticoagulation alone and decreases the development of PTS.

Several thrombolytic agents are available, including streptokinase, urokinase, alteplase

Placement of an IVC filter is indicated for patients who have manifestations of lower extremity VTE and absolute contraindications to anticoagulation, those that have a bleed- ing complication from anticoagulation therapy of acute VTE, or those who develop recurrent DVT or PE despite adequate anticoagulation therapy and for patients with severe pulmonary hypertension.

Operative Venous Thrombectomy. In patients with acute iliofemoral DVT, surgical therapy is generally reserved for patients who worsen with anticoagulation therapy and those with phlegmasia cerulea dolens and impending venous gangrene. If the patient has phlegmasia cerulea dolens, a fasciotomy of the calf compartments is first performed. In iliofemoral DVT, a longitudinal venotomy is made in the common femoral vein and a venous balloon embolectomy catheter is passed through the thrombus into the IVC and pulled back several times until no further thrombus can be extracted

Summary of American College of Chest Physicians recommendations regarding duration of long-term antithrombotic therapy for deep vein thrombosis (DVT)	
CLINICAL SUBGROUP	ANTITHROMBOTIC TREATMENT DURATION
First episode DVT/transient risk/surgery	VKA or LMWH for 3 months
First episode DVT/ unprovoked	VKA or LMWH for 3 months Consider for long-term therapy if: • Proximal DVT • Minimal bleeding risk • Stable coagulation monitoring
Distal DVT/unprovoked • **Symptomatic** • **Asymptomatic and no risk factors for progression**	VKA for 3 months Serial imaging in 2 weeks, if progression VKA for 3 months
Second episode DVT/ unprovoked **DVT and cancer**	VKA for extended therapy LMWH for extended therapy over VKA

SUPERFICIAL VEIN THROMBOPHLEBITIS

Superficial vein thrombophlebitis (SVT) most commonly occurs in varicose veins but can occur in normal veins. When SVT recurs at variable sites in normal superficial veins, thrombophlebitis migrans, it may signify a hidden visceral malignancy or a systemic disorder such as a blood dyscrasia and/ or a collagen vascular disease. SVT also frequently occurs as a complication of indwelling catheters, with or without associated extravasation of injected material.

Clinical signs of SVT include redness, warmth, and tender- ness along the distribution of the affected veins, often associated with a palpable cord. Patients with suppurative SVT may have fever and leukocytosis.

DUS should be performed in patients with signs and symptoms of acute SVT to confirm the diagnosis and to determine if any associated DVT is present. Concomitant lower extremity DVT may be present in 5% to 40% of patients with SVT; most occur in patients with greater saphenous vein SVT within 1 cm of the saphenofemoral junction. A follow- up DUS should be performed in 5 to 7 days in patients with SVT in the proximal GSV but without deep vein involvement

Treatment of SVT is quite variable. A Cochrane Review reported that LMWHs and nonsteroidal anti-inflammatory drugs both reduce the rate of SVT extension or recurrence. Topical medications appear to improve local symptoms. Surgical treatment, combined with the use of graduated compression stockings, is associated with a lower rate of VTE and SVT progression.77 The treatment is individualized and depends on the location of the thrombus and the severity of symptoms.

In patients with SVT not within 1 cm of the saphenofemoral junction, treatment consists of compression and administration of an anti-inflammatory medication such as indomethacin. In patients with suppurative SVT, antibiotics and removal of any existing indwelling catheters are mandatory. Excision of the vein may be necessary but is usually reserved for patients with systemic symptoms or when excision of the involved vein is straight- forward. If the SVT extends proximally to within 1 cm of the saphenofemoral junction, extension into the common femoral vein is more likely to occur. In these patients, anticoagulation therapy for 6 weeks and GSV ligation appear equally effective in preventing thrombus extension into the deep venous system

VARICOSE VEINS

DEFINITION:-

Varicose veins are defined as dilated, usually tortuous, subcutaneous

veins >=3 mm in diameter measured in the upright position with demonstrable reflux.

EPIDEMIOLOGY

The adult prevalence of visible varicose veins is 25–30 per cent

in women and 15 per cent in men.

<u>Factors affecting prevalence</u>

include:

- **gender: the vast majority of studies report a higher prevalence in women than men**
- **age: the prevalence of varicose veins increases with age.**
- **body mass and height: increasing body mass index and height may be associated with a higher prevalence of varicose veins;**
- **pregnancy: appears to increase the risk of varicose veins;**
- **family history: evidence supports familial susceptibility to varicose veins;**
- **occupation and lifestyle factors: there is inconclusive evidence regarding increased prevalence of varicose veins in smokers, patients who suffer constipation and occupations which involve prolonged standing.**

CLINICAL FEATURES (MOST COMMON SITE IN LEGS)

SYMPTOMS:

- **Varicose veins frequently cause symptoms, the most common being:**

- aching or heaviness, which typically increases throughout the day or with prolonged standing and is relieved by elevation or compression hosiery.

Other less common symptoms include ankle swelling and itching while complications

(bleeding, superficial thrombophlebitis, eczema, lipodermatosclerosis and ulceration) represent important indications for investigation and intervention.

SIGNS:

The presence of tortuous dilated subcutaneous veins are usually clinically obvious. These are confined to the long and lesser saphenous systems in approximately 60 and 20 per cent of cases, respectively.

The distribution of varicosities may indicate which superficial system is defective;

medial thigh and calf varicosities suggest long saphenous incompetence (Figure 1), posterolateral calf varicosities are suggestive of short saphenous incompetence(Figure 1),

anterolateral thigh and calf varicosities may indicate isolated incompetence of the proximal anterolateral long saphenous tributary (Figure 1).

Percussion over the varices may elicit an impulse tap by the fingers placed over the dilated trunk. (((we will talk about examination in details next up so don't worry))

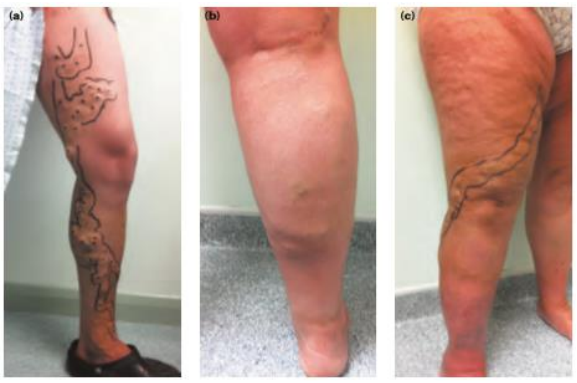


Figure 66

Other signs commonly found include:

- Telangectasia, which are essentially a confluence of dilated intradermal venules <1 mm in

diameter. These may be mild or severe (Figure 3). Synonyms include spider veins, thread veins and hyphen webs.

- Reticular veins are dilated, subdermal veins, 1–3 mm in diameter. The presence of telangiectasia and reticular veins are of dubious significance, are not necessarily associated with major varicose veins and are purely a cosmetic problem.
- In saphena varix (Figure 2), when there is a large groin varicosity which presents as a (usually painless) lump, emergent when standing and disappearing when recumbent. Gentle palpation over the varix during coughing may elicit a thrill.
- Atrophie blanche (Figure 4) is localised white atrophic skin frequently surrounded by dilated capillaries and hyperpigmentation, usually seen around the ankle.
- Corona phlebectasia are fan-shaped patterns of small intradermal veins on the medial or lateral aspects of the ankle or foot. Synonyms include malleolar or ankle flares.

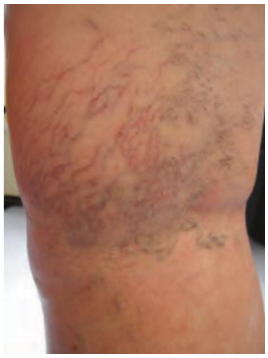


Figure 68

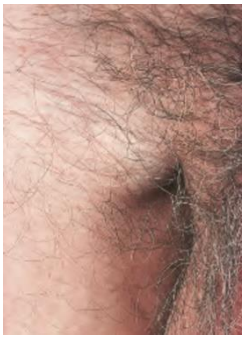


Figure 67

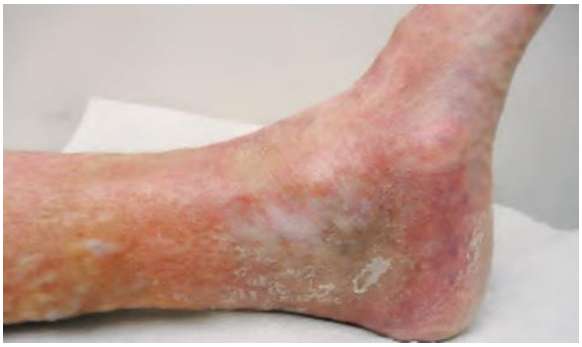


Figure 69

- Pigmentation (Figure 5) is usually a brown discolouration (because of haemosiderin deposition) of the skin, most frequently affecting the gaiter area, and may be associated with phlebitis and ulceration.

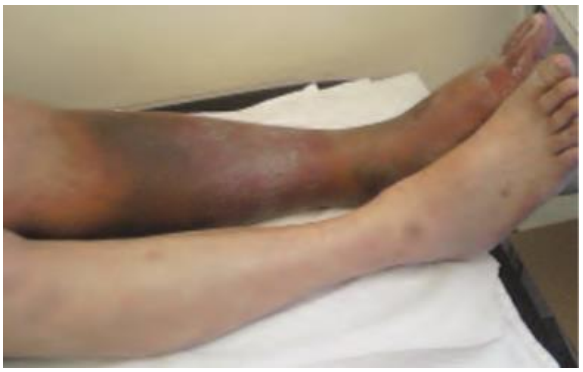


Figure 70

INVESTIGATION:

Tourniquet tests have now largely been abandoned. There is good evidence to support the policy of duplex ultrasound scanning for all patients with varicose veins prior to any intervention. This policy facilitates a more accurate surgical approach, reduces the incidence of varicose vein recurrence, and allows assessment regarding suitability for endovenous intervention.

This book doesn't support tourniquet use. so I will give other source which is brouse book because the examination is explained by our dr in the lecture and it need to be mentioned so before we begin we have to know some anatomy first :

ANATOMY AND PHYSIOLOGY

The veins of the lower limb are divided into superficial and deep systems, separated by the deep fascia of the leg. At certain points there are veins that pass through the deep fascia to provide a communication between the two systems. Normally the valves

in these communicating veins only allow blood to pass from the superficial into the deep system. The deep veins have many valves to ensure that blood only flows upwards against the force of gravity towards the heart.

Blood flow in the erect position is mostly produced by the calf muscle pump. Within the soleal muscles

of the calves are large venous sinusoids – thesoleal sinusoids – which are compressed during contraction of the calf muscles, e.g. during walking,

so that blood is forced out of the calf veins into the popliteal veins and on towards the heart. During calf muscle relaxation, the intramuscular veins open but blood is prevented from refluxing back into them from the proximal deep veins by the valves in the popliteal veins. The negative pressure in the deep veins then sucks blood in from the superficial system through the communicating veins to reduce the superficial venous pressure, incrementally, with each calf muscle contraction. The superficial veins all eventually join either the great (long) or lesser (short) saphenous system. These two major subcutaneous veins end where they communicate with the femoral and popliteal veins,

respectively. Both superficial systems are also joined to the deep veins by a number of other communicating (perforating) veins, the most important of whichare in the calf.

The main tributaries of the saphenous veins are the veins that become varicose because they, unlike the saphenous veins, do not contain a strong coat of smooth muscle in their wall. They lie in a more superficial position and are not bound down to the deep fascia.

The deep veins accompany the arteries of the lower leg join to form the popliteal vein, which also receives blood from the calf muscle sinusoids.

Don't forget to do full abdominal exam and scrotal exam in MALES

INSPECTION:

Look for large visible veins. Record their site, extent and size on large drawings of the front and back of the limbs. The skin of the leg should be inspected, especially the skin of the lower third of the medial side of the calf, for signs of chronic venous hypertension, lipodermatosclerosis, pigmentation, induration and inflammation as well as eczema and ulceration.

PALPATION

The examiner's dominant hand should be gently run over the course of the main veins and their tributaries because dilated veins can sometimes be more easily felt than seen, especially in fat legs.Veins in the lower leg often lie in a gutter of indurated subcutaneous fat. Dilated long and short saphenous veins are usually easy to feel. The termination of a distended short saphenous vein is easier to feel if the patient is asked to bend the knees slightly to relax the deep fascia covering the popliteal fossa.

Carefully palpate the sapheno-femoral junction

(2.5 cm below and lateral to the pubic tubercle) and the sapheno-popliteal junction, which has a variable termination in the popliteal fossa (high or low).

The patient should be asked to cough while the dilated veins are palpated to see if there is any impulse or thrill (a cough impulse) indicating that the valves at their junctions with the deep veins are incompetent and the back flow is turbulent.

Palpate the skin of the calf to define any areas of induration and tenderness (lipodermatosclerosis). Palpate the medial side of the calf for deficits in the deep fascia which may be the sites of incompetent calf communicating (perforating) veins.

(This is an unreliable technique, as large surface varicosities produce lacunae in the subcutaneous fat of the calf similar to the perforating vein deficits.)

PERCUSSION :

The distended, dilated trunks of the long and short saphenous systems will always transmit a percussion wave in an orthograde direction whether the valves are competent or not. The more distended the vein, the better the wave is transmitted.

If a percussion wave is transmitted retrogradely, i.e. downwards while the patient is standing, the valves must be incompetent. Percussion can also be used to help define the terminations of the long and short saphenous veins by placing the fingers of one hand gently over the upper end of the dilated saphenous trunk and percussing the vein below it using the middle finger of the other hand to 'flick' the distended varicosities.The process can then be repeated in reverse, with the upper end of the vein being 'flicked' and the lower hand detecting a

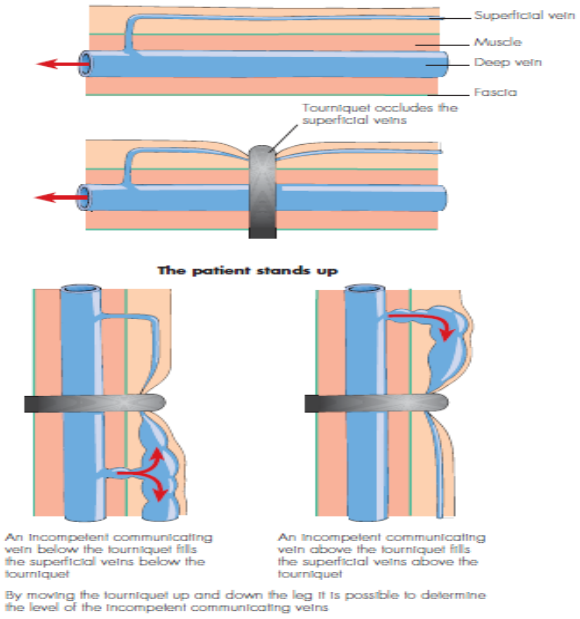
downward percussion wave, to check the competence of the valves.

AUSCULTATION:

Listen with your stethoscope over any large clusters

of veins, especially if they remain distended when the patient lies down and the limb is elevated. A machinery murmur over such veins indicates that they are secondary to an arterio-venous fistula.

TOURNIQUET TESTS



The patient should lie on a couch which has a small foot stool attached to it, onto which the patient can rapidly stand. The limb to be examined is then elevated – often by placing it on the examiner's shoulder – to empty the veins, a process that can be expedited by stroking the blood within the veins towards the heart.

A tourniquet made from a long length of 1 cm diameter soft rubber tubing is then pulled tight around the upper thigh and held in place by strong artery forceps (Spencer Wells). The rubber tubing must press firmly into the subcutaneous tissues of the thigh to ensure that the subcutaneous veins are completely occluded. The Velcro tourniquets used by many house officers for venesection do not apply enough pressure, but the soft tubing on a stethoscope is an effective alternative if rubber tubing is not available, provided it is long enough.

The patient is then asked to stand up quickly and the legs are observed for 10–15 seconds. If the saphenofemoral junction is the only site of superficial to deep valvular incompetence, the veins above the tourniquet will rapidly fill but those below it will remain collapsed. This can be confirmed by suddenly releasing the tourniquet and watching the veins below the site of the tourniquet rapidly distend from above, as blood regurgitates down the long saphenous vein.

If the veins below the tourniquet fill immediately the patient stands up, there must be other sites of superficial to deep incompetence below the level of the tourniquet.

This test can be repeated with the tourniquet moved progressively down the whole length of the leg to try to define all the sites of superficial to deep vein incompetence, but it is easier and simpler to apply it once below the knee to exclude short saphenous incompetence.

Tourniquet tests are often difficult to interpret in patients with recurrent varicose veins, and the value of applying multiple tourniquets in an effort to locate the precise level of calf communicating veins has never been scientifically verified.

A modification of the tourniquet test is to empty the limb as described above and apply direct digital pressure over the upper end of the long saphenous vein while the patient stands up to see if this prevents retrograde filling. This is called the Trendelenburg test.

Venous hypertension caused by proximal vein obstruction or the presence of an arterio-venous fistula should be suspected if varicose veins fail to collapse on elevation. This can be confirmed by asking the patient to stand up after placing a tourniquet just below the knee, to cut off both long and short saphenous reflux, and then to stand repeatedly on tiptoe and relax. In a normal limb this exercise will empty the superficial veins by sucking the blood in the surface varicosities into the deep veins, through competent perforating veins, and then pumping it up through the popliteal vein to

The causes of varicose veins in the lower limbs
Secondary • Obstruction to venous outflow by: • pregnancy • fibroids/ovarian cyst • abdominal lymphadenopathy • pelvic cancer (cervix, uterus, ovary, rectum) • ascites • iliac vein thrombosis • retroperitoneal fibrosis
Valve destruction • Deep vein thrombosis
High flow and pressure • Arterio-venous fistula (especially the acquired traumatic variety)
Primary Cause not known; often familial Probably a weakness of the vein wall that permits valve ring dilatation Very rarely, Congo mt al absence of valves

the heart. Failure to achieve superficial vein emptying

during this exercise indicates deep vein obstruction or reflux through incompetent valves in the deep or communicating veins. This is called Perthés' walking test.

MANAGEMENT

CONSERVATIVE:

COMPRESSION HOSIERY

Compression hosiery relies on graduated external pressure to improve deep venous return. Compression hosiery can be knee length or thigh length;

ULTRASOUND-GUIDED FOAM SCLEROTHERAPY

Ultrasound-guided foam sclerotherapy involves the injection of detergent directly into the superficial veins.

ENDOVENOUS LASER ABLATION

involves the insertion of a laser fibre into the lumen of an incompetent truncal vein, with subsequent thermal ablation of the vein. The vast majority of patients with primary and recurrent varicose veins are suitable for EVLA

RADIOFREQUENCY ABLATION

Radiofrequency ablation (RFA) is a minimally invasive endovascular therapy that uses a bipolar catheter to generate thermal energy to ablate the vein.

SURGERY:

The principles of the operation are to ligate the point of junctional incompetence and to remove the refluxing trunk and dilated tributaries.

TYPES:

Saphenofemoral ligation and long saphenous

Stripping

Saphenopopliteal junction ligation and lesser

saphenous stripping

Perforator ligation

Phlebectomies

THE THYROID GLAND

ANATOMY

The thyroid gland (thyreos =Greek for shield) has 2 lobes, right and left which are connected by a narrow isthmus, which overlies the second and third tracheal rings. The thyroid normally weighs 15-30 g and is invested by the pre-tracheal fascia, which binds it to the larynx, cricoid cartilage and trachea. The strap muscles (sternohyoid and sternothyroid) lie in front of the pre-tracheal fascia and must be separated to gain access to the gland.

The parathyroid glands are usually located within the thyroid gland, usually on its posterior aspect.

BLOOD SUPPLY

The arterial supply of the thyroid gland is rich. The superior thyroid artery runs down to the upper pole of the gland as a branch of the external carotid artery, whereas the inferior thyroid artery runs across to the lower pole from the thyrocervical trunk (a branch of the subclavian artery).

Blood from the thyroid drains through superior, meddle thyroid veins into the internal jugular vein, whereas the inferior drains into the brachiocephalic vein (innominate vein). Lymphatics drain laterally to the deep cervical chain and downwards to pre-tracheal and mediastinal nodes.

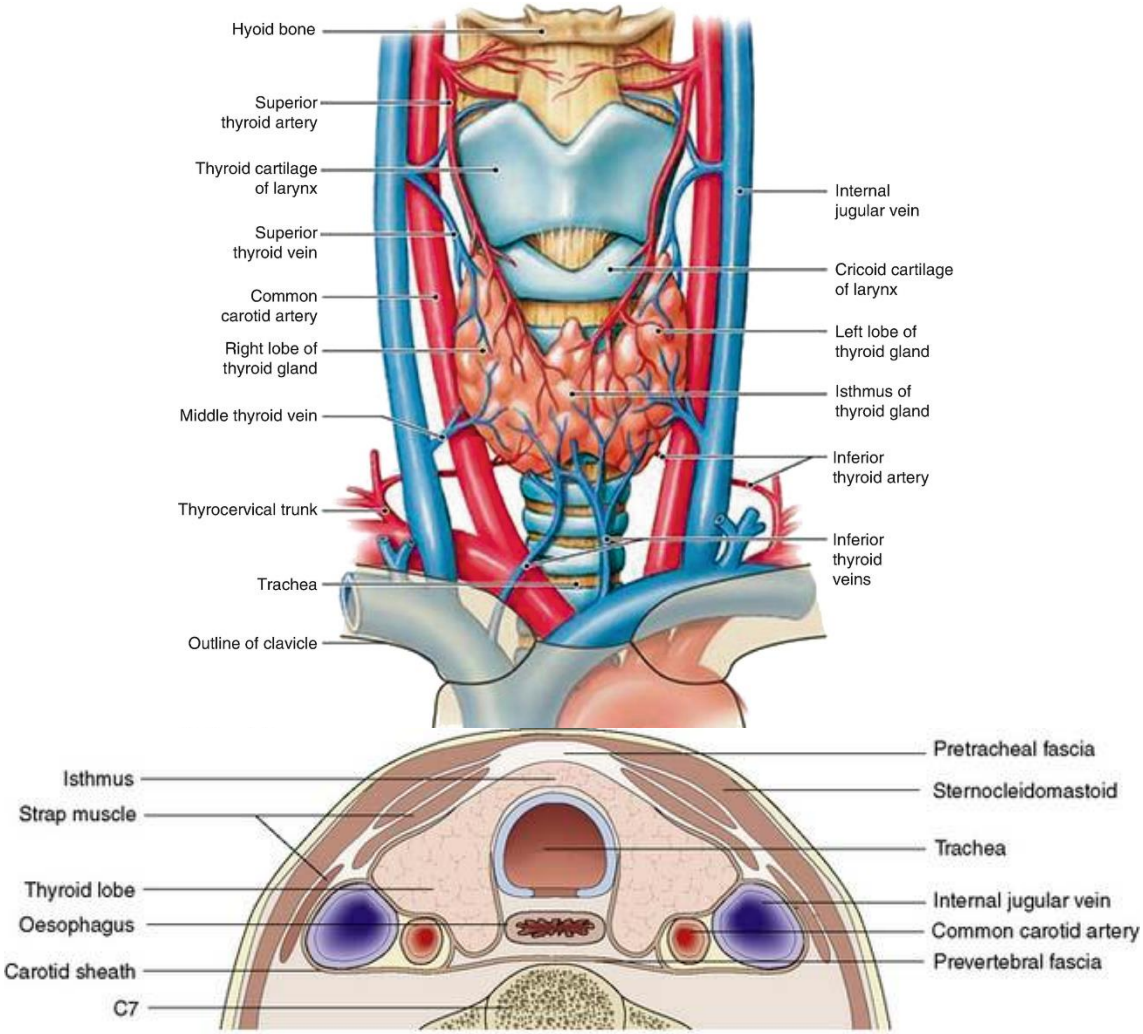


Figure 71 A-anterior view of the thyroid gland B-cross section of the neck at the level of the 7th cervical vertebra

IMPORTANT RELATIONS

The recurrent laryngeal nerve is a branch of the valgus, which passes upwards in the groove between the oesophagus and trachea to enter the larynx and supply all of its intrinsic muscles except the cricothyroid. The recurrent nerve also supplies sensation to the larynx below the vocal cords. As it nears the gland, the inferior thyroid artery usually passes in front of the recurrent laryngeal nerve , but may pass beneath it or branch around it (figure 2). The superior laryngeal nerve (also a branch of the vague) runs with the superior thyroid vessels and supplies the cricothyroid muscles (external branch), which tense the vocal cords. The internal branch of the superior laryngeal nerve provides sensation to the larynx above the cords. Both nerves (the superior and recurrent) are at risk of damage during thyroid surgery and the consequences, if permanent, can be disabling. by cuboidal epithelium (thyrocytes). The parafollicular or C cells may be seen between follicles (Figure 3). The functioning unit is the lobule supplied by a single arteriole and consisting of 24-40 follicles.

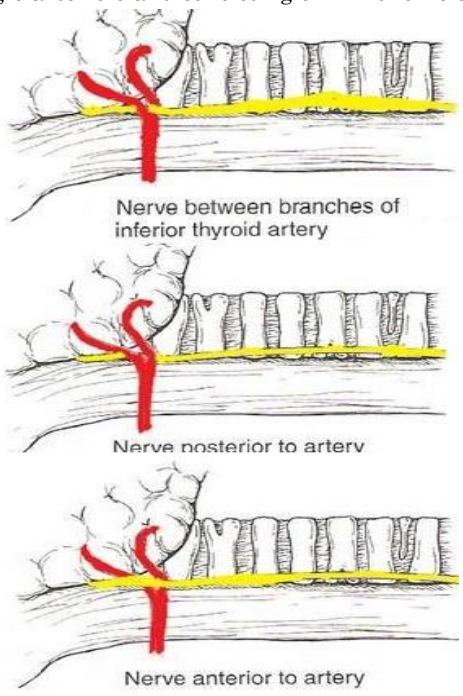


Figure 72 relation of the inferior thyroid artery to the recurrent laryngeal nerve

PHYSIOLOGY

The gland has an exceedingly rich blood supply. The thyrocytes secrete triiodothyronine (T3) and thyroxine (T4). T3 is the active hormone, and T4 is converted to T3 in the periphery. In the blood they are bound to serum proteins mainly, albumin, thyroxine-binding globulin (TBG). The small amount of hormone that remains free in the serum is biologically active.

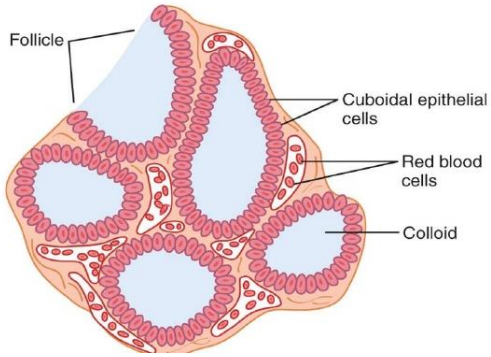


Figure 73 microscopic structure of the thyroid gland

Secretion of T3 and T4 is controlled by thyroid-stimulating hormone (TSH), which is secreted by the anterior pituitary. TSH release is in turn controlled by thyrotropin-releasing hormone (TRH) from the hypo-thalamus. Circulating levels of T3 (mainly) and T4 (in part) exert a negative feedback effect on the hypothalamus and anterior pituitary (figure 4).

HISTOLOGY

Histologically, the gland is made up of follicles containing colloid, which on haematoxylin and eosin staining appears pink. The follicles are spheroids lined

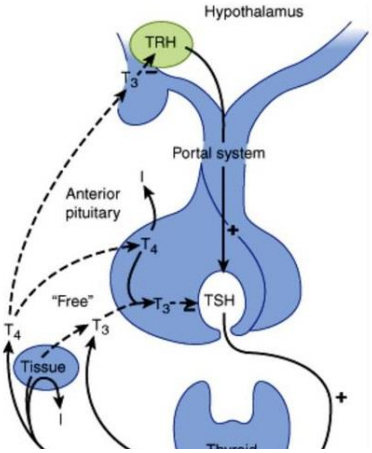


Figure 74 hypothalamic-pituitary- thyroid axis

The parafollicular cells produce calcitonin. This can be measured in the blood and is normally secreted in small amounts. Secretion is increased after food. Calcitonin lowers the serum calcium but it is not an essential hormone.

APPROACH TO THE PATIENT WITH THYROID DISEASE

HISTORY

A patient with thyroid disease may present with a variety of symptoms. Most commonly a patient may present with an apparent neck swelling noticed by the patient or by his peers or relatives. Usually if the swelling is not apparent, it may present with local pressure symptoms such as 'dysphagia (oesophagus), dyspnea and stridor (trachea), hoarseness of voice (recurrent laryngeal nerve), engorged neck veins and ear pain.

In some thyroid diseases (see later) a patient may not present with a thyroid swelling, rather with symptoms of hyperthyroidism or hypothyroidism (see the table below). Patients with malignant thyroid disease may present with remote or distal features of metastasis.

History taken from a patient suspected to have thyroid disease should emphasize on the family history, the drug history and history of radiation exposure.

EXAMINATION

The first step in the examination of a patient with neck swelling is to identify wither it is of thyroid origin. Swellings of thyroid origin are referred to as goitres (from the Latin *guttur* = the throat). The swelling characteristically moves upwards on swallowing because of the gland's attachment to the trachea (by the peritracheal fascia) and usually retains the shape of the normal gland.

After confirming clinically that this is a thyroid swelling, the examination should focus on the presence of a retrosternal extension, the presence of extrathyroidal signs (e.g. pretibial myxoedema, exophthalmos), a neurological examination and wither there is an associated lymphadenopathy (i.e. malignancy).

Symptoms and signs of hypo- and hyperthyroidism

Hyperthyroidism Symptoms	Hypothyroidism Symptoms
• **Weight loss**	• Weight gain
• **Heat intolerance**	• Cold intolerance
• **Diarrhea**	• Constipation
• **Sweating**	• Decreased sweating
• **Emotional lability**	• Depression
• **Irritability**	• Sluggishness
• **Palpitation**	• Myxedema
signs	Signs
• **hyperreflexia**	• Sluggish reflexes
• **Tachycardia**	• Bradycardia
• **hot, moist, palms** • **Tremor**	•. Dry puffy skin with hair loss

According to behaviour, goitres can be classified into benign or malignant. According to function they are classified as simple (nontoxic) or toxic. According to their consistency they can be either diffuse or nodular; the latter is either solitary or multinodular. The classification of thyroid disease based on those features is shown in the table below.

Classification of thyroid disease
Non-malignant goitres **Simple** • **Simple multinodular goitre** • **Physiological diffuse goitre** • **Solitary Nodule (cystic or solid)** • **Thyroiditis** **Hashimoto's Thyroiditis** • **Sub-acute (De quervain)** • **Thyroiditis Riedel's Thyroiditis** **Toxic** • **Grave's disease** • **Toxic multinodular goitre** • **• Toxic Adenoma (Autonomous Nodule)**
Malignant goitres **Primary** **• Differentiated thyroid cancers** • **Papillary carcinoma** • **Follicular carcinoma** **• Medullary carcinoma** • **Sporadic(majority).** • **Familial (M.E.N 2).** **• Anaplastic cancer** **• Lymphoma (non-Hodgkin's - B cell type)** **•Squamous Cell Cancer (very rare).**
Secondary (Metastasis) • **Lung** • **Kidney** • **Breast**

INVESTIGATIONS

- Thyroid function tests; Measurement of free T3 (10-30 nmol/l), T4(3.5-7.5 µmol/l) and TSH (0.3-3.3 mU/l) gives a biochemical estimation of thyroid function. TSH is totally suppressed in thyrotoxicosis and elevated in hypothyroidism. Pregnancy or estrogen administration increases the level of thyroxine-binding globulin, so that estimation of the ratio of free to bound hormone may be -- needed. TRH and TSH stimulation tests may be required to determine the site of failure of production of thyroid hormones
- Calcitonin level; Calcitonin level is also of value to confirm the extent of thyroid tissue involvement and diagnosis of medullary carcinoma.
- Antibodies; Detection of certain antibodies including anti-thyrocyte peroxidase antibody (anti-TPO) and the thyroid stimulating immunoglobulin (TSI) is of value in the diagnosis of hashimoto's thyroiditis and grave's disease, respectively.
- Ultrasound; The thyroid can be imaged routinely by ultrasonography and provides a good anatomical view of the gland (figure 5).
- Radioisotope scanning; radionuclide scanning
- -using radiolabelled iodine (123l) or 99mTc-sodium pertechnetate (which behaves like iodine and is 'trapped' by the gland) is used to differentiate between hot (actively functioning), warm (normally functioning) and cold (non-functioning) thyroid nodules. Total isotope uptake also reflects thyroid activity.
- CT, MR! and PET; computerised tomography (CT), magnetic resonance imaging (MRI) and positron emission tomography (PET) have no role in routine assessment of thyroid swellings and are reserved for the assessment of known malignancy and to assess the extent of retrosternal masses, staging of the malignancy and in the case of MRI for assessment of vascular invasion.
- Fine-needle aspiration (FNA) cytology; Fine- needle aspiration cytology is used to determine the nature of thyroid nodules and is best for discrete nodules.

THYROIDECTOMY

when doing thyroidectomy, the gland is exposed through a transverse skin-crease incision placed 2-3 cm above the sternal notch. The deep cervical fascia is divided longitudinally in the midline and the strap muscles are separated. Each lobe is mobilized by dividing first the vessels supplying the superior pole, then the middle and inferior thyroid veins, and finally the inferior thyroid artery. The recurrent laryngeal

nerves should be identified, so that they can be protected from injury. The amount of thyroid tissue removed depends on the indication for operation (see the table below). Care is taken to preserve the parathyroid glands.

Thyroid procedures

All thyroid operations can be assembled from three basic elements:

- **Total lobectomy**
- **Isthmusectomy**
- **Subtotal lobectomy**

Total thyroidectomy = 2 x total lobectomy + isthmusectomy

Subtotal thyroidectomy = 2 x subtotal lobectomy + isthmusectomy

Near-total thyroidectomy = total lobectomy + isthmusectomy + subtotal lobectomy

Lobectomy = total lobectomy + isthmusectomy

complications of thyroidectomy depend on the procedure used, but in general are shown in the table below.

Complications of thyroid surgery

- **Haemorrhage**
- **Damage to the external branch of the superior laryngeal nerve**
- **Damage to the recurrent laryngeal nerve**
- **('cadaveric' position of the vocal cord (i.e. midway between the closed and open positions)).**
- **Hypothyroidism**
- **Hypoparathyroidism**
- **Scar complications (hypertrophic or keloid)**

SIMPLE GOITERS

MULTINODULAR GOITRE

multinodular goitre is a multinodular swelling occurring in the thyroid gland secondary to overstimulation.

ETIOLOGY

Multinodular goitres are either endemic or sporadic. Endemic goitres occur in areas of iodine deficiency, such as the high mountains, were the soil is poor with iodine, goitre may develop as a result of stimulation of the thyroid gland by TSH, in response to a chronically low level of circulating thyroid hormones which require iodine for their synthesis.

The daily requirement for iodine is about 0.1-0.15 mg. the incidence of endemic goitre have decreased significantly during the past century due to the act of iodizing table salt.

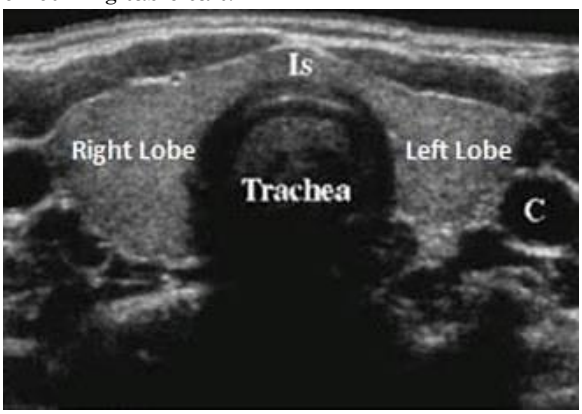


Figure 75 ultrasound of normal thyroid

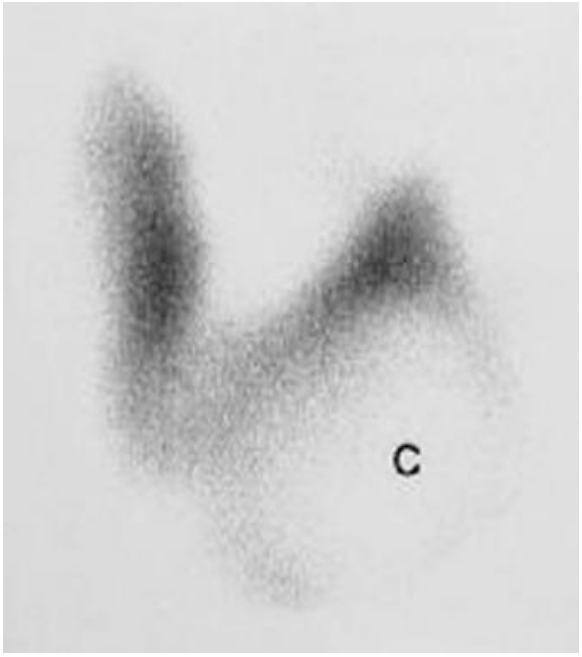


Figure 76 Technetium-99m pertechnetate thyroid scan demonstrates a cold nodule in the left lobe of the thyroid (C).

Sporadic goitres occur due to a defect in one of the enzymes involved in thyroid hormone synthesis, ingestion of antithyroid drugs or goitrogenic food (cabbage).

Goitres are more common females than males because of the presence of estrogen receptors in thyroid tissue.

PATHOLOGY

Initially, the gland is stimulated uniformly resulting in a diffuse hyperplastic goitre, which is reversible if stimulation ceases. Later, as a result of fluctuating stimulation, a mixed pattern develops with active and inactive lobules. Active lobules become more vascular and hyperplastic until haemorrhage occurs, causing central necrosis and leaving only a surrounding rind of active follicles. Necrotic lobules coalesce to form nodules filled with either iodine-free colloid or a mass of new but inactive follicles. Continual repetition of this process results in a nodular goitre.

CLINICAL FEATURE

Patients presenting early, usually during childhood, have diffuse hyperplastic goitre and may present with diffuse swelling that may grow large enough to cause discomfort. Patients presenting late may have nodular goitre (figure 7).

The patient is euthyroid, the nodules are palpable and often visible (smooth, usually firm and not hard) and the goitre is painless and moves freely on swallowing. Nodules may grow large enough to cause local pressure symptoms. Nodules may become suddenly enlarged and painful due to haemorrhage.

INVESTIGATIONS

Thyroid function tests are usually normal. Plain radiographs of the chest and thoracic inlet will rapidly demonstrate clinically significant tracheal deviation (figure 8). Ultrasound and CT give more detailed images and will illustrate tracheal compression (figure 9), however, they rarely influence clinical management.

Prevention and treatment **the incidence of goitre has been strikingly reduced by the introduction of iodized salt. In the early stages (diffuse hyperplastic goitre), the swelling may regress if thyroxine is given at a dose of 0.15-0.2 mg daily for a few months.**

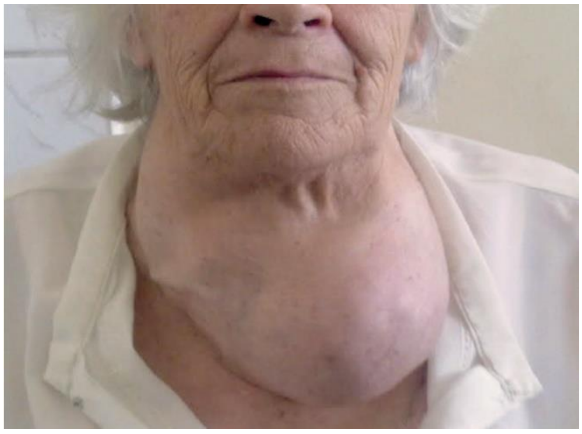


Figure 77 large multinodular goiter

Nodular goitre is relatively irreversible (some may regress after a long time). Indications for surgery include local pressure symptoms, cosmetic reasons and patient anxiety. Surgeries include total thyroidectomy with immediate and lifelong replacement of thyroxine (to prevent recurrence), subtotal thyroidectomy (to conserve the tissue especially the parathyroid glands) and total lobectomy (when there is a dominant lobe with nodules).

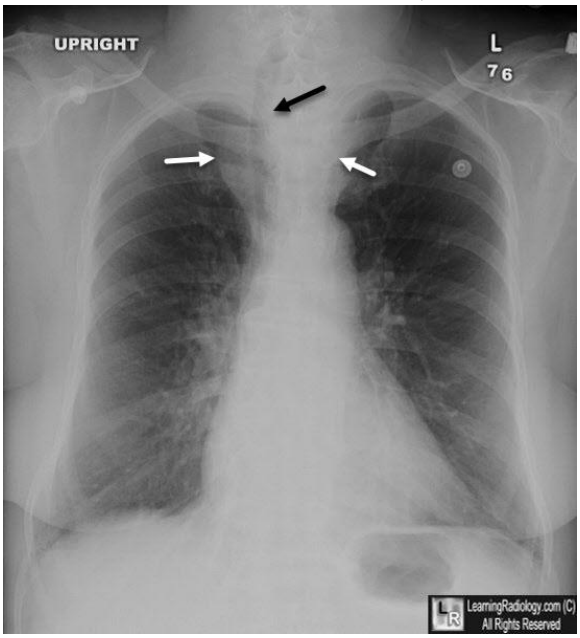


Figure 78 chest X-ray showing a retrosternal goiter with marked deviation of the trachea to the right (arrow).

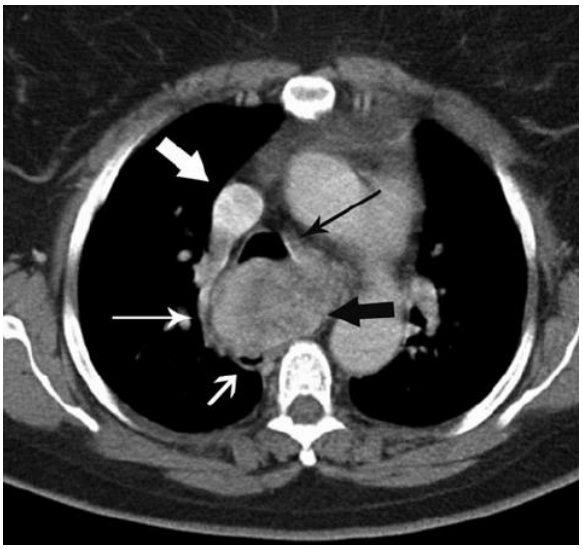


Figure 79 CT scan shows a massive goiter and tracheal compression on the right (arrow).

'PHYSIOLOGICAL' DIFFUSE GOITER

Transient enlargement may occur during puberty or pregnancy probably due to the increased demand of iodine and decreased (or below sufficient) compensation. It is actually, only during those periods of life, the thyroid gland is palpable.

SOLITARY THYROID NODULE

A full discussion of solitary thyroid nodules is made during the 'solitary thyroid nodule' seminar, please refer to that subject for further information. (the next subject).

THYROIDITIS

Autoimmune thyroiditis (Hashimoto's disease) is a disease caused the destruction of thyroid follicles by immunocompetent lymphocytes. Antibodies against various structures of the thyroid follicle are detected in the serum of these patients including antithyroglobulin antibodies (anti-Tg), anti-thyroid peroxidase antibodies (anti-TPO) and anti-microsomal antibodies. Initially patients are euthyroid but may become thyrotoxic and Later may become hypothyroid. Patients usually have diffused thyroid enlargement but a nodular form is also possible. Patients are usually treated medically and surgery is indicated when there are pressure symptoms.

Subacute thyroiditis (de Quervain's disease) is another rare condition that is associated with an influenza like illness resulting in an autoimmune mediated inflammation of the gland with resultant painful diffuse swelling of the gland and transient thyrotoxicosis. Surgery is only indicated for the rare patient with recurrent, disabling episodes of thyroiditis.

Riedel's thyroiditis is a very rare condition in which the thyroid is replaced by dense fibrous tissue, resulting in a firm painless swelling and tracheal compression. The cause is unknown. Surgical decompression of the trachea may be required.

TOXIC GOITERS

GRAVES' DISEASE

Graves' disease, also referred to as primary thyrotoxicosis, is an autoimmune disease that affects women mainly and result in symptoms of hyperthyroidism with other specific manifestations.

ETIOLOGY

In graves' disease, the TSH receptors in the thyroid are stimulated by circulating thyroid-stimulating immunoglobulins (TSI).

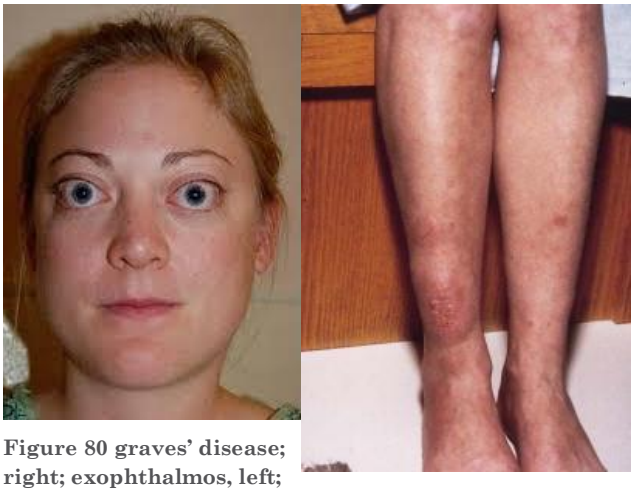


Figure 80 graves' disease; right; exophthalmos, left; pretibial myxedema

CLINICAL FEATURES

Other than hyperthyroidism, graves' disease can present with manifestations not related to the level of the thyroid hormone; Graves' ophthalmopathy, goitre and graves' dermopathy. Graves' ophthalmopathy refers to cluster of eye manifestations including exophthalmos (proptosis), lid retraction (retraction of the upper eyelid) and lid lag (failure of the upper eyelid to keep pace with downward moving

finger). Other manifestations include ophthalmoplegia and chemosis.

Graves' dermopathy (pretibial myxedema) is a rare condition characterized by thickening of the skin, usually in areas of trauma, by deposition of hyaluronic acid in the dermis and subcutis.

The thyroid gland of graves' disease is characterized by being moderately and diffusely enlarged and soft, and because of its vascularity a bruit may be audible.

INVESTIGATIONS

The diagnosis is usually obvious clinically. Raised T_3 and T_4 levels, coupled with low TSH levels, are confirmatory. Isotope- scanning shows a diffuse uptake. Elevated thyroid-stimulating immunoglobulin (TSI) are diagnostic of Graves' disease and are increased in approximately 90% of patients.

TREATMENT

The hyperthyroid symptoms require treatment with antithyroid drugs. These drugs block the incorporation of iodine into tyrosine and so prevent the synthesis of T_3 and T_4. They include Carbimazole. Side effects of carbimazole include rashes and agranulocytosis. These side effects are not shared with Propylthiouracil.

Many consider treatment with Radioactive iodine (RAI) to be the treatment of choice. If ablative doses of iodine are used, patients require thyroxine replacement. Thyroidectomy is a highly successful form of treatment for many patients, especially younger ones and also require thyroxine replacement.

TOXIC MULTINODULAR GOITER

A toxic multinodular goitre is responsible for thyrotoxicosis in about 25% of patients. It is more common in older patients.

ETIOLOGY

There is usually a long-standing nontoxic goiter in which one or more nodules become hyperactive and begin to function independently of TSH levels.

CLINICAL FEATURES

Patients usually have an insidious onset of hyperthyroidism. Patients usually have nodular enlargement of the gland.

INVESTIGATIONS

Thyroid functions tests show a rise in thyroid hormones with a decrease in the level of TSH. In a toxic multinodular goitre, the isotope scan demonstrates one or more areas of increased uptake.

TREATMENT

Hyperthyroidism must be adequately controlled with antithyroid drugs. Surgical resection is the preferred treatment method for patients with toxic multinodular goitre, with subtotal thyroidectomy being the standard procedure.

TOXIC ADENOMA

A single functioning adenoma is a rare cause of thyrotoxicosis (1-2% of patients). This is referred to as toxic adenoma or plummer's disease. The adenoma secretes thyroid hormones autonomously, TSH secretion is completely suppressed, and the remainder of the gland is non-functional. Physical examination usually reveals a solitary thyroid nodule without palpable thyroid tissue on the contralateral side. Isotope scanning show a hot nodule with suppression of the rest of the thyroid gland.

Patients with hyperthyroidism should be controlled (hyperthyroidism occur when the nodule is more than 3 cm in size). RAI ablation can be used for smaller nodules. Surgery (lobectomy and isthmusectomy) is recommended to treat young patients and those with larger nodules.

MALIGNANT GOITRES

Malignant goitres represent either primary or metastatic tumours. Primary malignant tumours are more common in females with a female to male ratio of 3.

PAPILLARY CARCINOMA

Papillary carcinoma is a thyroid gland primary malignant tumor that is derived from the follicular

epithelium and is the most frequent malignant thyroid tumor.

PATHOLOGY AND ETIOLOGY

Histologically, a papillary tumor is formed of complex papillary folds lined by several layers of cuboidal cells with orphan annie nuclei (empty nuclei) that project into what appear to be cystic spaces (Figure 11). The fibrovascular stroma often contains characteristic calcified 'psammoma bodies'. The tumor is usually mixed with normal follicular structure.

These tumors seem to show multiple foci throughout the gland mainly due to the rich lymphatic network. The tumors have high incidence of lymphatic spread but low incidence of spread by the blood root.

The incidence of these tumors increases markedly with radiation exposure and was most notable after the Chernobyl disaster in Ukraine.

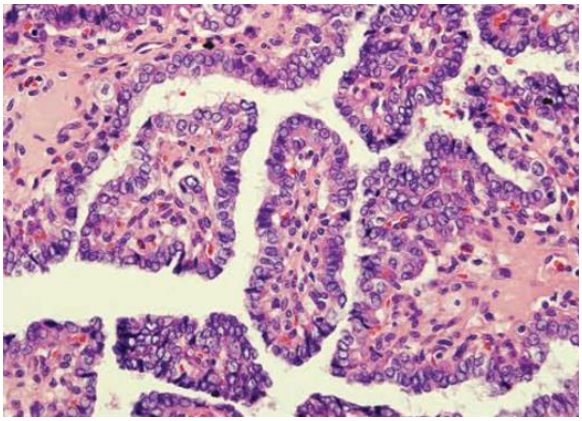


Figure 81 histopathology of Papillary carcinoma

CLINICAL FEATURES

This tumor is rare after the age of 40 years and presents as a slow-growing solitary thyroid swelling that is not particularly hard. Patient may also present with lymphadenopathy without any appearing thyroid swelling (lateral aberrant thyroid). Presentation with metastatic features is rare.

INVESTIGATIONS

Patients are usually approached as having a solitary thyroid nodule (refer to that seminar). Diagnosis is made by FNA.

TREATMENT

The disease is commonly multifocal, so that total thyroidectomy is indicated. Involved lymph nodes are removed. Hormone replacement therapy is needed.

The disease has an excellent prognosis, with 10-year survival rates approaching 90%.

FOLLICULAR CARCINOMA

Follicular carcinoma is a primary malignant thyroid cancer that is derived from the cuboidal epithelium of the follicles. It is second most frequent thyroid tumor after papillary carcinoma.

PATHOLOGY

Histologically, malignant cells are arranged in solid masses with rudimentary acini. Vascular and capsular invasion characterize this neoplasm and distinguish it from a benign follicular adenoma.

A subtype of follicular carcinoma; Hurthle cell carcinomas, account for approximately 3% of all thyroid malignancies. These tumors contain sheets of eosinophilic cells packed with mitochondria, which are derived from the oxyphilic cells of the thyroid gland.

CLINICAL FEATURES

This disease typically presents as a solitary thyroid nodule in patients aged 30-50 years. Lymph node metastases are much less common than hematogenous spread, and 20% of patients have deposits in the lungs, bone or liver.

INVESTIGATIONS

As in other thyroid malignancies, this tumor is approached as solitary thyroid nodule. These tumors cannot be diagnosed with FNA, as they differ from follicular adenomas only by capsular and vascular invasion.

TREATMENT

management depends on the extent of local invasion. A tumor with only microinvasion of the capsule has a *very* good prognosis and only requires lobectomy. If, however, there is gross capsular invasion or vascular invasion (or known remote metastases), total thyroidectomy is performed which is followed by RAI ablation of the metastatic lesions detected by radioisotope scan.

Patients with non-metastatic disease has a prognosis of 90% survival at 10 years.

PROGNOSTIC INDICATORS

papillary and follicular cancers are referred to as differentiated thyroid cancers. Several prognostic indicators have been incorporated into various staging systems, which enable patients with those types of cancers to be stratified into low-risk and high-risk groups. These scoring systems are used to plan the surgical treatment.

researchers at the Mayo Clinic, proposed the AGES scoring system, which incorporates *age,* histologic grade, extrathyroidal invasion and metastases, and tumor size to predict the risk of dying from papillary cancer (see the table below).

Low-risk patients	high-risk patients
• **Young**	• older
• **well-differentiated**	• poorly differentiated local invasion and distant metastases
• **no metastases**	
• **small primary lesions**	
	• large primary lesions

The MACIS scale is a more sophisticated postoperative system modified from the AGES scale. This scale incorporates distant metastases, age at presentation, completeness of original surgical resection, extrathyroidal invasion, and size of original lesion and classifies patients into four risk-groups based on their scores.

Other investigators had proposed the AMES system to classify differentiated thyroid tumors into low- and high-risk groups using age (men <40 years, women <50 years), metastases, extrathyroidal spread, and size of tumors (5 cm).

ANAPLASTIC CARCINOMA

These rapidly growing, highly malignant tumors tend to occur in older patients. local invasion may involve the recurrent laryngeal nerve(s) and cause hoarseness, or compress the trachea and cause dyspnea and stridor, and/or compress the oesophagus and cause dysphagia. Invasion of the cervical sympathetic nerves may cause Horner's syndrome. Pulmonary metastases are common. Death usually occurs within 6 months of diagnosis.

Resection is rarely possible, but surgery can relieve tracheal compression. Radiotherapy and chemotherapy are of marginal value.

MEDULLARY CARCINOMA

This tumor arises from the parafollicular C cells. There is hard enlargement of one or both thyroid lobes, and in 50% of patients the cervical lymph nodes are involved. It usually presents as a thyroid nodule in a sporadic form (70% of cases) but can be familial or associated with multiple endocrine neoplasia (see later). Calcitonin levels are elevated and can be used to monitor progress and screen relatives, postoperative calcitonin measurement is a useful tumor cell marker and can be used as a method of follow-up.

TREATMENT

consists of total thyroidectomy and dissection of the lymph nodes in the central compartment of the neck.

THYROID LYMPHOMA

This rare tumor usually presents in the elderly female with a background history of autoimmune (Hashimoto's) thyroiditis as a rapidly growing thyroid nodule. It can also occur as a primary tumor that originates in an otherwise normal gland. It may be diagnosed by FNA or Tru cut biopsy. As for

all lymphomas, it should be staged with a bone marrow aspirate and CT scan of the chest and ab

domen. If confined to the

thyroid alone, it may be treated by thyroid lobectomy with subsequent adjuvant radiotherapy and chemotherapy; otherwise it is treated by chemoradiation alone.

APPROACH TO PATIENT WITH SOLITARY THYROID NODULE

A solitary or isolated thyroid nodule is one which clinically appears to be isolated without apparent pathology in the rest of the gland (figure 12). the preferred term is dominant thyroid nodule for a similar swelling in a gland with clinical evidence of generalized abnormality in the form of a palpable contralateral lobe or generalized mild nodularity (e.g. multinodular goitre). The term discrete thyroid nodule refers to both solitary and dominant nodules in general. About 70% of discrete thyroid swellings are clinically isolated and about 30% are dominant.

ETIOLOGY

Causes of an isolated thyroid nodule are shown in the table below. The importance of discrete swellings lies in the increased risk of neoplasia compared with other thyroid swellings. Some 15% of isolated swellings prove to be malignant, and an additional 30-40% are follicular adenomas.

APPROACH TO THE PATIENT

the approach to the patient should focus on identifying which patients at risk of having malignancy and thus who might benefit from surgery. The approach begins with history and examination.

HISTORY

causes of a solitary thyroid nodule
Benign (85%) • **Cysts** • **Follicular Adenomas Toxic Adenoma Non-toxic adenoma** • **thyroiditis**
Malignant (15 %) • **Primary thyroid carcinomas** • **lymphoma or metastatic disease (Far less commonly)**

Details regarding the nodule, such as time of onset, change in size, and associated symptoms, such as pain, dysphagia, dyspnea, features which might suggest malignancy include;

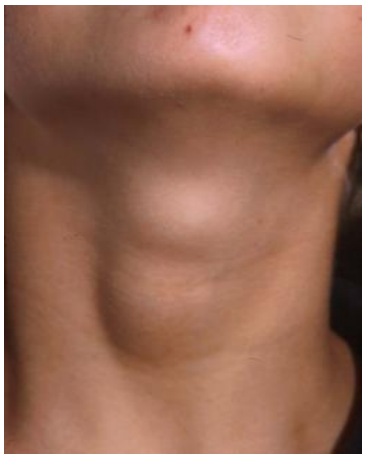


Figure 82 a solitary thyroid nodule

- a nodule of recent origin that is Increasing in size rapidly.
- A solitary thyroid nodule in a young patient less than 15 years or an old patient over 65 years.
- pain; Patients with medullary thyroid cancer may complain of a dull, aching sensation.
- A history of hoarseness is worrisome because it may be secondary to malignant involvement of the recurrent laryngeal nerves.
- History of exposure to ionizing radiation is of utmost importance as it is associated with high risk of malignancy. Patients should be inquired wither any member of the family has or had got thyroid cancer or any other endocrine cancer (MEN II in association with medullary thyroid cancer).

PHYSICAL EXAMINATION

A thyroid nodule moves on swallowing. Features of a thyroid nodule on examination which suggest malignancy include;

- Firm, Fixed and irregular
- Presence of cervical lymphadenopathy

INVESTIGATIONS

The objectives of investigation are as follows;

- Determine if nodule is autonomously functioning and possibly causing hyperthyroidism.
- Determine if nodule has high risk of malignancy.

DETERMINING NODULE FUNCTION

Thyroid function tests will direct further approach and should precede consideration of imaging studies and FNA biopsy. Patient with a low TSH level (toxic nodules) should be considered for radioisotope scans.

radioisotope scans in evaluation of solitary nodules should be limited to patients with a low TSH level to identify autonomously functioning nodules. Nodules with increased uptake (hot nodules) are toxic adenomas and almost never malignant.

Nodules that accumulate radioisotopes equal to surrounding tissue (warm nodules), or nodules with low uptake (cold nodule), are most often benign, But may be malignant and therefore require FNA biopsy.

radioisotope scans are unnecessary in setting of a normal TSH level, and one may proceed directly to FNA biopsy.

DETERMINING THE RISK OF MALIGNANCY

Ultrasonography Often reveals multinodular goitre rather than solitary thyroid nodule. It can be used to guide needle placement during FNA biopsy as it Provides visualization of needle tip during procedure to ensure accurate sampling. It may reveal sonographic features that may be suggestive of cancel;

- Irregular margins
- intranodular vascular spots
- microcalcifications
- hypoechogenicity within the nodule

FNA (fine needle aspiration) biopsy is the most accurate test for the evaluation of thyroid nodules. Tissue samples are obtained for cytologic analysis using 22- to 25-gauge needles, with or without local anaesthesia. Specimen adequacy requires ≥ 2 slides showing ≥ 6-8 cell clusters. False-positive and false-negative results are uncommon when a high-quality sample is obtained.

A cytopathologist cannot distinguish between a follicular adenoma and follicular carcinoma; this can only be achieved on definitive histopathology by looking for capsular or vascular invasion.

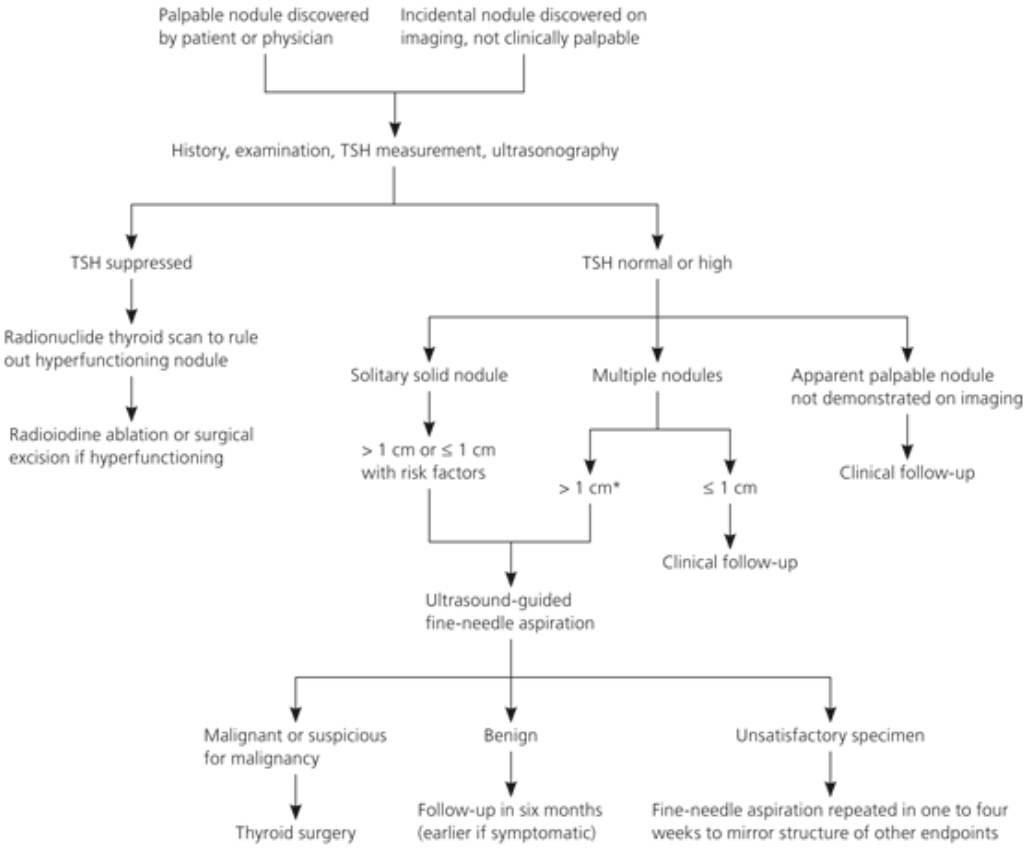
MANAGEMENT

The management mainly depends on the result of the cytology sample from the FNA specimen. Malignant lesions require surgical resection with the appropriate modality corresponding to the type of cancer (refer to the thyroid gland lecture, head line malignant goitres).

Benign lesions should be removed as indicated. usually if asymptomatic can be left alone and monitored periodically. Since the malignant nature of follicular neoplasms cannot be determined by cytology, surgical resection is indicated.

About 30% of FNA samples turns out to be thyroid cysts. Aspiration will resolve 25-50% of cysts, but fluid re-accumulation is common. Surgical resection is indicated for growing or painful cysts.

Toxic adenoma can be treated with RAI ablation, surgery and antithyroid drugs.


Palpable nodule discovered by patient or physician
Incidental nodule discovered on imaging, not clinically palpable
History, examination, TSH measurement, ultrasonography
TSH suppressed
TSH normal or high
Radionuclide thyroid scan to rule out hyperfunctioning nodule
Radioiodine ablation or surgical excision if hyperfunctioning
Solitary solid nodule
Multiple nodules
Apparent palpable nodule not demonstrated on imaging
> 1 cm or ≤ 1 cm with risk factors
> 1 cm*
≤ 1 cm
Clinical follow-up
Clinical follow-up
Ultrasound-guided fine-needle aspiration
Malignant or suspicious for malignancy
Benign
Unsatisfactory specimen
Thyroid surgery
Follow-up in six months (earlier if symptomatic)
Fine-needle aspiration repeated in one to four weeks to mirror structure of other endpoints

PITUITARY GLAND

The pituitary gland is the master gland in the body and serves as the major link between the hypothalamus with the organs outside the nervous system, it is small (6,9,12 mm in dimensions) and weighs about 600 mg.

The pituitary has two parts: the anterior pituitary (adenohypophysis) and the posterior pituitary (neurohypophysis), It is enclosed within a bony shell, the sella turcica (sphenoid bone), which is sealed superiorly by a fold of dura mater, the diaphragma sellae. The infundibulum connects the pituitary to the hypothalamus.

Master gland serves as the major link between hypothalamus with the organs outside the nervous system.

THE ANTERIOR PITUITARY → DERIVED FROM ORAL ECTODERM (RATHKE'S POUCH)

The anterior pituitary contains solid cords of secreting cells that used to be classified as acidophil, basophil or chromophobe on staining with haematoxylin and eosin. The anterior pituitary secrets the following hormones;

- Thyroid stimulating hormone (TSH) or thyrotropin; targets the thyroid gland and triggers the release of thyroid hormones.

- Adrenocorticotropic hormone (ACTH), also known as corticotropin, stimulates the release of steroid hormones by the adrenal cortex.

- Follicle stimulating hormone (FSH) and luteinizing hormone (LH) are called gonadotropins, because they regulate the activities of the gonads. Follicle stimulating hormone, promotes follicle development in females and, in combination with luteinizing hormone, stimulates the secretion of estrogens) by ovarian cells. Luteinizing hormone induces ovulation.

- Prolactin (PRL), works with other hormones to stimulate mammary gland development. In pregnancy and during the nursing period that follows delivery, PRL also stimulates milk production by the mammary glands. Prolactin production is inhibited by prolactin inhibiting hormone (PIH) the neurotransmitter dopamine.

- Growth hormone (GH), or somatotropin, stimulates cell growth and replication by accelerating the rate of protein synthesis.

- Melanocyte stimulating hormone (MSH): secreted form pars intermedia a small rudimentary part in anterior pituitary, released during fetal life and along with estrogen giving hyperpigmented skin to pregnant mothers, becomes significant in pathologies that increase the release of ACTH (cushing's disease, addison's disease) because both of them have the same precursor proopiomelanocortin (POMC).

The infundibulum contains a portal venous system (figure 1) that connects capillaries in the median eminence of the hypothalamus with capillaries and sinusoids of the anterior pituitary. This system carries neurosecretory hormones that stimulate or inhibit specific endocrine cells in the pituitary. The most important messengers are GH-releasing and inhibiting factors, corticotrophin-releasing factor (CRF), gonadotrophin-releasing hormone (GnRH), TRH arid prolactin-inhibiting factor (PIF).

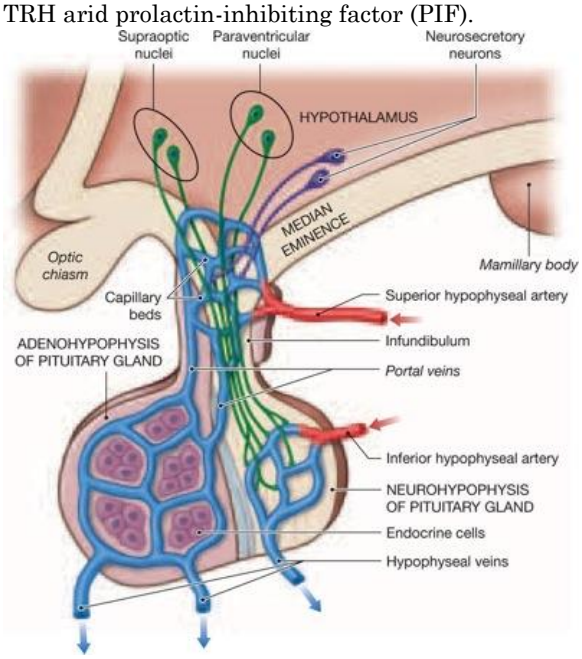


Figure 83 anatomy of the pituitary gland

THE POSTERIOR PITUITARY → DERIVED FROM NEUROECTODERM

ADH and Oxytocin are released from nuclei in the hypothalamus (supraoptic & paraventricular), fibers pass from these nuclei via the infundibulum to the posterior pituitary. The nerve cells secrete antidiuretic hormone (ADH) and oxytocin, both of which pass down the nerve fibers by the help of carrier proteins (neurophysins) to be stored in vesicles in the pituitary.

Supraoptic nucleus mainly releases ADH, Paraventricular nucleus mainly releases Oxytocin

The antidiuretic hormone is released in response to low blood volume and high serum osmolarity and promotes water retention at the kidneys (thus concentrating urine). In women, oxytocin stimulates smooth muscle tissue in the wall of the uterus, promoting labor and delivery. After delivery, oxytocin stimulates the contraction of myoepithelial cells around the secretory alveoli and the ducts of the mammary glands, promoting the ejection of milk. Oxytocin is believed to be **involved in ejaculation** by increasing **sperm** number and contracting ejaculatory tissues.

PITUITARY TUMORS:

Mostly benign adenomas (PROLACTENOMA)

PATHOLOGY

The major effects of a pituitary tumor can be categorized into those of mass-pressure effect and those of hormone hypersecretion (hyperpituitarism).

MASS-PRESSURE EFFECT

compression of the optic chiasma → bitemporal hemianopia

compression of Cavernous sinus → paralysis of extraocular muscles (damaged III, IV, VI nerves)

compression of Internal Carotid artery → cerebral infarction

Increased intracranial pressure → headache, nausea, vomiting, altered mental status/coma

Obstruction of the 3rd ventricle → Closing the aqueduct of sylvius → hydrocephalus

Hypersecretion symptoms: any hormone from the pituitary gland can be oversecreted. However, most of pituitary adenomas are prolactinomas.

Features of note in the initial assessment include any history of galactorrhea (suggestive of prolactinoma), and Cushingoid or acromegalic features pointing to ACTH- or growth hormone-secreting tumors, respectively. Baseline assessment of pituitary function should include serum prolactin, follicle stimulating hormone and luteinizing hormone together with testosterone in males or estradiol in females, thyroid function tests and fasting serum growth hormone and cortisol. Preoperative prolactin levels are crucial since prolactinomas may be managed without the need for surgery. Prolactinomas are managed initially with dopamine agonists such as bromocriptine and cabergoline. Growth hormone-secreting tumors may also respond to dopamine agonists or to somatostatin analogues such as octreotide. The cortisol level is also important, since deficiency must be corrected, especially in the perioperative period. Diagnosis of ACTH-secreting tumors can be difficult and may require the use of specialized tests such as petrosal sinus sampling and the dexamethasone suppression test. Effective treatment requires close cooperation between the neurosurgical team and an endocrinologist. Compression of the chiasm with any evidence of visual compromise is the main indication for urgent surgical intervention. Surgical resection is usually performed by a transsphenoidal approach through the nose, using a microscope or endoscope. Sometimes large tumors also require a craniotomy. After operation patients are at risk of CSF leak (3%), and pituitary insufficiency. Diabetes insipidus resulting from manipulation of the pituitary stalk is common in the immediate postoperative period and usually resolves spontaneously. Where it is suspected, the patient will require hourly measurement of urine output, and blood and urine samples for calculation of sodium concentration and osmolality. If confirmed, the condition can be managed with DDAVP in consultation with endocrinology.

Other pituitary pathologies:

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE HYPERSECRETION (SIADH)

is characterized by excessive release of antidiuretic hormone from the posterior pituitary gland which might result in a decreased blood osmolarity and fluid overload and concentrated urine. **Caused** by: ectopic ADH (small lung ca), pulmonary disease, medications (cyclophosphamide)

Treatment: fluid restriction (first line), salt tablets, IV hypertonic saline, diuretics, ADH antagonists (eg, conivaptan, tolvaptan, demeclocycline).

CENTRAL DIABETES INSIPIDUS

is characterized by diminished production of the ADH that causes severe thirst and excessive production of very dilute urine (polyuria) which can lead to dehydration. In contrast to nephrogenic type which is caused by resistance to the action of ADH. Mostly it's idiopathic.

Treatment involve giving ADH in the form of desmopressin + hydration

CUSHING'S DISEASE

excess Cortisol from overproduction of ACTH from the pituitary gland/CRH from hypothalamus

Clinical findings:

Hypertension, weight gain, moon facies, truncal obesity, buffalo hump, skin changes (eg, thinning, striae), hirsutism, osteoporosis, hyperglycaemia (insulin resistance), amenorrhea, immunosuppression.

Diagnosis

Screening tests include: t free cortisol on 24-hr urinalysis, t late night salivary cortisol, and no

suppression with overnight low-dose dexamethasone test.

Treatment:

The first-line treatment of Cushing's disease is surgical resection of ACTH-secreting pituitary adenoma; this surgery involves removal of the tumour via transsphenoidal surgery (TSS). Pituitary radiation therapy is another option for treatment of postoperative persisting hypercortisolemia following unsuccessful transsphenoidal surgery.

ACROMEGALY

caused by excess GH in adults from pituitary adenoma

Clinical findings:

Large tongue with deep furrows, deep voice large hands and feet, coarsening of facial growth features with aging, frontal bossing, diaphoresis (excessive sweating), impaired glucose tolerance (insulin resistance), hypertension. increased risk of colorectal polyps and cancer.

Diagnosis:

increased serum IGF-1; failure to suppress serum GH following oral glucose tolerance test; pituitary mass seen on brain MRI.

Treatment:

Pituitary adenoma resection. If not cured, treat with octreotide (somatostatin analog) or pegvisomant (CH receptor antagonist), dopamine agonists (eg, cabergoline).

SHEEHAN SYNDROME

one of the causes of hypopituitarism, caused by pituitary infarction after postpartum bleeding

Clinical findings: agalactorrhea (failure of lactation), amenorrhea, hypothyroidism

Treatment: Lifelong hormone replacement therapy for the hormones that are missing

INVESTIGATIONS

The clinical picture of a patient with a pituitary adenoma determines which investigation is to be taken. The endocrine evaluation of these patients is shown in the table below.

Endocrine evaluation for pituitary-tumors

- **Prolactin**
- **Growth hormone**
- **Luteinizing hormone, Follicle stimulating hormone, Testosterone and Estrogen.**
- **Cortisol and ACTH**
- **Electrolytes**
- **Glucose**
- **Thyroid function tests**
- **Urine specific gravity (sodium; diabetes insipidus)**

Assessment of the tumor itself by imaging can be done by a CT scan but is best with MRI. Angiography might be needed in cases of cerebral infarction caused by internal carotid artery invasion.

MANAGEMENT

The main stay of treatment of pituitary adenomas is surgical excision **(hypophysectomy)** in conjunction with radiation therapy. In cases of functioning pituitary tumors medical treatment might be required.

After **total hypophysectomy,** replacement therapy is required for life.

THE PARATHYROID GLANDS

ANATOMY

There are two pairs of parathyroid glands, each weighing 40-50 mg. The upper glands arise from the fourth branchial arch and are usually found at the back of the thyroid above the inferior thyroid artery. The lower glands arise from the third arch (in association with the thymus) and are less constant in position. They are usually found posterior to the lower pole of the thyroid lobes but can lie within the gland, some distance below it, in the upper mediastinum or within the thymus. These glands receive a rich blood supply from the inferior thyroid artery.

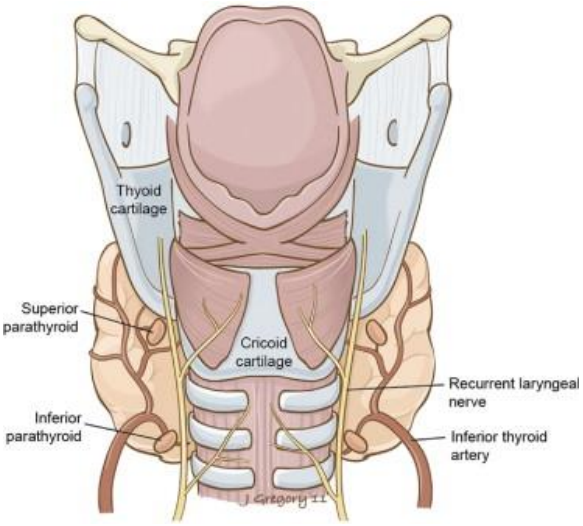


Figure 84 anatomy of the parathyroid gland

HISTOLOGY

Histologically, the glands contain chief (principal) cells (classified as dark, light and water-clear) that secrete the parathyroid hormone. After the age of 5-7 years, oxyphil cells appear; their function is unknown.

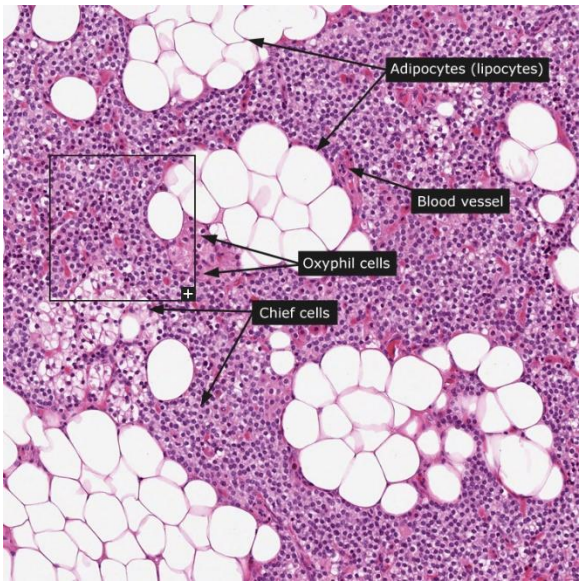


Figure 85 Normal histology of the parathyroid

PHYSIOLOGY

Plasma calcium levels are kept constant in the range 2-2.5 mmol/L (8.5-10.3 mg/dL). The level is regulated by the parathyroid hormone, vitamin D and to a lesser degree by the hormone calcitonin.

When the calcium concentration of the blood falls below normal, the chief cells secrete parathyroid hormone (PTH), or parathormone . The net result of PTH secretion is an increase in calcium concentration in body fluids. Parathyroid hormone has the following effects (figure);

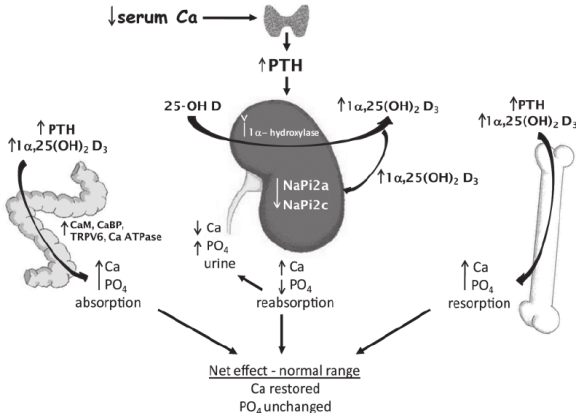
• It stimulates osteoclasts, accelerating mineral turnover and the release of calcium from bone. It also inhibits osteoblasts, reducing the rate of calcium deposition in bone.

• It enhances the reabsorption of calcium at the kidneys, reducing urinary losses.

• It stimulates the formation and secretion of 1,25-dihydroxyvitamin D_3 (vitamin D) at the kidneys. In general, the effects of calcitriol complement or enhance those of PTH, but one major effect of calcitriol is the enhancement of calcium and phosphate absorption by the digestive tract.

Calcitonin is produced by thyroid C cells and functions as an antihypercalcaemic hormone by inhibiting osteoclast-mediated bone resorption. At the kidney, calcitonin increases phosphate excretion by

inhibiting its reabsorption. It has minimal or no role in calcium regulation.



HYPERPARATHYROIDISM

Hyperfunction of the parathyroid glands or hyperparathyroidism may be classified as primary, secondary, or tertiary.

PRIMARY HYPERPARATHYROIDISM

Primary hyperparathyroidism (PHPT) arises from increased PTH production from abnormal parathyroid glands and results from a disturbance of normal feedback control exerted by serum calcium which results in hypercalcaemia and hypophosphatemia.

ETIOLOGY

In 85% of patients, primary hyperparathyroidism is due to an adenoma, in 12% it results from hyperplasia (usually affecting all four glands), and in less than 1% it results from parathyroid carcinoma. The causes of primary hyperparathyroidism are shown in the table below, primary hyperparathyroidism is also seen in M.E.N I and II.

Causes of primary hyperparathyroidism

- **Solitary Adenoma (Autonomous); 85%**
- **Four glands hyperplasia; 10%**
- **two glands hyperplasia; 2%**
- **carcinoma; 1%**

CLINICAL FEATURES

Women are affected twice as often as men. The disease can be asymptomatic. The symptoms of primary hyperparathyroidism can be remembered by the aide memoire; "bones, stones, abdominal groans and psychic moans".

Renal effects include nephrocalcinosis and the formation of urinary calculi owing to increased excretion of phosphate. Polyuria is an early sign of hyperparathyroidism. Bone effects include gross demineralization (osteopenia), subperiosteal bone resorption (seen typically in the middle and distal phalanges of the fingers, figure 4), cysts in the long bones and jaw, and the moth-eaten appearance of the skull gave rise to the descriptive term 'osteitis fibrosa cystica' (figure 5). Multiple pathological fractures were also once common.

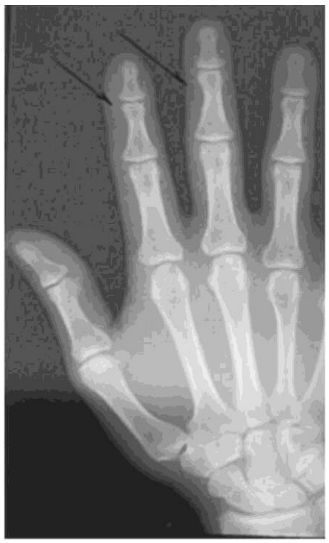


Figure 4 X-ray of the hand showing subperiosteal bone resorption most apparent along the radial aspect of the middle phalanx. characteristic of osteitis fibrosa cystica.

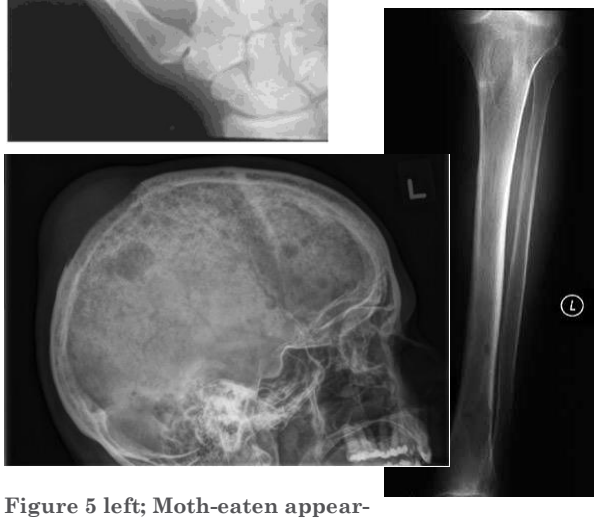


Figure 5 left; Moth-eaten appearance of the skull, right; Osteitis fibrosa cystica of the tibia. Arrows point to the brown tumors which are typical.

Other manifestations of hyperparathyroidism include peptic ulceration, acute and chronic pancreatitis, lethargy, muscle weakness and psychotic symptoms.

Rarely, patients present with a hypercalcaemic crisis characterized by marked hypercalcaemia (> 3.5 mmol/l), mental confusion, nausea and vomiting.

INVESTIGATION

The plasma levels of calcium and parathyroid hormone are usually high with a decrease in the plasma phosphate, Alkaline phosphatase (skeletal) levels may be raised, even if there is no radiological evidence of bone disease.

Radiological techniques (radioisotope scanning (sesamoid) or ultrasound) along with surgical exploration are used to identify the gland pathology.

MANAGEMENT

Surgery is the only definitive treatment for primary hyperparathyroidism. In cases of adenoma or less commonly carcinoma, the abnormal gland is completely removed and the other glands left in situ. When there is glandular hyperplasia, all four glands are removed (total parathyroidectomy). Some surgeons leave a small parathyroid tissue

SECONDARY HYPERPARATHYROIDISM

In secondary hyperparathyroidism, there is a chronic abnormal stimulus to PTH production and the parathyroid glands undergo diffuse hyperplasia. This occurs most commonly in chronic renal failure (including nearly all patients on dialysis to some degree) but may also occur in vitamin D deficiency.

In chronic renal failure the glomeruli are damaged, so that there is retention of phosphate which can lead to hyperphosphatemia. Hyperphosphatemia inhibits calcium absorption by the gut. In addition, Renal tubular injury leads to reduced renal production of 1,25- dihydroxyvitamin D3. The low blood calcium that results causes the negative feedback system to increase elaboration of PTH. This promotes osteoclastic activity, restoring calcium levels but causing the bone disorders of osteomalacia and osteitis fibrosa cystica. The raised product of calcium and phosphate in the blood leads to ectopic calcification in a variety of abnormal sites.

These patients show high serum levels of phosphate and parathormone with low serum calcium. The condition is managed initially by giving 1-α-hydroxyvitamin D3 (alfacalcidol) to increase calcium absorption or phosphate binders. Long term treatment involves dietary restriction. Renal transplantation is definitive in most of the cases.

TERTIARY HYPERPARATHYROIDISM

Excessive PTH secretion in secondary hyperparathyroidism may become autonomous; it is then termed tertiary hyperparathyroidism. This may occur after renal transplantation. Patients tend to have normal or high calcium level with high PTH and normal phosphate level. Total parathyroidectomy may be needed, with autotransplantation of parathyroid tissue (equivalent in size to one normal gland) into an arm muscle (where it can be readily located if problems persist).

HYPOPARATHYROIDISM

The most common cause of hypoparathyroidism is surgical removal or devascularisation of the parathyroid glands during thyroid or parathyroid surgery.

CLINICAL FEATURES

Hypoparathyroidism presents clinically with the effects of hypocalcaemia. A fall in the plasma calcium level increases neuromuscular excitability causing cramps or even tetany in severe cases. An early symptom of hypocalcaemia is paraesthesia, especially around the lips. Clinical tests for hypocalcaemia include tapping over the parotid gland. This provokes transient contraction of the facial muscles and is known as Chvostek's sign. A further test involves inflating a sphygmomanometer cuff on the upper arm to above systolic pressure. This induces

carpal spasm within about 3 minutes (Trousseau's sign).

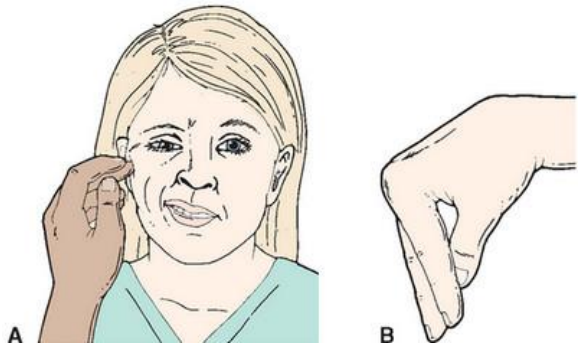


Figure 6 tetany; Chvostek's sign and Trousseau's sign

MANAGEMENT

Acute symptomatic hypocalcaemia is a medical emergency and requires urgent correction by intravenous injection of calcium.

Oral calcium supplemented by 1-3 µg daily of 1-a-vitamin D3 if necessary, should be given with a view to gradual withdrawal over the next 3-12 months. After that patient should be put on a diet high with calcium and vitamin D.

THE ADRENAL GLANDS

ANATOMY AND BLOOD SUPPLY

The adrenal glands are situated at the upper poles of the kidneys in the retroperitoneum. The weight of a normal adrenal gland is approximately 4 g. A rich blood supply is essential for the optimal function of the adrenal glands. Each gland is supplied by the superior, middle and inferior adrenal arteries, which arise from the inferior phrenic artery, abdominal aorta and renal artery respectively. The left adrenal vein drains into the left renal vein while the right adrenal vein drains into the inferior vena cava.

HISTOLOGY

The adrenal gland is divided into two parts: a superficial adrenal cortex and an inner adrenal medulla. The cortex, like the gonads, is derived from mesoderm. The outer zona glomerulosa secretes the mineralocorticoid, aldosterone. The zona fasciculata and zona reticularis act as a functional unit and secrete glucocorticoids (cortisol) and androgenic steroids.

The medulla is derived from the neural crest cells. its cells (called chromaffin) secrete the catecholamines, adrenaline (epinephrine), noradrenaline (norepinephrine) and dopamine, and is supplied by pre-ganglionic sympathetic nerves.

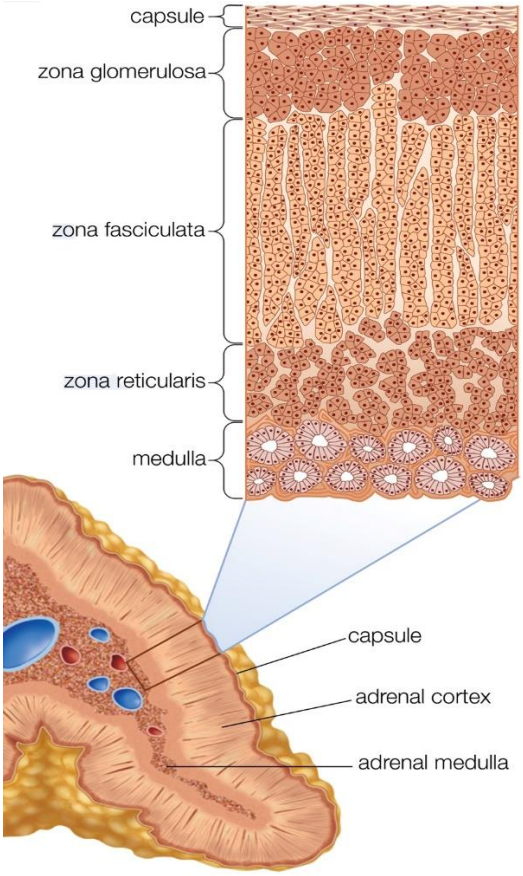


Figure 87 the adrenal cortex an medulla

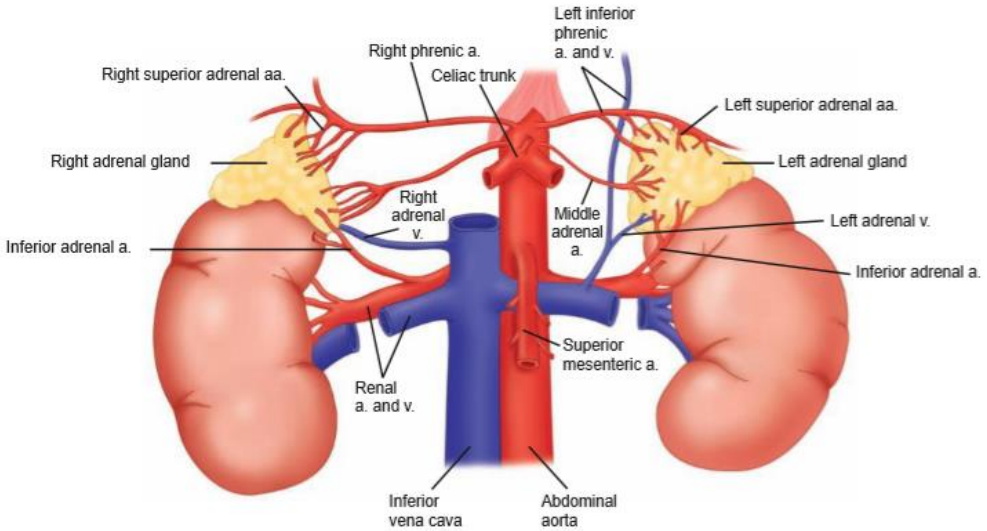


Figure 86 anatomy of the adrenal glands

PHYSIOLOGY OF THE ADRENAL CORTEX

The zona glomerulosa cells produces the hormone aldosterone. Aldosterone increases the blood pressure by promoting sodium and water retention in the kidneys. It also promotes potassium loss at the same site. The two well-known stimulators of aldosterone secretion are angiotensin II (released by activation of the renin-angiotensin system after hyponatremia or hypovolemia) and an increase in the level of serum potassium (hyperkalemia).

Cells of the zona fasciculata and zona reticularis synthesize cortisol and the adrenal androgens. Cortisol secretion is regulated by adrenocorticotrophic hormone (ACTH), which is produced by the anterior pituitary gland. The hypothalamus controls ACTH secretion by secreting corticotropin-releasing hormone (CRH). The cortisol level inhibits the release of CRH and ACTH via a closed-loop system (figure 3). Cortisol is the most important steroid and has numerous metabolic and immunological effects

HYPERALDOSTERONISM

PRIMARY HYPERALDOSTERONISM

Primary hyperaldosteronism, also known as Conn's syndrome, is a disease of the adrenal cortex that is characterized by hypersecretion of the hormone aldosterone.

AETIOLOGY

In about 70% of the cases, the cause is a benign adrenal adenoma and is most common in young or middle-aged women. The adenoma is small, single, canary yellow on bisection and composed of cells of the glomerulosa type. Only rarely is the syndrome due to adrenal carcinoma, bilateral adrenal hyperplasia or multiple microadenomas.

CLINICAL FEATURES

Retention of sodium increases plasma volume and produces hypertension, often in association with headaches and visual disturbance. Muscle weakness may result from hypokalemia.

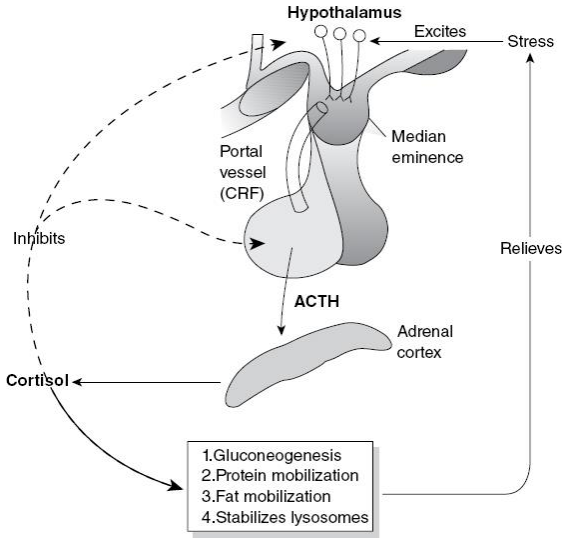


Figure 88 regulation of cortisol secretion

INVESTIGATIONS

Because aldosterone promotes potassium loss patients with primary hyperaldosteronism have hypokalemia in addition to hypertension, the syndrome can progress to severe hypokalemic alkalosis. Patients have high serum or urinary aldosterone levels, measurement of plasma renin is the critical investigation to exclude secondary hyperaldosteronism; renin levels are increased in secondary hyperaldosteronism but undetectable in the primary disease.

If primary hyperaldosteronism is confirmed biochemically, attempts should then be made to localize the adenoma by CT scan

MANAGEMENT:

Primary hyperaldosteronism due to an adenoma or carcinoma is treated by removal of the affected gland (unilateral adrenalectomy) after correcting the hypokalemia with oral potassium and spironolactone. Bilateral hyperplasia can be cured with (bilateral adrenalectomy).

SECONDARY HYPERALDOSTERONISM

Hyperaldosteronism is most commonly secondary to excessive renin secretion (and stimulation of the zona glomerulosa by angiotensin) in chronic liver, renal or cardiac disease. This is referred to as secondary hyperaldosteronism. It is distinguished

from primary hyperaldosteronism by the high renin level.

CUSHING'S SYNDROME

Cushing's syndrome is a syndrome caused by hypersecretion of the hormone cortisol and was first described by the American neurosurgeon, Harvey Cushing.

AETIOLOGY

The causes of Cushing's syndrome are shown in the table below.

Causes of Cushing's syndrome
ACTH dependant • **Pituitary Adenoma (Cushing disease)** • **Ectopic ACTH secretion**
ACTH independent • **Adrenal Adenoma** • **Adrenal Carcinoma** • **Bilateral Hyperplasia** • **Iatrogenic**

Adrenal adenoma is the most common adrenal cause of Cushing's syndrome. It is unilateral and the contralateral gland is usually atrophied (due to ACTH inhibition). the tumor contains clear cells like those of the zona fasciculata. Adrenal carcinoma is a rare cause of Cushing's syndrome that occurs more frequently in young adults and children. Pituitary tumors causing Cushing's syndrome are of ACTH-secreting cells. Cushing's syndrome caused by a pituitary tumor is referred to as cushing's disease. Inappropriate secretion of ACTH-like peptide by tumors of non-pituitary origin (e.g. pancreas, bronchus, thymus) is a rare cause. This is referred to as ectopic ACTH secretion.

The most striking feature is truncal obesity, a 'buffalo hump' (due to redistribution of water and fat) and 'mooning' of the face (figure 4). As a result of protein loss, the skin becomes thin, with purple striae, dusky cyanosis and visible dermal vessels. Proximal muscle weakness is prominent. Other features include increased capillary fragility, purpura, osteoporosis, acne, loss of libido, hirsutism, diabetes, hypertension and amenorrhea.

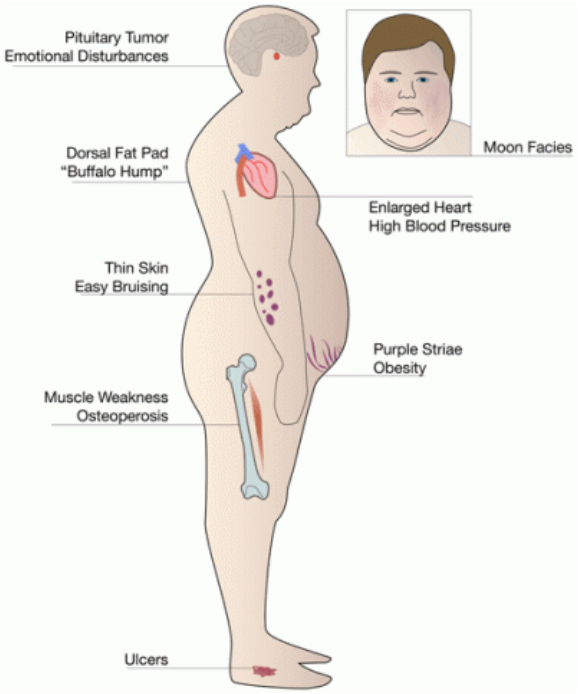


Figure 89 features of Cushing's syndrome

INVESTIGATIONS

In Cushing's syndrome plasma cortisol levels are high, diurnal variation is lost and secretion is not suppressed by low-dose dexamethasone suppression test (normally it is suppressed).

The high-dose dexamethasone suppression test is used differentiate among the various types of Cushing's syndrome. A high dose dexamethasone exerts negative feedback on pituitary ACTH producing cells but not on ectopic ACTH producing cells or adrenal adenoma (or carcinoma). Thus, the urine or serum cortisol is expected to be lower after high dose dexamethasone in Cushing's disease, while unchanged in adrenal adenoma and ectopic ACTH secretion.

Another point of differentiation is the ACTH level. In Cushing's disease due to a pituitary adenoma, plasma ACTH levels are inappropriately high. In ectopic ACTH syndrome, the ACTH levels are often exceedingly high. Patients with an adrenal adenoma have low ACTH levels.

Imaging is important for localizing and measuring the extent of the tumours in the adrenals. CT scan and radioisotope scanning are the commonly used methods.

MANAGEMENT

Adrenal adenomas are rarely bilateral and unilateral adrenalectomy is indicated. Cortisone replacement is needed until the other gland regains full function (it has been suppressed by the other gland).

Adrenal carcinomas should be completely removed whenever possible. Surgery can be used in conjunction with chemotherapy (debulking operation). Adrenal hyperfunction due to a pituitary adenoma or ectopic ACTH secretion is treated accordingly (see later).

PHYSIOLOGY OF THE ADRENAL MEDULLA

Cells of the adrenal medulla synthesize mainly adrenaline(epinephrine) but also noradrenaline (norepinephrine) and dopamine. These catecholamines act as hormones as they are secreted directly into the circulation. Their effects, which are mediated through α and ß receptors on target organs, include activation of the cardiovascular system, resulting in an increase in blood pressure and heart rate; vasoconstriction of vessels in the splanchnic system and vasodilatation of vessels in the muscles; bronchodilatation; and increased glycogenolysis in liver and muscles.

PHEOCHROMOCYTOMA

Pheochromocytoma is a tumour of the adrenal medulla, which is derived from chromaffin cells and which produces catecholamines, the overall prevalence of pheochromocytomas in autopsy specimens is 0.05%. Pheochromocytomas usually subscribe to a convenient "rule of 10s" (see the table below).

The rule of 10s of pheochromocytoma

- **10% arise in association with one of several familial syndromes**
- **10% are extra-adrenal**
- **10% are bilateral**
- **10% are biologically malignant**
- **10% calcify**
- **10% in kids**

ETIOLOGY

About 90% of pheochromocytomas exhibits a benign adenoma like growth, the remaining 10% being malignant. 10% of these tumors show an association with familial syndromes such as those shown in the table below.

Syndromes associated with pheochromocytomas

- **von Hippel—Lindau syndrome**
- **Renal carcinoma**
- **Hemangioblastoma**
- **pancreatic carcinoma**
- **Neurofibromatosis type I (Von Recklin-Hausen's disease)**

CLINICAL FEATURES

Excess catecholamines secretion causes hypertension, the blood pressure rise tends to be paroxysmal and usually presents as headache, palpitation, sweating, extreme anxiety, and chest and abdominal pain.

5 P's: pressure(↑ BP), pain(headache), perspiration, palpitation, pallor

INVESTIGATIONS

The first step in the diagnosis of a pheochromocytoma is the determination of adrenaline, noradrenaline, metanephrine and normetanephrine levels in a 24- hour urine collection. The biochemical diagnosis is followed by the localization of the pheochromocytoma and/or metastases. MRI is preferred because contrast media used for CT scans can provoke paroxysms. Classically, pheochromocytomas show a Swiss cheese configuration (Figure 5).

123I-M1BG (metaiodobe zylguanidine) single-photon emission computerized tomography (SPECT) will identify about 90% of primary tumors and is essential for the detection of multiple extra-adrenal tumors and metastases.

MANAGEMENT

Surgical removal of the tumour is the treatment of choice. Once a pheochromocytoma has been diagnosed, an α-adrenoreceptor blocker (phenoxybenzamine) is used to block catecholamine excess and its consequences during surgery. Additional ß-blockade is required if tachycardia or arrhythmias develop; this should not be introduced until the patient is α- blocked

THE PANCREAS

ANATOMY

The pancreas lies retroperitoneally, behind the lesser sac and stomach at the level of L1-L2. it weighs about 75-100 gm and measures about 15-20 cm in length, the head of pancreas lies within the C-shaped concavity of the duodenum, projecting from the lower part of the head is the uncinate process, which passes posterior to the superior mesenteric vessels, the neck of pancreas is anterior to the superior mesenteric vessels, and, posterior to the neck of the pancreas, the superior mesenteric and the splenic veins join to form the portal vein, the tail of pancreas ends as it passes between layers of the splenorenal ligament.

BLOOD SUPPLY

the celiac artery give rise to the common hepatic Branch which in turn give rise to the gastroduodenal artery; this artery ends as the superior pancreaticoduodenal artery. The inferior pancreaticoduodenal artery arises from the superior mesenteric artery. Both of these arteries anastomose and supply the head and the neck region of the pancreas.

The body and tail are supplied by small branches from the splenic artery. About 10 branches arise from the splenic artery but the major branches are the major, the dorsal and the caudal pancreatic arteries.

VENOUS AND LYMPHATIC DRAINAGE

Veins from the pancreas drain into the superior mesenteric vein and splenic vein. The superior mesenteric vein runs upwards anterior and to the left of the uncinate process, and joins the splenic vein behind the neck of the pancreas to form the portal vein.

Lymphatic vessels and nodes from the head and neck region drain into the celiac (through the pyloric nodes) and superior mesenteric lymph nodes. The body and tail are drained by the pancreatic-lineal lymph nodes. The pancreatic tissue contains about 70 lymph node.

NERVE SUPPLY

The pancreas is innervated by the sympathetic (splanchnic nerves) and parasympathetic nervous (vagus nerve) systems. The parasympathetic system stimulates endocrine and exocrine secretion and the sympathetic system inhibits secretion. The pancreas is rich with sensory nerves.

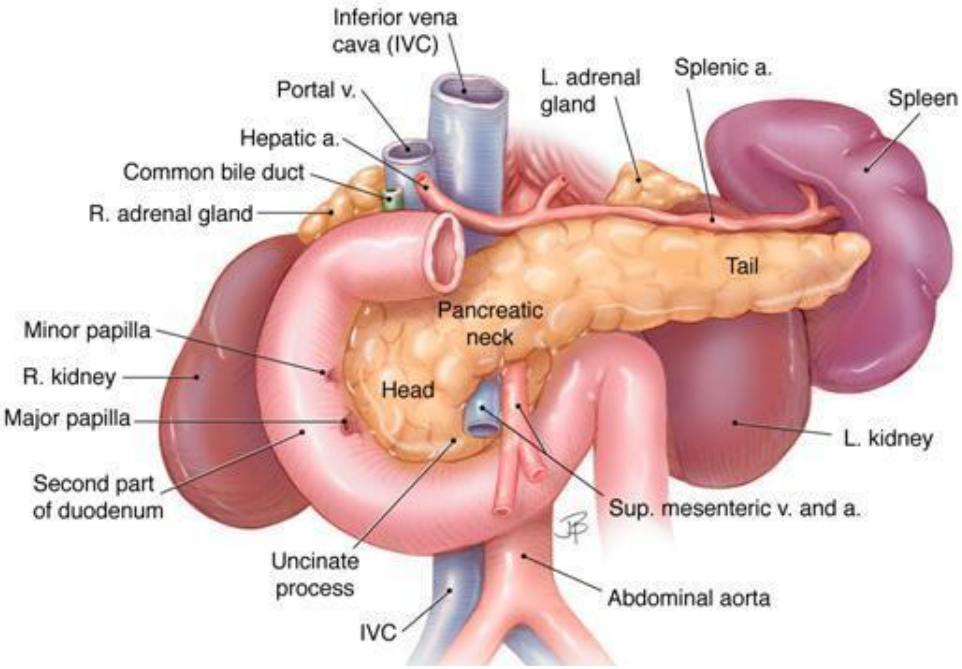


Figure 90 the pancreas anatomy

PANCREATIC DUCT ANATOMY AND EMBRYOLOGY

An understanding of embryology is required to appreciate the common variations in pancreatic duct anatomy. The pancreas develops from separate ventral and dorsal buds of endoderm (figure 2). The duct from the smaller ventral bud, which arises from the hepatic diverticulum, connects directly to the common bile duct. The duct from the larger dorsal bud, which arises from the duodenal bud, drains directly into the duodenum. During gestation, the duodenum rotates clockwise on its long axis (figure 2-A), and the bile duct and ventral pancreas pass round behind it to fuse with the dorsal pancreas. Most of the duct that drains the dorsal pancreas joins the duct draining the ventral pancreas to form the main pancreatic duct (of Wirsung); the rest of the dorsal duct becomes the accessory pancreatic duct (of Santorini) and enters the duodenum proximal to the main duct, (Figure 2-B, C). In approximately 5% of individuals, the ducts draining the dorsal and ventral pancreas fail to fuse, giving rise to pancreas divisum.

The main pancreatic duct mean pressure is between 15-30 mmHg but is always higher than the pressure inside the common bile duct (7-17 mmHg), so that the main pancreatic duct drains into the common bile duct.

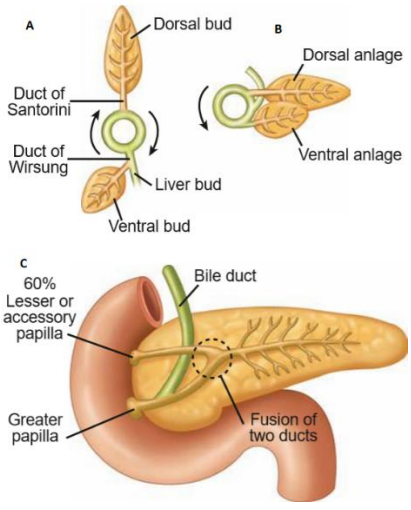


Figure 91 embryology and anatomy of the pancreatic duct

HISTOLOGY

About ;90% of the pancreatic tissue is composed of exocrine acinar tissue, which is organized into lobules. The main pancreatic duct branches into many duct which finally end in acini (figure 3). The main duct is lined by columnar epithelium, which becomes cuboidal terminally. Acinar cells are clumped around a central lumen, which communicates with the duct system.

Clusters of endocrine cells, known as islets of Langerhans, are distributed throughout the pancreas, islet cells consist of differing cell types: 75% are β cells (producing insulin); 20% are α cells (producing glucagon); and the remainder are δ cells (producing somatostatin) and a small number of pancreatic polypeptide cells. Within an islet, the β cells form an inner core surrounded by the other cells. What is interesting is the fact although these islets constitute 2% of the total pancreatic weight, they receive 30% of its blood supply.

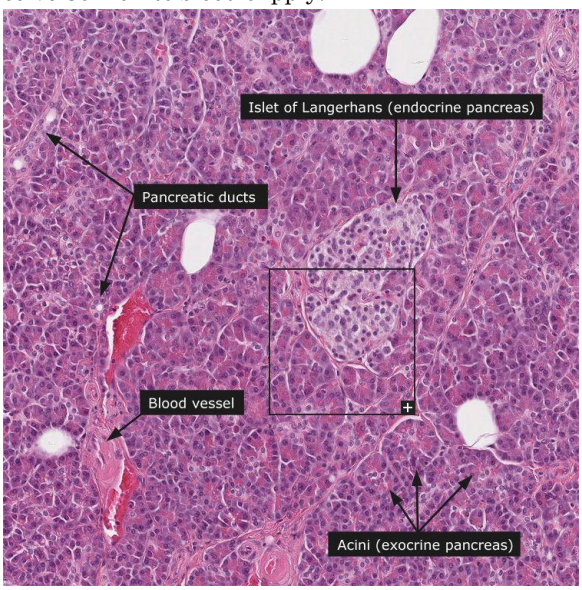


Figure 92 normal histology of the pancreas

PHYSIOLOGY

The pancreas secretes 500-800 ml of alkaline (pH 7.5- 8.8) enzyme-rich juice each day. The enzymes are synthesized by the acinar cells and stored there as zymogen (inactive enzyme) granules. Trypsin is the key proteolytic enzyme; it is released in an inactive form (trypsinogen) and is normally only activated within the duodenum by the brush border enzyme, enterokinase. Once trypsin has been activated, a cascade is established whereby the other proteolytic enzymes become activated in turn.

Lipase (for digestion of fat) and amylase (for digestion of carbohydrates) are secreted as active enzymes.

Pancreatic secretion is stimulated by eating. Hormonal (cholecystokinin, secretin) and neural (vagal) mechanisms are involved.

the endocrine function of the pancreas is important in the regulation of glucose levels in the blood through the hormone's insulin (decrease glucose level), glucagon (increase glucose level). Somatostatin has an inhibitory function on gastrointestinal secretions.

PANCREATITIS

Pancreatitis is inflammation of the gland parenchyma of the pancreas. For clinical purposes, it is useful to divide pancreatitis into acute, which presents as an emergency, and chronic, which is a prolonged and frequently lifelong disorder resulting from the development of fibrosis within the pancreas. In the united kingdom, pancreatitis represents 3% of all abdominal pain referrals.

ACUTE PANCREATITIS

Acute pancreatitis is defined as an acute condition presenting with abdominal pain and is usually associated with raised pancreatic enzyme levels, in the blood or urine as a result of pancreatic inflammation. Acute pancreatitis may be categorized as mild or severe. **Mild (edematous) acute pancreatitis** is characterized by interstitial edema of the gland and minimal organ dysfunction. 80% of patients will have a mild attack of pancreatitis, the mortality from which is around 1%. **Severe (hemorrhagic) acute pancreatitis** is characterized by pancreatic necrosis, a severe systemic inflammatory response and often multi-organ failure.

ETIOLOGY

The two major causes of acute pancreatitis are **biliary calculi** (Gallstone pancreatitis), which occur in 50-70%' of patients, and **alcohol abuse**, which accounts for 25% of cases. Obstruction of the common bile duct with a stone distal to the insertion of the main pancreatic duct promotes the reflux of bile into the pancreatic duct and/or impair the normal flow of pancreatic juice (figure 4). Alcohol is thought to initiate inflammation at the acinar level.

Other less common causes of pancreatitis are shown in the table below.

Causes of acute pancreatitis

- **Biliary calculi**
- **Alcohol**
- **Trauma**
- **Post ERCP**
- **Hyperlipidemia**
- **Hyperparathyroidism**
- **Hypothermia**
- **Vascular**
- **Drugs**
- **Tumors**
- **Immunological**
  - **Systemic lupus erythematosus**
  - **Rheumatoid arthritis**
  - **Polyarteritis nodosa**
  - **Vasculitis**
- **Infection**
  - **bacterial**
  - **Viral (mumps)**
  - **Parasitic**
  - **Fungal**

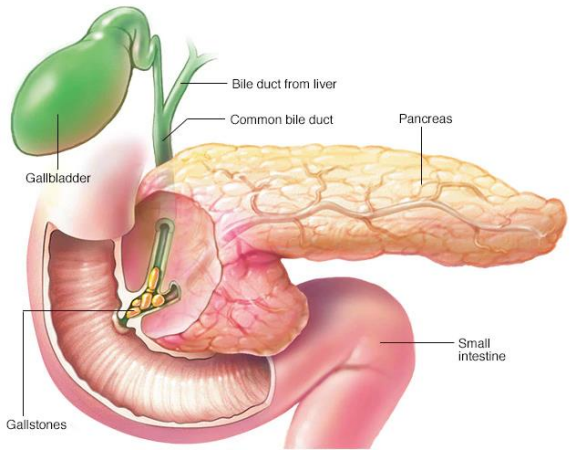


Figure 93 association between biliary stones and acute pancreatitis

PATHOPHYSIOLOGY

two theories have been proposed for explaining the pathogenesis of pancreatitis; (1) premature activation of pancreatic enzymes within the pancreatic duct network (2) and activation of zymogens inside the acinar cells. The end result is injury of the epithelium covering the duct system and the possibility of acinar cell injury. Acinar cell injury may result in the release and premature activation of the zymogens leading to a process of autodigestion. The

continued release of activated proteolytic enzymes is responsible for increased capillary permeability, protein exudation, retroperitoneal edema and peritoneal exudation. With the development of haemorrhagic pancreatitis (which is determined by certain factors, see later), the release of cytokines throughout the blood stream may initiate the systemic inflammatory response syndrome and result in a multi-organ failure (shock/DIC, ARDS,…).

CLINICAL FEATURES

Pain is the cardinal symptom. Pain is usually experienced first in the **epigastric region** but may be localized to either upper quadrant or felt diffusely throughout the abdomen. It characteristically sudden, severe, persistent, refractory that radiates to the back and is relieved in half of the cases by leaning forward. Nausea, vomiting and retching are often marked. The patient may have anorexia.

On examination the patient may show tachycardia, tachypnea, hypotension, and fever. There is usually muscle guarding in the upper abdomen, although marked rigidity is unusual. Abdominal examination may also reveal distension due to ileus. A mass can develop in the epigastrium due to inflammation. Bleeding into the facial planes can produce bluish discoloration of the flanks (Grey Turner's sign) or umbilicus (Cullen's sign) (figure 5). Neither sign is pathognomonic of acute pancreatitis. A pleural effusion is present in 10-20% of patients. Severe pancreatitis can result in massive retroperitoneal hemorrhage with resultant shock and DIC.

INVESTIGATIONS

- **serum pancreatic enzymes**; Serum amylase concentration increases almost immediately with the onset of pancreatitis and peaks within several hours (figure 6). The levels are usually 3 times the normal (the value required for diagnosis; 1000 U/l) at the first 24-48 hours, It remains elevated for 3 to 5 days before returning to normal. Reasons for false positive elevated serum amylase include salivary gland disease (elevated salivary amylase), bowel obstruction, infarction, cholecystitis, and a perforated ulcer. If the serum lipase level can be checked, it provides a slightly more sensitive and specific test than amylase (because it remains elevated for longer time (figure 6)). Other enzymes (e.g. trypsin, elastase, chymotrypsin) are also elevated.

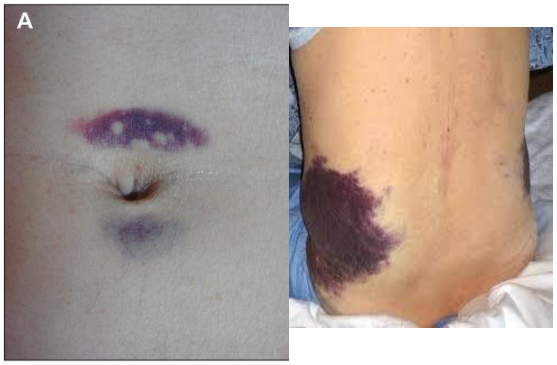


Figure 94 A- Cullen's sign B- Gray's sign

Figure 95 elevation of serum amylase and lipase after the onset of acute pancreatitis

- **Complete blood count**; the hemoglobin might be elevated initially due to loss of fluid (hemoconcentration) but with development of hemorrhagic pancreatitis the hemoglobin levels usually drop. Moderate polymorphonuclear leukocytosis is common.
- **Serum chemistries**; Serum calcium levels may fall along with magnesium in severe disease (due to loss of albumin with exudation and formation of fat soaps). Liver function tests may show hyperbilirubinemia and elevation of liver enzymes, particularly in patients with gallstone pancreatitis.
- hypovolemia may result in acute renal failure and elevation of creatinine level. Methemalbumin is raised during the first 24 hours of the attack and is produced via the oxidation of heme that is initially produced from the breakdown of retroperitoneal hemoglobin into heme by pancreatic enzymes. Heme is oxidized to hematin, which is absorbed into the systemic circulation

to combine with al-bumin and form methe-malbumin.

- Blood gas analysis; initially, tachypnic patients may show respiratory alkalosis but with the development of severe disease acidosis may ensue.
- Chest and abdominal X-ray; A pleural effusion or features of ARDS (i.e. diffuse infiltrate, atlectasis elevated hemidiaphragm) may be seen on chest X-ray. A bowel empty of gas except for a 'sentinel loop' (figure 7-A) of jejunum may reflect local ileus, and in some cases gas is seen midtransverse colon but seems not to extend beyond the splenic flexure -the 'colon cut-off sign (figure 7-B).
- Ultrasound; Ultrasonography may reveal swelling of the pancreas with peripancreatic fluid collections and edema, and may detect gallstones.
- CT scan; CT is not usually performed as part of the initial diagnostic assessment but may also reveal pancreatic and peripancreatic swelling, the development of necrosis (contrast-enhanced) and the presence of gallstones. MRI is less frequently used.
- ERCP; is used as a mean of diagnosis and therapy in gallstone pancreatitis.

ASSESSMENT OF SEVERITY

An early discrimination between mild edematous and severe hemorrhagic forms of the disease is of the utmost importance in order to provide optimal care to the patient. In 1974, Ranson identified a series of prognostic signs (Ranson score) for early identification of patients with severe pancreatitis. Out of these 11 objective parameters, five are measured at the time of admission, whereas the remaining six are measured within 48 hours of admission (see the table on the side). Morbidity and mortality of the disease are directly related to the number of signs present. If the number of positive Ranson signs is less than two, the mortality is generally zero; with three positive signs, mortality is increased to 30%, 40% with 4-5 factors. The mortality rate increases almost 100% when there are more than seven positive Ranson signs.

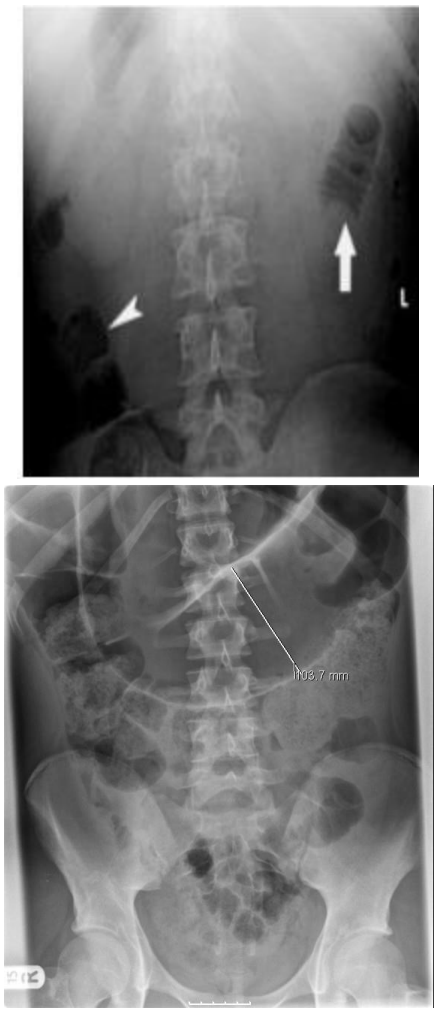


Figure 96 A-a single dilated Jejunal loop (sentinel loop, white arrow) in the upper abdomen B-dilated colon to the mid-transverse colon with no air seen beyond the splenic flexure. This is due to extension of inflammation along mesocolon.

It should be appreciated that the ranson score require 48 hours for full evaluation, by which time clinical assessment is almost as accurate. Progress can be monitored by regular clinical evaluation, and a rising APACHE II score or a rising level of C-reactive protein may help to identify patients in need of urgent investigation and surgical intervention.

Ranson's Prognostic Sign of Pancreatitis	
At admission	After 48 hours
• **Age >55 y**	• Hematocrit fall >10 points
•**WBC>16,000/mm^3**	• BUN elevation >5 mg/dL
• **Blood glucose >200 mg/dL**	• Serum calcium <8 mg/dL
	• Arterial PO_2<60 mm Hg
• **Serum LDH >350 IU/L**	• Base deficit >4 mEq/L
• **Serum AST >250 U/dL**	• Estimated fluid sequestration >6 L

MANAGEMENT

Patients who are considered to have mild acute pancreatitis are treated conservatively (fluids, NPO, antibiotics, analgesia, non-invasive monitoring). Patients whom their prognostic criteria meet a severe attack of pancreatitis should be treated as with a more aggressive approach

- **Fluid therapy**; Aggressive fluid resuscitation is important, guided by frequent measurement of vital signs, urine output and central venous pressure, fluid sequestered is isotonic, thus, ringer lactate (because of acidosis) is appropriate.
- **oxygen**; Supplemental oxygen should be administered and serial arterial blood gas analysis performed.
- **Nasogastric decompression;** A nasogastric tube is not essential but may be of value in patients with vomiting.
- **Nutritional support**; If nutritional support is felt to be necessary, enteral nutrition (e.g. feeding via a nasogastric tube) should be used.
- **Pain relief**; Severe pain requires the administration of opiates.
- **Antibiotics**; (there is some evidence to support the use of prophylactic antibiotics in patients with severe acute pancreatitis for the prevention of local and other septic complications. The duration of antibiotic prophylaxis should not exceed 14 days.
- **Inhibition of pancreatic secretion or enzymes**; the use somatostatin analogue, octreotide along with keeping the patient NPO should suppress pancreatic function.
- **Monitoring**; You should always bear in mind correction of abnormalities. focus must be on urine output, coagulation profile, Hyperkalemia, hypocalcemia, and hypomagnesemia.

If gallstones are the cause of an attack of predicted or proven severe pancreatitis, or if the patient has jaundice, cholangitis or a dilated common bile duct, urgent ERCP should be carried out within 72 hours of the onset of symptoms. When gallstones are found, consideration should be given to cholecystectomy.

MANAGEMENT OF COMPLICATIONS

When there is organ failure, appropriate supportive therapies may include inotropic support for hemodynamic instability, hemofiltration in the event of renal failure, ventilatory support for respiratory failure (due to ARDS) and correction of coagulopathies (including DIC).

Acute fluid collection usually occurs early in the course of acute pancreatitis and is located in or near the pancreas. The fluid is sterile, and most such collections resolve. No intervention is necessary unless a large collection causes symptoms or pressure effects, in which case it can be percutaneously aspirated under ultrasound or CT guidance. **A pancreatic abscess** is a circumscribed intra-abdominal collection of pus, usually in proximity to the pancreas. Treatment consists of adequate external drainage under antibiotic cover.

A pancreatic pseudocyst is a collection of pancreatic secretions and inflammatory exudate enclosed in a wall of fibrous or granulation tissue. It differs from a *true* cyst in that the collection has no

epithelial lining and is surrounded by inflammatory tissue. Small pseudocysts are asymptomatic and resolve spontaneously. Treatment is indicated only if the pseudocyst is enlarging, and aims to avoid infection of the contents, hemorrhage or rupture. It normally consists of drainage of the pseudocyst into a Roux loop of jejunum (pseudocyst-jejunostomy) (figure 8-A), the stomach (pseudocyst-gastrostomy) (figure 8-B) or duodenum (pseudocyst-duodenostomy), whichever appears most appropriate.

The term **pancreatic necrosis** refers to a diffuse or focal area of non-viable parenchyma that is typically associated with peripancreatic fat necrosis. Necrotic areas can be identified by an absence of contrast enhancement on CT. These are sterile to begin with, but can become subsequently infected, probably due to translocation of gut bacteria. Treatment is with **Blunt finger debridement.**

CHRONIC PANCREATITIS

Chronic pancreatitis is a progressive chronic inflammatory condition characterized by fibrosis and the destruction of exocrine and endocrine pancreatic tissue.

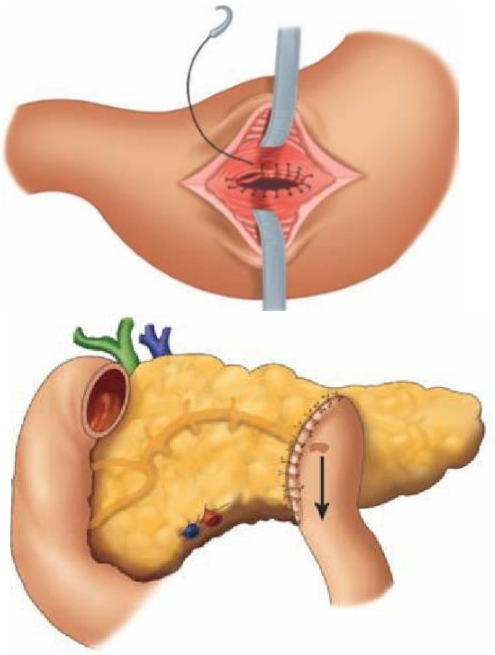


Figure 97 A-pseudocyst-gastrostomy B-pseudocyst-jejunotomy

ETIOLOGY

Chronic pancreatitis is a relatively rare disease but its incidence may be increasing with the growing problem of alcoholism. Alcohol is the most common etiologic factor. Other factors contributing to the development of chronic pancreatitis are shown in the table below.

Causes of chronic pancreatitis
• **Alcohol (60-70%)** • **Hereditary pancreatitis** • **hyperparathyroidism** • **tropical pancreatitis** • **ductal obstruction (strictures, gallstones)** • **idiopathic**

CLINICAL FEATURES

Pain is the outstanding feature in most cases. It is characteristically epigastric with marked radiation through to the back, and is often eased by leaning forward or getting down on all fours, it is aggravated by food especially fatty meals.

Weight loss is usual and reflects a combination of inadequate intake, a poor diet and malabsorption**. Steatorrhea** is common, and occurs due to malabsorption of fat, the bowel motion being pale, bulky, offensive, floating on water, and difficult to flush.

Diabetes mellitus develops in about one-third of patients, but islet function is often preserved for some years following the onset of exocrine insufficiency.

INVESTIGATIONS

Abdominal plain films and CT scans may reveal the speckled calcification typical of chronic pancreatitis (figure 9).

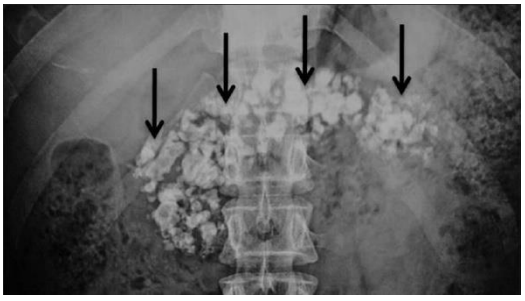


Figure 98 Plain abdominal radiograph; chronic pancreatitis. Multiple opacities can be seen in the region of the head and tail of the pancreas

Ultrasonography and CT can be used to detect pancreatic enlargement, atrophy, dilatation of the pancreatic duct and splenomegaly. ERCP is of great value and must always be performed to reveal the architecture of the pancreatic duct; in mild chronic pancreatitis may show moderate dilatation while severe chronic pancreatitis multiple dilatation with intervening stenosis and such appearance is referred to as 'chain of lakes' (figure 10).

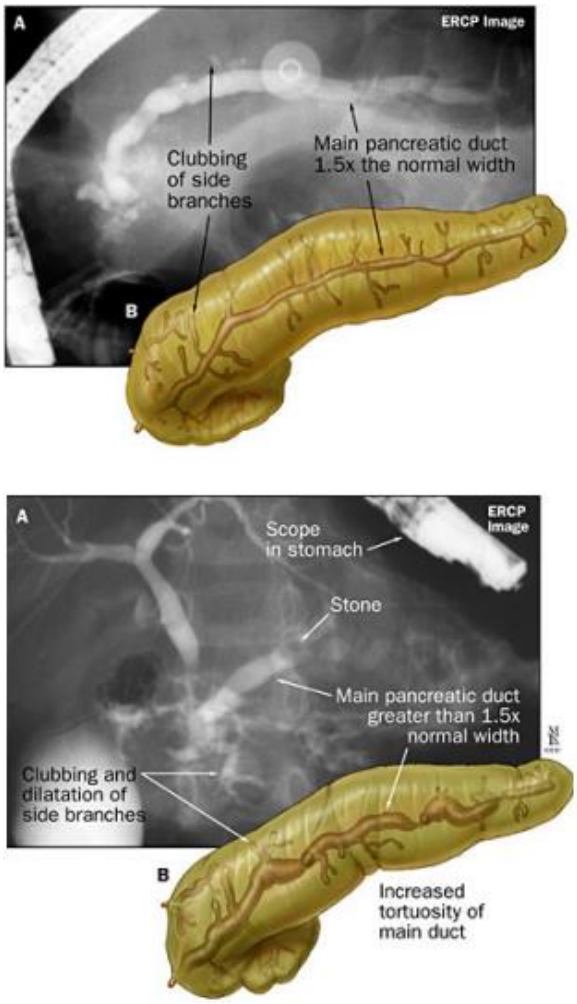


Figure 99 A-ERCP demonstrating moderate chronic pancreatitis. B- ERCP demonstrating severe chronic pancreatitis.

Exocrine function can be measured in a multitude of ways, but insufficiency may not be detectable until 90% of the pancreatic parenchyma is destroyed. Furthermore, function tests do not differentiate between chronic pancreatitis and pancreatic cancer. If necessary, fecal fat excretion can be measured over 3-5 days while the patient's fat intake is controlled at 100 g/day (normal individuals excrete less than 5 g/day), or fat absorption can be measured by isotopic labeling of dietary fat. More often, a trial of oral pancreatic supplements is attempted. Measurement of enzymes and bicarbonate level in the stool is also of value in determining pancreatic function.

MANAGEMENT

Management plan should focus on encouraging abstinence from alcohol as the diseases is progressive with the use of alcohol. Replacement of pancreatic enzymes is of vital importance for treating malabsorption and steatorrhea. Analgesia of high importance to the patient.

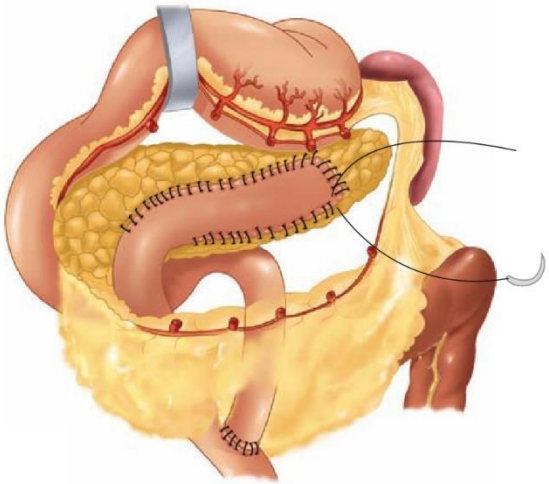


Figure 100 longitudinal pancreatojejunostomy

Surgery is indicated when the pain is intractable, when there is complications or when cancer cannot be excluded, the objective is to relieve pain or compression, while at the same time conserving as much pancreatic tissue and function as possible. This is achieved by anastomosing the dilated pancreatic ducts with jejunum (longitudinal pancreatojejunostomy figure 11). If drainage is not feasible, part or all of the pancreas will have to be respected by distal pancreatectomy if the disease is confined to the distal part of the gland, and pancreaticoduodenectomy if affecting the head of the gland.

PANCREATIC NEOPLASMS

95% of pancreatic neoplasm are malignant pancreatic cancers. The remaining are endocrine tumors.

PANCREATIC CANCER

Pancreatic cancer is by no means a rare cancer and is the sixth leading cause of cancer death. It has a high incidence in western countries and Japan with the highest being in the united states. It affects people in their seventh decade with a slight male preponderance.

PATHOLOGY

More than 85% of pancreatic cancers are ductal adenocarcinomas. Acinar adenocarcinoma constitutes 5% of pancreatic cancers. These tumors infiltrate locally and metastasis to distant organs is common. Ductal adenocarcinomas arise most commonly in the head of the gland . The cancer spreads locally and disseminates to nerve bundles, to local lymphatics, and to lymph nodes around the gland.

ETIOLOGY

The cause of pancreatic cancer is unknown. Factors thought to increase the risk of pancreatic cancer include tobacco smoking and a positive family history. Other possible predisposing factors are shown in the table below.

Predisposing factors for pancreatic cancer

- Cigarette smoking (high association)
- High fat diet
- Alcohol consumption
- Coffee drinking
- Following gastric resection
- post cholecystectomy
- Positive family history (7%)
- Abnormal glucose tolerance (80%)

CLINICAL FEATURES

Obstructive jaundice, that is painless and progressive is seen in patients with tumors of the pancreatic head (figure 12). Jaundice may occur after metastasis to the liver in patients with tail or body tumors. Weight loss is invariable and may be the first symptom. Weight loss might be caused by a combination of anorexia and malabsorption. Pain is present in about 70% of patients at the time of diagnosis and most have ill- defined upper abdominal pain or discomfort. Other less specific symptoms include vomiting, diarrhea, fever (due to necrosis of some parts of the tumor which might release interlukin-1), and hematemesis or melena secondary to duodenal invasion.

On examination, there may be evidence of jaundice, weight loss, a palpable liver and a palpable gall bladder. Courvoisier first drew attention to the association of an enlarged gall bladder and a pancreatic tumor in 1890, when he noted that, when the common duct is obstructed by a stone, distension of the gall bladder (which is likely to be chronically inflamed) is rare; when the duct is obstructed in some other way, such as a neoplasm, distension of the normal gall bladder is common. This had led to what is now referred to as Courvoisier's law which states that any obstructive jaundice with a palpable gallbladder is cancer of the head of the pancreas until proven otherwise.

Other signs that can be noted on the examination in¬clude a palpable abdominal mass with or without ten¬derness, palpable supraclavicular nodes (troisier's sign) and ascites (secondary to portal vein invasion).

INVESTIGATIONS

Patients over the age of 60 years who present with jaundice and weight loss are considered as a case of pancreatic cancer until proven otherwise and thus

should always be investigated further. The following investigations are usually done;

- Tumor markers; CA19-9 is a mucin-associated carbohydrate antigen that can be detected in the serum of patients with pancreatic cancer. Serum levels are elevated in about 75% of patients with pancreatic cancer, however it is not specific. Other tumor markers include the carbohydrate antigen CA50. K-ras oncogene is activated in this tumour.
- Ultrasonography will detect dilatation of the biliary tree, will exclude gallstones, and may show the mass lesion in the pancreas or reveal liver metastases.
- CT scan; spiral (helical) CT with thin-cut examination of the pancreas is used for the staging of pancreatic malignancy (see below).
- Cholangiography; If biliary obstruction is present, cholangiography is used to define the site and nature of the obstruction; MRCP is preferred to ERCP as it is less invasive and displays both pancreatic and biliary duct systems, so determining the need for therapeutic intervention such as stent insertion.
- Cytology; this is important for pathological diagnosis; Pancreatic tissue can be safely obtained by percutaneous fine-needle aspiration under ultrasound or CT scan guidance.
- MRI angiography; this is used to confirm vascular invasion during staging.

STAGING

Staging is achieved by spiral CT scan, MRI angiography and other metastatic workup. The staging of pancreatic cancer is shown in the table above.

Staging of pancreatic tumor using the TNM method	
T1	Limited to pancreas, <2 cm
T2	Limited to pancreas, >2 cm
T3	Extension into duodenum or bile duct
T4	Extension into portal vein, superior mesenteric vein, superior mesenteric artery, stomach, spleen, colon
N0	No nodal metastases
N1	Regional nodal metastases
M0	No distant metastases
Ml	Distant metastases (liver, lung)
Stage TNM	
I	$T_{1,2}N_0M_0$
II	$T_3N_oM_o$
III	$T_{1\text{-}3}N_1M_0$
IVA	$T_4N_{0,1}M_0$
IVB	$T_{1\text{-}4}N_{0,1}M_1$

MANAGEMENT

The treatment of pancreatic cancer is surgical with the combination of chemo- or radiotherapy. Surgery is either curative or palliative. Curative surgery is attempted for respectable tumors. The standard operation of radical pancreaticoduodenectomy (Whipple's procedure) entails block resection of the head of the pancreas, the distal half of the stomach, the duodenum, gallbladder and common bile duct (figure 12).

The relief of jaundice and pruritus (by cholecystic-jejunostomy or choledochojejunostomy), pain (celiac block) and vomiting due to duodenal obstruction (by

gastrojejunostomy) is the objective of palliative treatment.

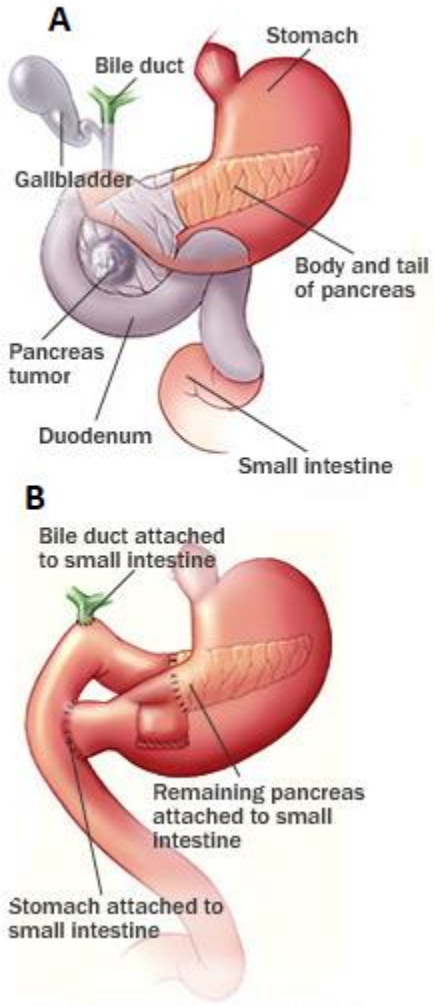


Figure 101 A- The resected area. B-The anastomoses performed. (The gallbladder Is usually removed.)

OTHER PANCREATIC TUMORS

Cystic tumors of the pancreas may be serous or mucinous. They are more common in females (F:M= 6), tend to be large (8-10 cm) and are more likely to occur in the body and tail. Serous cystadenomas are typically found in older women, and are large aggregations of multiple small cysts, almost like bubblewrap. They are benign. Mucinous tumors, on the other hand, have the potential for malignant transformation (mucinous cystadenocarcinoma). The clinical features and management of these tumors is the same as for pancreatic cancer.

Endocrine tumors of the pancreas give rise to defined syndromes as a result of the over-secretion of peptide products. The most common islet cell tumor is the **insulinoma**. It arises from ß cells and results in the over-secretion of insulin, with episodes of hypoglycemia. insulinomas are usually single, small (< 2 cm in diameter) benign tumors and may affect any part of the pancreas. Less than 10% of insulinomas are malignant, but such tumors are often larger than 2 cm in diameter.

Gastrinomas arise from G cells and give rise to the Zollinger-Ellison syndrome. Tumors of the α cells producing glucagon are known as **glucagonomas,** and excessive secretion of vasoactive intestinal peptide (VIP) from **vipomas** produces pancreatic cholera. **Somatostatinoma** is a tumor of the δ cells that produces somatostatin. It is associated with diabetes mellitus and abnormal glucose tolerance.

THE BREAST

ANATOMY

The mature female breast (the mammary glands) are specialized organs of the integumentary system that are controlled mainly by hormones of the reproductive system and by the placenta. The breast is composed of 15 to 20 lobes (Figure 1), each composed of several lobules. Ducts leaving the lobules converge, giving rise to a single lactiferous duct in each lobe. Near the nipple, that lactiferous duct enlarges, forming an expanded chamber called a lactiferous sinus (ampulla). Fibrous bands of connective tissue travel through the breast (suspensory ligaments of Cooper), play an important role in the change in appearance of the breast that often accompanies the development of inflammatory carcinoma of the breast in which blockage of the local lymphatic ducts causes swelling of the breast. Because the skin remains tethered by the suspensory ligaments of Cooper, it takes on a dimpled appearance reminiscent of the peel of an orange (peau d'orange) insert perpendicularly into the dermis, and provide structural support.

The areola contains involuntary muscle arranged in concentric rings as well as radially in the subcutaneous tissue. The areolar epithelium contains numerous sweat glands and sebaceous glands, the latter of which enlarge during pregnancy and serve to lubricate the nipple during lactation (Montgomery's tubercles). The nipple is covered by thick skin with corrugations. Near its apex lie the orifices of the lactiferous ducts. The nipple contains smooth muscle fibres arranged concentrically and longitudinally; thus, it is an erectile structure, which points outwards.

The female breast is generally described as overlying the second to the sixth ribs and extending from the lateral border of the sternum to the anterior axillary line. The breast lies between the skin and the pector¬al fascia, to which it is loosely attached (through the retromammary space).

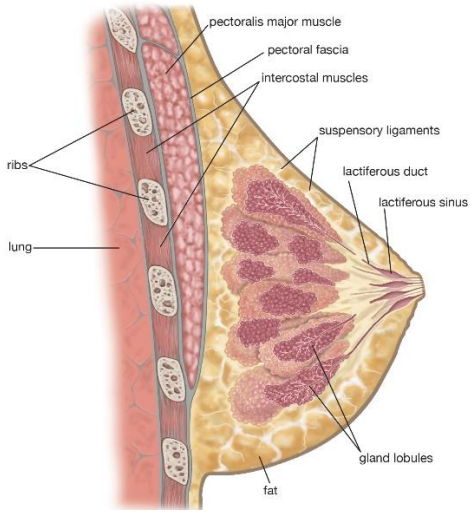


Figure 102 functional anatomy of the breast

BLOOD SUPPLY AND VENOUS DRAINAGE

The breast is related to the thoracic wall and to structures associated with the upper limb; therefore, vascular supply and drainage can occur by multiple routes (Figure 2):

- laterally, vessels from the axillary artery, thoracoacromial and lateral thoracic arteries (lateral mammary branches);
- medially, branches from the internal thoracic artery (medial mammary branches);
- the second to fourth intercostal arteries via branches that perforate the thoracic wall and overlying muscle (lateral mammary branches of the intercostals arteries, figure 2).

Veins draining the breast parallel the arteries and ultimately drain into the axillary, internal thoracic, and intercostal veins.

LYMPHATIC DRAINAGE

The axillary nodes receive approximately 85% of the drainage and are arranged into lateral, along the axillary vein; anterior, along the lateral thoracic vessels; posterior, along the subscapular vessels; central, embedded in fat in the center of the axilla; interpectoral, lying between the pectoralis major and minor muscles; apical, which lie above

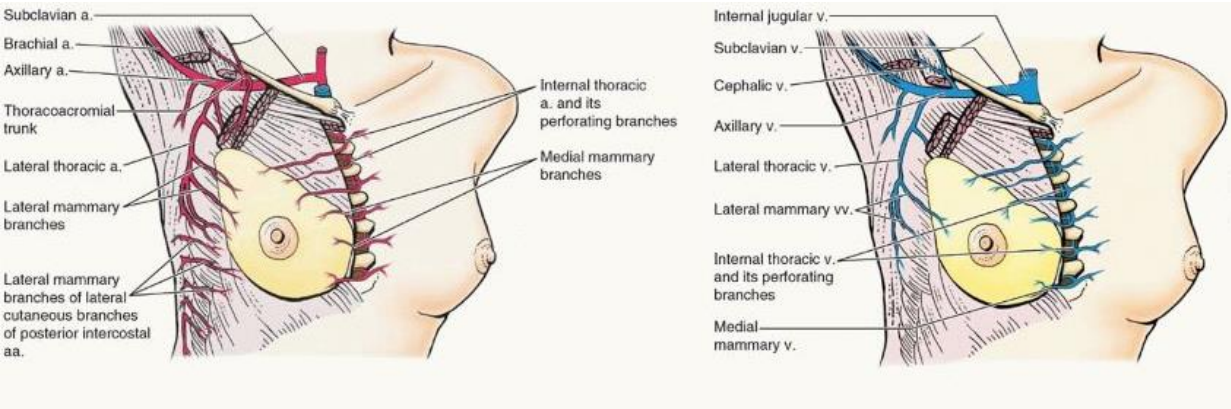


Figure 2 blood supply and venous drainage of the breast

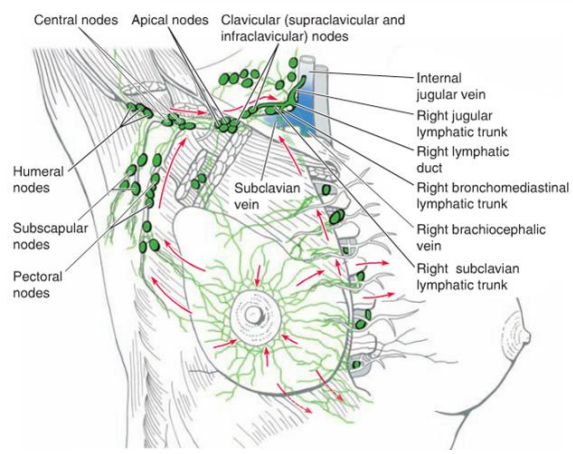


Figure 3 lymph drainage of the breast

the level of the pectoralis minor tendon in continuity with the lateral nodes and which receive the efferent of all the other groups.

The apical nodes are also in continuity with the supraclavicular nodes and drain into the subclavian lymph trunk, which enters the great veins directly or via the thoracic duct or jugular trunk.

The rest of the breast is drained by the internal mammary (parasternal) nodes.

TOPOGRAPHY

The breast has a conical shape with a circular base. Topographically, the breast is divided into four quadrants by two perpendicular lines that pass through the nipple (figure 4). The upper outer quadrant of the breast contains a greater volume of tissue than do the other quadrants. The axillary tail of Spence extends laterally across the anterior axillary fold between the pectoral muscles and latissimus dorsi to blend with the axillary fat.

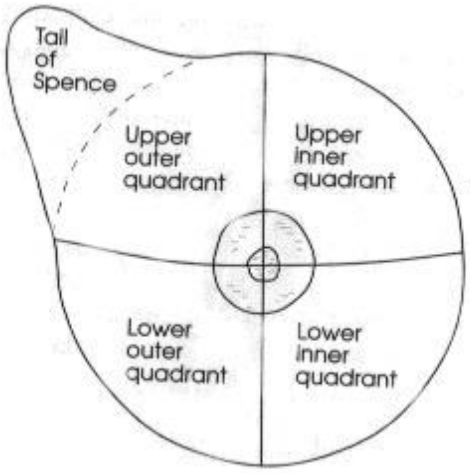


Figure 4 surface anatomy of the breast

HISTOLOGY

The inactive, or resting, mammary gland is dominated by a duct system rather than by active glandular cells. The size of the mammary glands in a nonpregnant woman reflects primarily the amount of adipose tissue present, not the amount of glandular tissue. The secretory apparatus does not complete its development unless pregnancy occurs. The active mammary gland is a tubuloalveolar gland, consisting of multiple glandular tubes that end in secretory alveoli.

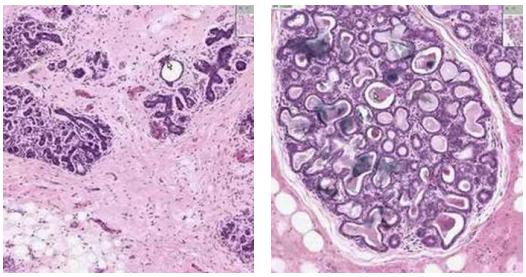


Figure 5 A- inactive breast B- active breast tissue

MASTITIS

Mastitis is inflammation of the breast. The pathology is usually a diffuse cellulitis but an abscess can occur as a result of inadequately treated cellulitis.

TYPES OF MASTITIS

- MASTITIS NEONATORUM
- MASTITIS OF PUBERTY
- LACTATING MASTITIS

60% of mastitis occur in lactating women and is referred to as lactating or puerperal mastitis. Staphylococcus aureus and Streptococcus species are the organisms most frequently recovered from nipple discharge of those patients and they gain entrance through fissures in the nipples, which usually develop during the early weeks of nursing. Staphylococcal infections usually induce single or multiple abscesses. Early infection is treated with the appropriate antibiotic. An established abscess should be treated by re-current aspiration, or by incision and drainage

Excluding the postpartum period, infections of the breast are rare. They can occur in association with syphilis, tuberculosis and actinomycosis.

Mastitis neonatorum is the infections of the breast tissue that predominantly occurs up to the age of two months. It is usually local inflammation and unilateral. It is a very uncommon condition resulting when the breast is squeezed to remove the milk (witch's milk) which usually comes out of the breast because of the maternal

Clinically infants have features of inflammation that is heat, pain, redness, and swelling. Occasionally there may be purulent discharge from the nipple, Systemic signs which are rarely seen includes fever, vomiting, refusal of feeds, irritability and lethargy

Treatment includes antibiotics, analgesics. If an abscess occurs, needle aspiration is performed. Surgical drainage is considered when a needle aspiration is unsuccessful because an operation may damage the breast bud and result in reduction of adult breast size

Puberty mastitis (mastitis adolescentium) can occur due to an endocrine dysfunction or a local trauma and affects girls reaching puberty. The condition is benign and self-limiting.

BENIGN BREAST CONDITIONS

- FIBROADENOMA
- FIBROCYSTIC DIS
- DUCTECTASIA
- BENIGN CYSTS
- LIPOMA:VERY RARE

FIBROADENOMA

Fibroadenomas (so-called 'breast mouse' because of its mobility) are the most common breast lumps in young women but can occur at any time during the reproductive years. Although traditionally thought to be benign neoplasms, they are so common that they are probably best considered an aberration of normal development (see later). Support for this hypothesis comes from the fact that they develop from the whole lobule (unlike cancers) and their epithelium is normal

A fibroadenoma is usually felt as a lump in the breast which is smooth to the touch and moves easily under the skin. Fibroadenomas are usually painless, but sometimes they may feel tender or even painful, particularly just before a period.

They may also get bigger during pregnancy, breastfeeding, or while taking hormone replacement therapy.

Giant fibroadenomas (usually > 5 cm) can occur and can be difficult to distinguish from phylloides tumors both clinically and on core biopsy. In contrast to fibroadenomas, phylloides tumors (previously known as cystosarcoma phylloides) can recur locally and should be surgically removed with wide

excision. In exceptional cases phylloides tumors can metastasize and behave in a malignant fashion; an indication of their malignant potential can be obtained from the number of mitoses per high power field seen on microscopic examination.

INVESTIGATION AND MANAGEMENT

Like all breast lumps, the investigation of a fibroadenoma involves triple assessment (see later) and it is perfectly acceptable to leave small fibroadenomas (< 2 cm) in the breast unless the patient requests excision. However, it is worth emphasizing that a discrete breast lump even in a woman in her early twenties should not be considered a fibroadenoma until all relevant investigations are complete. All giant fibroadenomas should be excised.

DUCT ECTASIA

The major subareolar ducts dilate and shorten with age; when symptomatic, this is known as duct ectasia. By the age of 70,40% of women are affected, some of whom present with nipple discharge or retraction. The discharge is usually cheesy and the retraction is classically slit-like (figure 6), which contrasts with breast cancer, in which the whole nipple is pulled in. Surgery is indicated if the discharge is troublesome or if the patient wishes the nipple to be everted.

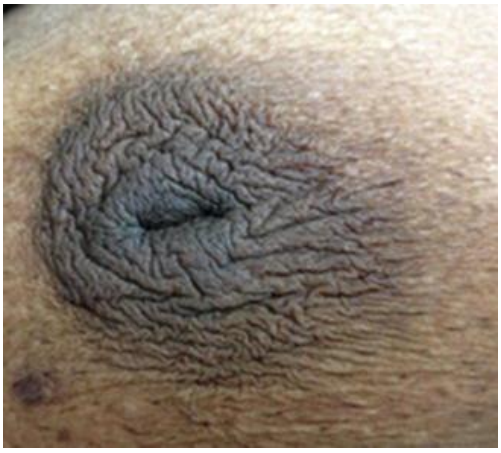


Figure 6 slit-like retraction of the nipple

CYSTOSARCOMA PHYLLOIDES

This disease was thought to be malignant in the pest, and thus, cystosarcoma phylloides (serocystic disease of brodie) is considered a misnomer as the disease is strictly *benign from the onset.* It is now called phylloid tumor. They present as a large, sometimes massive mass. Occasionally, ulceration of overlying skin occurs because of pressure necrosis. Despite their size they remain mobile on the chest wall. Histologically, there is a wide variation in their appearance, with some of low malignant potential resembling a fibroadenoma and others having a higher mitotic index, which are histologically worrying.

Surgery is indicated in large masses, recurrent tumors and worrying histology. Surgery is either wide local excision or mastectomy.

FIBROCYSTIC DISEASES

Fibrocystic disease or known (f is a spectrum of diseases that are characterized by breast lumpiness and is now refered to as Aberrations of Normal Development and Involution (ANDI). Changes vary, but involve cystic changes, fibrosis, Hyperplasia and Papillomatosis of ductal epithelium lining. The cause is yet to be identified but involves affection of the breast tissue from the hormonal changes throughout the menstrual cycle.

CLINICAL FEATURES

Symptoms vary, but in general include lumpiness and tenderness that increases before the onset of menstrual cycle in a middle-aged woman. This is why the tenderness is referred to as cyclical mastalgia. The onset of a non-cyclical mastalgia should call attention for periductal mastitis. The appearance of discrete lumps merits further assessment.

DIAGNOSIS AND MANAGEMENT

The diagnosis can be misleading, but the change of symptoms during the menstrual cycle can help significantly. Discrete lumps require triple assessment, reassurance is usually enough.

BREAST CYSTS

Cysts constitute approximately 15% of all discrete breast masses. Clinically, they are smooth discrete lumps that can be painful and are sometimes visible. Cysts are easily diagnosed by ultrasonography. Cysts are either benign or malignant. The causes of cysts are shown in the table below.

Causes of breast cysts

- **ANDI**
- **Lymphatic cysts**
- **Hydatid cyst**
- **Galactocele**
- **Serocystic disease of brodie**
- **Intracystic papilliferous cancer**
- **Colloid degeneration of cancer**
- **Papillary cystadenoma**

Symptomatic palpable cysts are treated by aspiration and, provided the fluid is not blood-stained, it is discarded. If aspiration results in the disappearance of the mass, then the patient can be reassured. Cysts that contain blood-stained fluid, did not disappear completely on aspiration or have recurred in 6 weeks or show cell on ultrasound, require excision to exclude an associated intracystic cancer.

NIPPLE DISCHARGE

Nipple discharge is a very common complaint in the- breast clinic. The causes of breast discharge are shown in the table below.

Causes of nipple discharge

Nonbloody

- **fibrocystic disease**
- **ductectasia**

Bloody

- **duct papilloma; most common duct ectasia**
- **duct carcinoma; very rare**

It is important to determine if the discharge is from a single duct or multiple ducts and whether it is unilateral or bilateral, spontaneous or not

A unilateral blood-stained nipple discharge is usually due to an intraductal papilloma, although an intraductal carcinoma must of course be ruled out. These patients must have a full triple assessment, with ultrasound of the retroareolar area being useful. The patient should undergo microdochectomy (surgical excision of the duct) performed via a periareolar incision. But first, one must Determine which orifice or segment is bleeding by pressing around the areola. Sending the excised segment or duct for histopathology, allows full assessment of the cause of the discharge.

BREAST CANCER

Breast cancer is the most common female malignancy, accounting for 26% of malignancies in women. It is also the most common cause of cancer death in middle-aged women

ETIOLOGY AND RISK-FACTORS

The cause of breast cancer is uncertain. Several epidemiological and biological risk factors had been identified;

- Age: Breast cancer is a rare disease in women under 35 years old (1/20,000 incidence at 25 years of age) but its incidence at doubles every 5 years until the age of 50, when it affects 1 in 50 women per year. This incidence continues to increase very slightly through to the eighth and ninth decades. The incidence at the age 80 years is about 1 in 10 women
- Family history: Women with close relatives who've been diagnosed with breast cancer have a higher risk of developing the disease.
  If you've had one first-degree female relative (sister, mother, daughter) diagnosed with breast cancer, your risk is doubled. If two first-degree relatives have been diagnosed, your risk is 5 times higher than average.
- Personal History of Breast Cancer: you've been diagnosed with breast cancer, you're 3 to 4 times more likely to develop a new cancer in the other breast or a different part of the same breast. This risk is different from the risk of the original cancer coming back (called risk of recurrence).
- Race and geographical distribution; The incidence and mortality rates for breast cancer are about 5 times higher in North America and northern Europe than they are in many and African countries. For example, the incidence and mortality rates are five times higher in the United States than in Japan.
- Hereditary causes; About 5% to 10% of breast . cancer cases are hereditary, resulting from mutations in the BRCA1 (on chromosome 17q21) or BRCA2 gene (on chromosome 13q12.3). These genetic mutations also increase the risk for

ovarian cancer, both of these genes are thought to function in DNA repair. They act as tumor suppressor genes, since cancer arises when both alleles are inactive or defective-one caused by a germ-line mutation and the second by a subsequent somatic mutation. Women with a mutated BRCA1 orBRCA2 gene have up to a 50% to 85% risk for developing breast cancer by 65 years of age. Families who carry a BRCA1 or BRCA2 abnormality usually have a history of at least four affected relatives with the disease, breast cancer at a young age (under 40), bilateral breast cancer, breast and ovarian cancer same individual (BRCA1) or male breast cancer (BRCA2). Other genes implicated in the etiology of breast cancer include the ataxia telangiectasia and hereditary non-polyposis coli genes. Mutations of the well-known tumor suppressor genes p53 may also be present.

- Estrogen exposure; Patients who take the oral contraceptive pill have an increased relative risk of breast cancer while they are on the pill 1.24 times that of the general population. This rapidly falls to normal after stopping the pill. Hormone replacement therapy (HRT) increases breast cancer risk. The risk is significant after 5 years of use. Combined estrogen and progestogen HRT is associated with a greater risk than preparations containing estrogen alone.
- Life style factors; there is a close correlation between the incidence of breast cancer and dietary fat intake. This is also seen with obesity, lack of exercise and regular ingestion of high amounts of alcohol.
- Breastfeeding History: Breastfeeding can lower breast cancer risk, especially if a woman breastfeeds for longer than 1 year.
- Menstrual History: Women who started menstruating (having periods) younger than age 12 have a higher risk of breast cancer later in life. The same is true for women who go through menopause when they're older than 55.
- Radiation; Ionizing radiation to the chest increases the risk of breast cancer. The magnitude of the

risk depends on the radiation dose, the time since exposure, and age.

PATHOLOGY

Breast cancers are classified into those that have not penetrated the limiting basement membrane (non-invasive) and those that have (invasive) (see the table below).

Classification of breast cancer
Non-invasive   • **Ductal carcinoma in situ (DCIS; intraductal carcinoma)**   • **Lobular carcinoma in situ (LOS)**   • ***Invasive (infiltrating)***   • **Invasive ductal carcinoma ("not otherwise specified")**   **Invasive**   • **lobular carcinoma**   • **Medullary carcinoma**   • **Colloid carcinoma (mucinous carcinoma)**   • **Tubular carcinoma**   • **Other types**

NON-INVASIVE CANCERS (CARCINOMA IN SITU)

Ductal carcinoma *in situ* (DCIS) is a *premalignant* condition of the breast where the cells have taken on a malignant phenotype but invasion through the basement membrane has not yet taken place (proliferation of malignant ductal epithelial cells completely contained within breast ducts (figure7)) Ductal carcinoma in situ is very early cancer that is highly treatable, but if it's left untreated or undetected, it can spread into the surrounding breast tissue.

About 80% non- palpable, detected by screening mammogram, represented by microcalcification. Ductal carcinoma *in situ* carries a high risk for the development of breast cancer in the area of the breast in which it occurs, the risk being proportional to the grade of ductal carcinoma *in situ.* Treatment is wide excision of the area with a rim (1 cm) of normal breast tissue around the abnormality (lumpectomy). Radiotherapy may also be used to decrease local recurrence.

Lobular carcinoma *in situ* is characterized by lobules filled with malignant-type cells (neoplastic cells completely contained within breast lobule). Lobular carcinoma *in situ* increases the risk of development of breast cancer but the risk is

considerably less than that associated with ductal carcinoma *in situ,* and in contrast it less often represents a premalignant lesion. Lobular carcinoma does not present as a palpable mass, and has no mammographic findings, but rather is an incidental finding on breast biopsy for another indication. Patients with this condition are usually managed by close monitoring rather than surgery.

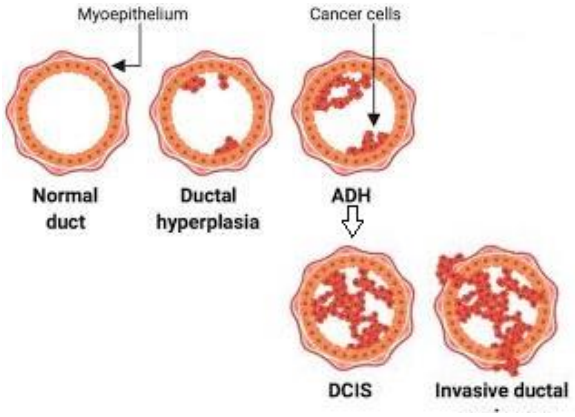


Figure 7 progression of ductal cancer, example of non-invasive breast cancer and pathological variants

INVASIVE CANCERS

The majority (85%) of breast cancer is invasive ductal carcinoma. Carcinomas of "no special type" or "not otherwise specified" are synonyms for ductal carcinomas. Most ductal carcinomas produce a desmoplastic response, which replaces normal breast fat (resulting in a mammographic density) and forms a hard, palpable mass. Ductal carcinoma can be graded from 1 to 3 (1, well differentiated, low grade; 3, poorly differentiated, high grade). This grading system is based on tubule formation, mitotic index and differentiation/nuclear pleomorphism and correlates with tumor outcome. About two-thirds express estrogen or progestagen receptors.

Lobular carcinoma accounts for about 10% of breast cancers. Because does not form micro calcifications, harder to detect mammographically, this makes LCIS virtually always an incidental finding. The disease could be multifocal and it is bilateral in 20% of cases. In unilateral cases always watch the other breast, almost all of these carcinomas express hormone receptors.

A small percentage of breast cancers belong to special types, including medullary (lymphocytic infiltration, better prognosis), colloid or mucinous, and tubular. These are usually towards the well-differentiated end of the spectrum and consequently have a better prognosis.

All breast cancers have a tendency to become adherent to the pectoral muscles or deep fascia of the chest wall, with consequent fixation of the lesion, as well as adherence to the overlying skin, with retraction or dimpling of the skin or nipple, respectively (figure 8). Involvement of the lymphatic pathways may cause localized' lymphedema. In these cases, the skin becomes thickened around exaggerated hair follicles, a change known as peau d'orange (orange peel).

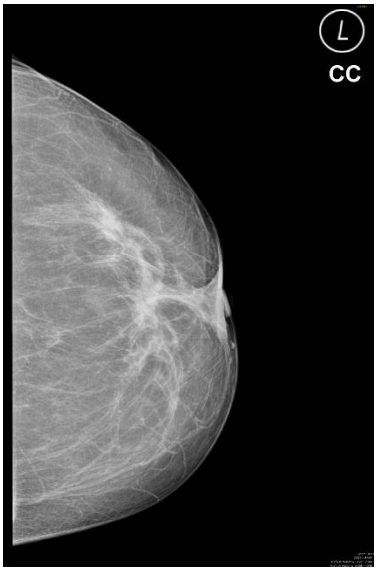


Figure 8 A cut section of an Invasive ductal carcinoma of the breast. The lesion Is retracted, Infiltrating the surrounding breast substance, with other features of breast cancers.

CLINICAL FEATURES

- Breast lump; The presenting complaint in about 70% of patients with breast cancer is a lump (usually painless) in the breast. Less frequently, an axillary mass or swelling of the arm may be the first symptom. The relative frequency of carcinoma in various anatomic sites in the breast is shown in Figure 9.

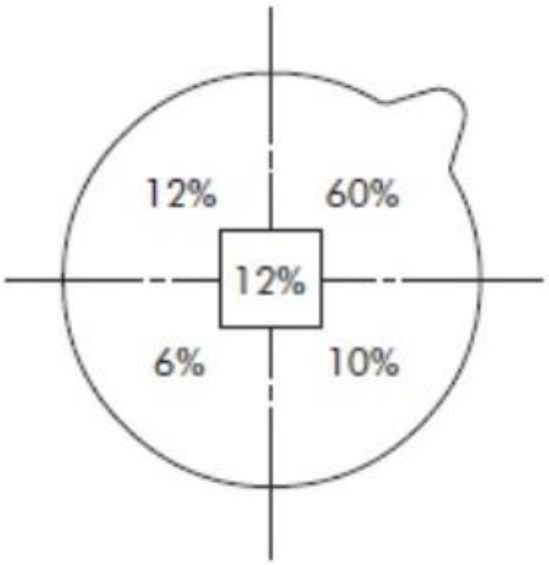


Figure 9 frequencies of breast cancer in each quadrant.

- Nipple changes; these are less frequently seen and include nipple discharge; erosion, retraction, enlargement, or itching of the nipple.
- Skin changes; Skin irritation or changes are occasionally seen and includes puckers, dimples, scaliness, or new creases, the breast can be red and swollen and the skin may become thickened around exaggerated hair follicles, a change known
- Breast pain alone is a rare presenting feature of breast cancer.

In many patients, the cancer is asymptomatic and because of this and because of the high benefit of early detection, screening by mammography and breast self-examination is justified.

PAGET'S DISEASE

Patients with breast cancer occasionally present with a dry scaling or red weeping appearance of the nipple known as Paget's disease and is caused by the extension of malignant cells up to the lactiferous ducts and into the contiguous skin of the nipple (figure 11); thus, it signifies an underlying invasive or non-invasive cancer. it should be differentiated from eczema. Paget's disease (as opposed to eczema) differ in:

- THERE IS AN UNDERLYING BREAST CANCER
- UNILATERAL
- NIPPLE DESTRUCTION
- BOUNDRIES OF THE LESION IS WELL
- DEMARKATED
- DOES NOT RESPOND TO STEROID LOCAL THERAPY

INFLAMMATORY BREAST CANCER

Inflammatory carcinoma (mastitis carcinomatosa) rare and very aggressive disease in which cancer cells block lymph vessels in the skin of the breast. This type of breast cancer is called “inflammatory” because the breast often looks swollen and red, or inflamed, usually without a palpable mass. The underlying carcinoma is generally poorly differentiated and diffusely invades the breast parenchyma (it is considered to be the most malignant of all breast cancers).

The blockage of numerous dermal lymphatic spaces by carcinoma results in the clinical appearance. True inflammation is minimal or absent, thus it is differentiated from mastitis (both occur at young age and has the same appearing breast) by the absence of constitutional symptoms (no fever, no leucocytosis). Clear cut differentiation can only be made by corecut biopsy. ultrasound and mammography are useless because, there is usually no mass.

ASSESSMENT

All patients presenting with a breast lump should have a clinical, radiological and pathological assessment (known as triple assessment) carried out during their first visit to the clinic;

- Clinical assessment; Clinical assessment consists of history and physical examination. Important points in the history include the patient's age, age at menarche (and menopause if applicable), family history of breast cancer (number of affected first- and second-degree relatives, age at disease onset), number of children, age at childbirth and drug history (oral contraceptive pill/hormone replacement therapy). Breast examination is separately discussed in the following lecture.
- Radiological assessment; In women under 35 years of age ultrasound is the preferred

technique, whereas in women over 35 mammography is usually performed (because women under 35; their dense breast tissue gives false positive results). Ultrasound is particularly useful in assessing whether a lump is solid or cystic. With mammography, benign lumps are usually very well defined and may have a surrounding halo, whereas breast cancers are commonly associated with speculation, architectural distortion (spiky dense irregular mass) or malignant microcalcification (usually 5-6 clusters).

- Pathological assessment; Fine-needle aspiration (FNA) allows cells to be taken from the lump. A fine needle attached to a syringe is inserted into the lump and cells are withdrawn by making several passes through the lump with negative pressure (6 mL). A major advantage of this technique is that it allows drainage of a cyst (if fluid is present, then the diagnosis is invariably benign). Once cells are withdrawn from a breast lump they can be sent for cytology assessment. FNA has a 5% false negative mostly due to sampling error. When cytology is inadequate or unhelpful the next step in diagnosis is corecut (truecut) biopsy. This is a minimally invasive procedure where, under local anaesthesia, a sliver of breast tissue is obtained from the breast lump. This carries some advantages over fine-needle aspiration cytology in that a histological assessment of tumor grade (when a lump is malignant) and invasiveness can be made and estrogen receptor status can also be assessed. When a pathology shows atypical hyperplasia or florid hyperplasia, malignancy is indicated. Metaplasia and mild hyperplasia carry no risk.

Other means of diagnosis are MRI and PET. MRI is the most sensitive test for diagnosing breast cancer and can pick up carcinoma in-situ differentiate between local recurrence and fibrosis. It can be used to visualize a palpable mass which is not seen on us or mammogram and is useful in young women as it exposes here to no radiation (a significant risk factor for breast cancer). Advantages in staging are numerous and includes locating the breast cancer with axillary lymph node metastasis. It can also detect multicentric lesion. Disadvantages include, the inability to detect calcifications, being contraindicated in coronary catheterized patients and being extremely expensive. PET is of utmost benefit in delineating multifocal lesions.

SPREAD AND STAGING

Spread occurs through lymphatic and hematogenous channels and by direct extention. spread is most commonly seen to lungs, liver, brain, kidney, adrenals and bone (most prominently to lumbar vertebrae). Metastatic work up is shown in the table below.

Metastatic workup in a patient with breast cancer

- **Blood**
  - **Alkaline phosphatase**
  - **Liver enzymes**
- **Radiological**
  - **Chest X-ray**
  - **Abdominal ultrasound (focusing on the liver)**
- **Nuclear**
  - **Bone scans**

Staging is important for determining the plan of treatment and the prognosis. Stage I and II are categorized as early breast cancer or potentially curable disease while stage III and IV are categorized as advanced cancers (incurable disease).

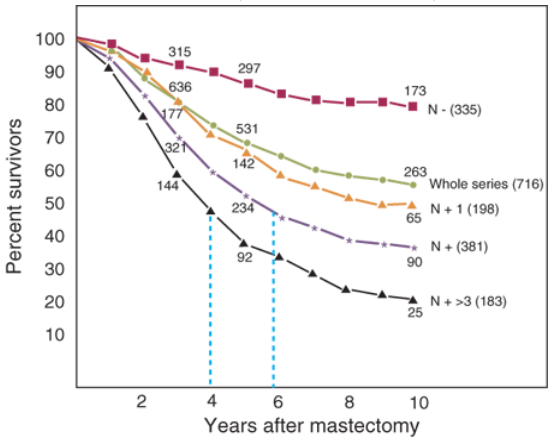


Figure 10 kaplan meier plot of survival by lymph nodes number in patients with breast cancer.

Staging of breast cancer.	
T0	Carcinoma ***in situ or*** microinvasion
T1	Tumour < 2 cm
T2	Tumour 2-5 cm
T3	Tumour >5 cm
T4	Overlying skin or underlying muscle attachment
N0	No axillary nodal involvement
N1	Free axillary nodes (histologically less than three involved nodes)
N2	More than three involved nodes or fixed axillary nodes
N3	Supraclavicular nodes involved
M0	No metastases
M1	Metastatic disease present
Stage	Description
Stage I	T1N0M0
Stage II	
A	T1N1M0
B	T2N0M0/T3N0M0
Stage III	
A	T0N2M0/ T1N2M0/ T2N2M0/ T3N1M0/T3N2M0
B	T4 any N M0/any T N3 M0
Stage IV	**Any T, any N with M1**

The single most important prognostic factor is the number of axillary lymph nodes involved. When there are no lymph nodes involved the 5-year survival rate is about 85%. 5-year survival is 50% when there are 3 lymph nodes involved and 25-40% when the number is more than 3 nodes (figure 10). Other prognostic factors include the stage of the tumor at diagnosis, histological grade, histological type, the presence of lymphatic or vascular invasion, markers of proliferation (HER2; epidermal, growth factor), and hormone receptor content.

MANAGEMENT OF BREAST CANCER

MANAGEMENT OF EARLY BREAST CANCER

Breast-conserving treatment involves excising the tumor with a 1 cm margin of macroscopically normal tissue (wide excision or lumpectomy). Wide excision should be combined with an axillary node staging procedure. This involves either removing the first node or nodes draining the tumor (sentinel node biopsy), sampling (removing four nodes from the lower axilla) or axillary clearance (removing all nodes at levels I, II and III). Wide excision should be followed by radical radiotherapy.

Simple Mastectomy removes all breast tissue with some overlying skin (usually including the nipple), but leaves the chest wall muscles intact. It is preferable in women who have more than one focus of cancer in their breast cancers, those with large cancers in relation to breast size and those with cancers involving the nipple. Radical mastectomy means to remove the breast, do an axillary clearance and excise the pectoralis major and minor muscles. This has been abandoned. The modified radical mastectomy is more commonly performed and implies removing the whole breast and all of the fat, fascia and lymph nodes of the axilla. The axillary vein, the long thoracic nerve and nerves to the latissimus dorsi should be preserved. The intercostals brachial nerves are usually divided in this operation and the patient should be warned about sensation changes postoperatively.

Recurrence after lumpectomy alone is seen more commonly than that after mastectomy. Radiotherapy is given after mastectomy to patients who are at high risk of local recurrence.

OTHER MODALITIES IF TREATMENT

CHEMOTHERAPY

Systemic treatment may be given as adjuvant therapy after surgery and/or radiotherapy, or as primary or neoadjuvant treatment before surgery and/or radiotherapy. Adjuvant treatments consist of chemotherapy or hormonal therapy. Commonly employed champagnes include adriamycin, cyclophosphamide, 5- fluorouracil, epirubicin and methotrexate. Adjuvant hormonal treatments consist of oophorectomy, tamoxifen (estrogen receptor blocker) and the aromatase inhibitor anastrozole (which block the conversion of androgens to estrogen). The benefits of tamoxifen and oophorectomy are greatest in patients with tumours that are rich in estrogen receptors.

BIOLOGICALLY TARGETED THERAPY

10-15% of cancers over-express the oncogene HER2 and these cancers have a worse prognosis than those that are HER2-negative. A humanized monoclonal antibody (trastuzumab or Herceptin) has been shown to reduce the risk of cancer recurrence by up to 50% in women whose cancers overexpress HER2. Other agents currently available include bevacizumab, a vascular growth factor receptor inhibitor, and lapatinib, a combined growth factor receptor inhibitor.

OTHER RELATED CONDITIONS

CANCER-EN-CUIRASSE

The skin of the chest is infiltrated with carcinoma and has been likened to a coat. It may be associated with a grossly swollen arm. This usually occurs in cases with local recurrence after mastectomy and is occasionally seen to follow the distribution of irradiation to the chest wall. The condition may respond to palliative systemic treatment but prognosis in terms of survival is poor.

LYMPHANGIOSARCOMA

Lymphangiosarcoma is a rare complication of lymphoedema with an onset many years after the original treatment. It takes the form of multiple subcutaneous nodules in the upper limb and must be distinguished from recurrent carcinoma of the breast. The prognosis is poor but some cases respond to cytotoxic therapy or irradiation.

MALE BREAST

GYNECOMASTIA

Gynecomastia (the growth of breast tissue in males to any extent in all ages) is entirely benign and usually reversible. Causes include excess alcohol intake and drugs including cannabis, spironolactone and finasteride, cirrhosis, hypogonadism and, rarely, testicular tumours. Rapidly progressive gynecomastia is an indication for an assessment of hormonal profile. Embarrassment or persistent enlargement is an indication for surgery.

MALE BREAST CANCERS

Fewer than 0.5% of all breast cancers occur in men, and breast cancer comprises 0.7% of all male cancers. The peak incidence in males is 5-10 years later than in women. Cancer usually presents with an eccentric breast mass or retraction of the overlying skin; the disease is more likely to be advanced at diagnosis. Mammography and core biopsy or fine-needle aspiration cytology confirms the diagnosis. Treatment for localized breast cancer is by mastectomy and the removal of axillary nodes, usually followed by post-operative radiotherapy to the chest wall. Adjuvant chemotherapy can significantly decrease recurrence.

DYSPHAGIA

THE PHYSIOLOGY OF SWALLOWING

There are three phases involved in swallowing: oral, pharyngeal, and esophageal. The oral phase is voluntary, and the pharyngeal and esophageal phases are controlled by reflexes. The oral phase is initiated when the tongue forces a bolus of food back toward the pharynx, which contains a high density of somatosensory receptors. The purpose of the pharyngeal phase is to propel the food bolus from the mouth through the pharynx to the esophagus in the following steps: (1) The soft palate is pulled upward, creating a narrow passage for food to move into the pharynx so food cannot reflux into the nasopharynx. (2) The epiglottis moves to cover the opening to the larynx, and the larynx moves upward against the epiglottis to prevent food from entering the trachea. (3) The upper esophageal sphincter relaxes, allowing food to pass from the pharynx to the esophagus. (4) A peristaltic wave of contraction is initiated in the pharynx and propels food through the open sphincter. Breathing is inhibited during the pharyngeal phase of swallowing.

The esophageal phase of swallowing is controlled in part by the swallowing reflex and in part by the enteric nervous system. In the esophageal phase, food is propelled through the esophagus to the stomach. Once the bolus has passed through the upper esophageal sphincter in the pharyngeal phase, the swallowing reflex closes the sphincter so food cannot reflux into the pharynx. A primary peristaltic wave, also coordinated by the swallowing reflex, travels down the esophagus, propelling the food along. If the primary peristaltic wave does not clear the esophagus of food, a secondary peristaltic wave is initiated by the continued distention of the esophagus. The secondary wave, which is mediated by the enteric nervous system, begins at the site of distention and travels downward.

DEFINITIONS

Dysphagia is difficulty swallowing. The condition results from impeded transport of liquids, solids, or both from the pharynx to the stomach. A very common condition, dysphagia affects 1.6-15% of the middle- aged and 13-35% of the elderly populations.

Dysphagia is classified as **oropharyngeal** or e**sophageal**, depending on where it occurs.

Oropharyngeal dysphagia (transfer dysphagia): There's problem in initiating the swallowing as there's difficulty in emptying material from the oropharynx into the esophagus. Most often, oropharyngeal dysphagia occurs in patients with neurologic conditions or muscular disorders that affect skeletal muscles. It will be associated with coughing, chocking, nasal regurgitation.

Esophageal dysphagia:The swallowing in initiating but the problem is difficulty passing food down the esophagus. It results from either a motility disorder or a mechanical obstruction, be it intrinsic narrowing or extrinsic compression of the esophagus.

Dysphagia should not be confused with globus sensation *{Globus pharyngeus);* a feeling of having a lump in the throat, which is unrelated to swallowing and occurs without impaired transport.

Odynophagia refers to painful swallowing and usually indicates an inflammatory lesion.

SIGNIFICANCE AND COMPLICATIONS :

Other than the fact that dysphagia sometimes points to a serious underlying pathology, thus requiring assessment, severe long standing dysphagia can lead to tracheal aspiration of ingested material,' oral secretions, or both. Aspiration can cause acute pneumonia; recurrent aspiration may eventually lead to chronic lung disease.
Prolonged dysphagia often leads to inadequate nutrition and weight loss. Also, choking is a serious complication as food gets stuck in the throat , if it completely blocks the airway it can cause death if no early intervention was done.

APPROACH TO THE PATIENT

Dysphagia is a serious symptom and requires assessment.

A-HISTORY

History alone can provide a tentative diagnosis in nearly 80% of cases. Thus, detailed discussion of history as a tool for diagnosis is very important.

Causes of dysphagia

Oropharyngeal dysphagia

Neurologic

- **Stroke**
- **Parkinson's disease**
- **Multiple sclerosis**
- **Some motor neuron disorders (amyotrophic lateral sclerosis, progressive bulbar palsy, pseudobulbar palsy)**
- **Bulbar poliomyelitis**

Muscular

- **Myasthenia gravis**
- **Dermatomyositis**
- **Muscular dystrophy**
- **Cricopharyngeal incoordination**

Esophageal dysphasia

Motility disorder

- **Achalasia**
- **Diffuse esophageal spasm**
- **Systemic sclerosis(scleroderma)**
- **Eosinophilic esophagitis**

Mechanical obstruction

- ***Extramural***
  - **enlarged left atrium**
  - **aortic aneurysm**
  - **aberrant subclavian artery [termed dysphagia lusoria]**
  - **substernal thyroid**
  - **cervical bony exostosis thoracic tumors**
  - **Pressure of enlarged lymph nodes**
  - **secondary to cancer or lymphoma**
  - **retrosternal goitre**
  - **bronchial carcinoma**
- ***Intramural***
  - **Peptic stricture**
  - **Esophageal cancer**
  - **Lower esophageal rings(Schatzki's ring)**
  - **Caustic ingestion**
  - **esophageal or cardia tumors**
  - **pharyngeal pouch**
  - **plummer-vinson syndrome with esophageal web.**
- ***Intraluminal***
  - **Foreign body**

HISTORY OF PRESENT ILLNESS

Certain points in the history are helpful in leading to a diagnosis;

- Age and gender;
- Onset; Sudden onset suggests a foreign body.
- Duration and course of symptoms: is it progressive (2 weeks to month) or is it intermittent (For several years) ; As if it's progressive it suggests causes such malignancy(It progresses faster than the benign causes) , peptic stricture , scleroderma , achalasia but it's intermittent for several years it'll suggest benign causes such as lower esophageal sphincter and diffuse esophageal spasm.
- Site; The actual site of obstruction correlates poorly in general to where the patient feels the discomfort, although some patients who feel the obstruction to be high may have a pharyngeal pouch.
- Severity; Severity can be determined by whether patients have difficulty swallowing solids, liquids, or both. Difficulty in swallowing solids is initially typical of carcinoma, whereas achalasia tends to be associated with dysphagia to liquids at first.
- Associated features; pain with swallowing is termed odynophagia. Pain in conjunction with dysphagia suggests spasm or achalasia (although pain may occur in these conditions without concurrent difficulty in swallowing). Pain on swallowing saliva alone is characteristic of serious mucosal inflammation. This is seen after causitic ingestion. Hiccups point to difficulty in the terminal portion of the esophagus. Heart burn or reflex indicates peptic stricture or scleroderma.

INTERPRETATION OF FINDINGS

As a poorly identified rule, motor disease (e.g. achalasia) is suggested by gradual onset, slow progression, chronic course, equal difficulty with liquids and solids. Mechanical obstruction is characterized by a more rapid onset and progressive course, more difficulty with solids than with liquids.

Intermittent dysphagia that occurs only with solid food is indicative of. a lower esophageal (Schatzki's) ring. Patient with oropharyngeal dysphagia (transfer dysphagia) reports the symptom as beginning immediately on trying to swallow with aspiration and regurgitation of fluid into the nose and are accurately localized to the suprasternal area.

REVIEW OF SYMPTOMS

Review of symptoms should focus on symptoms suggestive of neuromuscular; gastrointestinal, and connective tissue disorders and on the presence of complications (see the table below). These are important for the surgeon as they may rule out the need for further assessment of medical diseases with no surgical importance.

Past medical history should ascertain known diseases that may dysphagia.

PHYSICAL EXAMINATION

Examination focuses on findings suggestive of neuromuscular, Gl, and connective tissue disorders and on the presence of complications. The following points should be covered;

• General examination should evaluate nutritional status (including body weight) especially in long standing dysphagia. Lymph nodes are palpated in the neck and elsewhere to detect enlargement suggestive of neoplasm or infection. Skin is examined for rash and thickening or texture changes (hyperkeratosis indicates cancer), particularly on the fingertips (Raynaud's phenomenon) and for signs of scleroderma (sclerodactyly, telangiectasia, calcinosis).

• A complete neurologic examination is essential, with attention to any resting tremor, rigidity (Parkinson's), and spasticity (stroke). The cranial nerves should be thoroughly examined, especially the vagus and the glossopharyngeal nerves (note the gag reflex may normally be absent), and muscle strength. Patients who describe easy fatigability should be observed performing a repetitive action (e.g. blinking, counting aloud) for a rapid decrement in performance. The patient's gait should be observed, and balance should be tested.

• Muscles are inspected for wasting and fasciculations and are palpated for tenderness (dermatomysitis, myopathy).

• The mouth should be examined carefully for inflammatory lesions, ill-fitting dentures, and pharyngeal masses. The HIV-positive patient with oral candidiasis is at increased risk for esophageal involvement.

History and cause of dysphagia	
Finding	Possible cause
Tremor, ataxia, balance disturbance	Parkinson's disease
Muscle fasciculation,- wasting, weakness	Motor neuron disease, myopathy
aphasia, diplopia, or **dysphonia**	Myasthenia graves
Rapidly progressive, constant dysphagia, no neurologic findings	Esophageal obstruction, probably cancer
Intermittent dysphagia	Lower esophageal ring or diffuse esophageal spasm
Slow progression (months to years) of dysphagia to solids and liquids, sometimes; with nocturnal regurgitation	Achalasia
Dusky, erythematous rash, muscle tenderness	Dermatomyositis
Raynaud's phenomenon, arthralgias, skin tighten- ing/contractures of fingers	Systemic sclerosis
Cough, dyspnea, lung congestions	Pulmonary aspiration
Smooth tongue , anemia, koilonychia	Plummer-vinson syndrome
Palpable lymph nodes **in the Neck and** **supraclavicular fossea and in** **upper abdomen**	Carcinoma of cardia or esophegus

• The neck is evaluated for thyromegaly or other mass. The thyroid is palpated for a goiter that might extrinsically compress the esophagus; especially a substernal one (cannot feel the trachea).

• The abdomen is checked for masses, tenderness, and organomegaly and the stool for occult blood (suggestive of neoplasm and esophagitis).

Regurgitation of a small amount of food on lateral compression of the neck is virtually diagnostic of pharyngeal diverticulum.

TESTING

Many findings suggest specific disorders but are of varying sensitivity and specificity and thus do not rule in or out a given cause; however, they can guide testing.

• Fiberoptic esophagoscopy: enables biopsies to be taken to confirm malignancy, and permits therapeutic dilation or intubation if indicated .

Acute onset of odynophagia suggests severe esophageal inflammation and, as seen after caustic ingestion or due to an infectious etiology. This may also require diagnose with endoscopy.

• Barium Swallow; Barium swallow is the test of choice in the assessment of transfer dysphagia. Its sensitivity is also excellent for determining the location and severity of an obstructing mass lesion or stenosis or dilation such in achalasia , cervical web , but it often lacks precision in identifying the nature of the lesion, particularly in distinguishing cancer from post-inflammatory scarring and stenosis. When an obstructing lesion is suspected, endoscopy with biopsy is required, especially when the history suggests malignancy (e.g. Rapid progression, marked weight loss, solids more problematic than liquids). By providing fluoroscopic evidence of esophageal function, the barium swallow can sometimes help in documenting motor disorders, although test sensitivity is not high. The findings are explained in the 'esophagus' lecture.

• Manometry; Strong clinical suspicion of motor dysfunction and failure of endoscopy and barium swallow to reveal a probable etiology raise the question of proceeding to manometry. The diagnoses of nutcracker esophagus is especially dependent on manometric data (peristaltic waves more than 140 mmHg in amplitude). Continuous manometric monitoring is needed for other motility disorders.

OTHER TESTS

Other tests for specific causes are done as suggested by findings.

TREATMENT

Treatment is directed at the specific cause. If complete obstruction occurs, emergent upper endoscopy is essential. If a stricture, ring, or web is found, careful endoscopic dilation is performed. Pending resolution, patients with oropharyngeal dysphagia may benefit from evaluation by a rehabilitation specialist. Sometimes patients benefit from changing head position while eating, retraining the swallowing muscles, doing exercises that improve the ability to accommodate a food bolus in the oral cavity, or doing strength and coordination exercises for the tongue. Patients with severe dysphagia and recurrent aspiration may require a gastrostomy tube.

THE ESOPHAGUS

ANATOMY

The esophagus (figurel) extends from the cricoid cartilage (at the level of C6 vertebra) to the gastric cardia and is 25 cm long. It has cervical (5 cm), thoracic (18 cm) and abdominal (2 cm) portions. The esophagus has an upper esophageal sphincter (UES), the cricopharyngeus, and a lower esophageal sphincter (LES) that cannot be defined anatomically but is a 3-5 cm high-pressure area located in the region of the esophageal hiatus of the diaphragm.

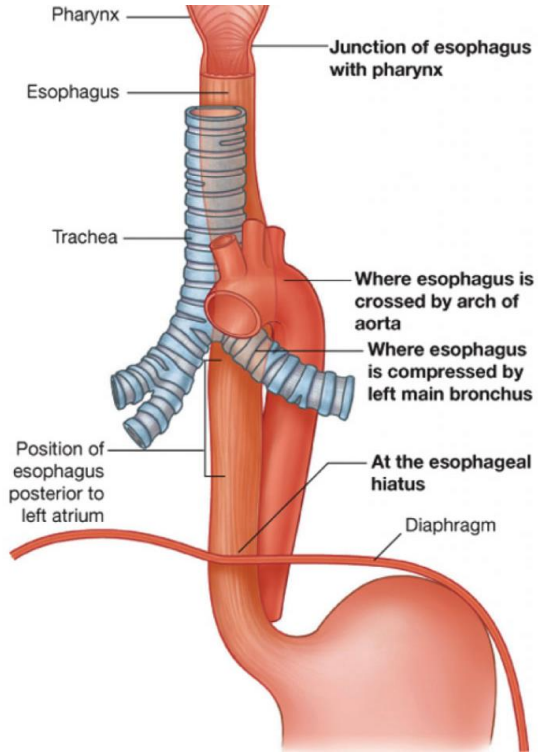


Figure 103 Anatomy of the esophagus

Three normal areas of esophageal narrowing are evident on the barium esophagogram (figure 2). The uppermost narrowing is located at the entrance into the esophagus and is caused by the cricopharyngeal muscle. The middle narrowing is due to an indentation of the anterior and left lateral esophageal wall caused by the crossing of the left main stem bronchus and the aortic arch. The lowermost narrowing is at the hiatus of the diaphragm and is caused by the gastroesophageal sphincter mechanism.

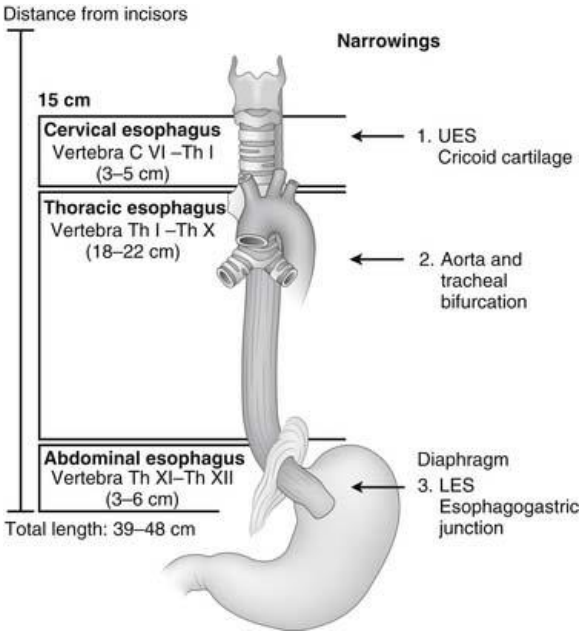


Figure 104 anatomical narrowing of the esophagus

PHYSIOLOGY

The main function of the esophegus is to transfer food from the mouth to the stomach in a coordinated fashion and prevents stomach acid and content reflux upward. The initial movement is voluntary. The esophagus usually prevents reflux by the following mechanisms (the antireflux barrier (ARB), figure 3);

• the lower esophageal sphincter (LES); a physiological high-pressure zone (not a true sphincter) in the lower end of the esophagus.

• the gastroesophageal valve (GEV); the angle at which the esophagus joins the stomach between the left border of the esophagus and the fundus (angle of His) is called the gastroesophageal valve and represents the most important entity in the function of the antireflux barrier.

• Other mechanisms include the phrenoesophageal ligament, the diaphragmatic sling (crura), the intraabdominal effect and the mucosal rosette at the cardia.

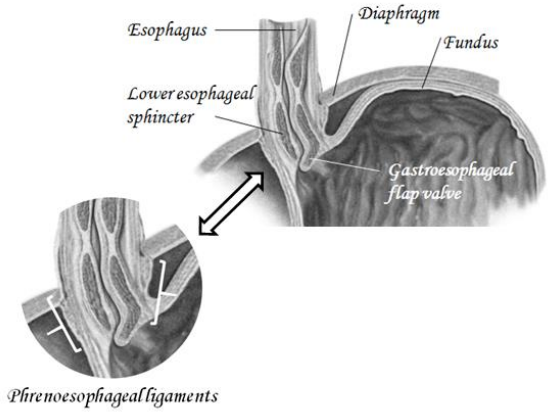


Figure 105 the ant reflex barrier

GASTSO-ESOPHAGEAL REFLUX DIS-EASAS

Gastro-esophageal reflux disease (GERD) is a term used to collectively describe the problems and symptoms that occur when acid from the stomach washes up into the esophagus. it is a common disease that accounts for approximately two thirds of esophageal pathology.

ETIOLOGY

Normal competence of the gastro-esophageal junction os maintained by the LOS . In the early stages of GERD , most pathological reflux results for increased number of TLOSRs(transient LOS relaxations).

The antireflux barrier can also be affected by the intraabdominal length of the esophagus. The absence of an intra-abdominal length of the esophagus results in a **sliding hiatus hernia.** The normal condensation of peritoneal fascia over the lower esophagus (the phrenoesophageal ligament) is weak, and the crural opening widens allowing the upper stomach to slide up through the hiatus. The loss of normal anatomical configuration exacerbates reflux, although sliding hernia alone should not be viewed as the cause of reflux sliding hiatus hernia is associated with GERD and may make it worse but, as long as the LOS remains competent, pathological GERD does not occur. Other factors which might exacerbate or produce reflux include cases where there is delayed gastric emptying or increased intraabdominal pressure.

PATHOLOGY

Esophagitis resulting from reflux is referred to as reflux esophagitis. The pathological events involve a break in the mucosa due to destruction by the stomach contents and initiation of inflammation. Mild esophagitis may appear macroscopically as simple hyperemia while more severe cases may show epithelial erosions (see the table below).

Pathological grading of esophagitis
Grade I; Hyperemic mucosa
Grade II; superficial ulcerations
Grade III; Dilatable stricture
Grade IV; fixed not dilatable stricture

The Los Angeles classification is the most recently developed system for the grading of reflux esophagitis. typical symptoms of GERD are caused by esophagitis.

Los Angeles classification	
NERD (60%)	Visually normal mucosa, diffuse hyperemia
Grade A	One or more mucosal breaks < 5 mm in maximal length
Grade B	One or more mucosal breaks > 5mm, but without continuity across mucosal folds
Grade C	Mucosal breaks continuous between > 2 mucosal folds, but involving less than 75% of the esophageal circumference
Grade D	Mucosal breaks involving more than 75% of esophageal circumference

CLINICAL FEATURES

The **typical** symptoms of GERD are represented by The classical triad retrosternal burning pain (heartburn), epigastric pain (sometimes radiating through to the back) and regurgitation. Regurgitation of acid or food may occur in more severe cases. Symptoms are often provoked by food, particularly those that delay gastric emptying (e.g. fats, spicy foods). Thus patients may develop fatty dyspepsia.

Atypical symptoms might occur. Dysphagia is usually a sign that a stricture has occurred (especially if it occurs to solid foods), but may be caused by an associated motility disorder, patient may describe a sensation of choking. A proportion of patients have odynophagia with hot beverages, citrus drinks or alcohol. Less commonly patients may present with hematemesis.

Some patients present with extra-esophageal atypical symptoms such as angina-like chest pain, cough, asthma and hoarseness of voice.

DIAGNOSIS

The diagnosis of GERD usually requires the complementary use of careful history, upper gastrointestinal contrast studies, endoscopy, pH monitoring and manometric studies.

• History; The history should focus on the symptoms (typical and atypical) and their analysis (duration, timing, exacerbation. etc).

• Bariums studies; A barium swallow and meal might demonstrate a hiatus hernia, the presence

of severe ulceration, benign strictures and reflux of contrast from the stomach into the esophagus in the head-down position.

• Endoscopy; endoscopy will confirm reflux if esophagitis is seen and allow taking biopsies to detect complications (Barrett's esophagus).

• pH monitoring; Ambulatory 24-hour pH monitoring is the gold standard in establishing the diagnosis of acid reflux. A pH probe is positioned 5 cm above LES, measuring the total number of reflux episodes (reading < 4 PH), the longest episode of reflux (> 5 minutes) and thus, the extent of reflux (beyond the scope of the lecture).

• Manometry; esophageal manometry is a study performed to evaluate the pressure of the esophagus in various stages along its length. Recordings are usually made by passing a multilumen catheter with three to eight recording orifices at different levels down the esophagus and into the stomach (figure 4). While the main purpose of the test is objectively to quantify the extent of reflux disease, it is also used to rule out a diagnosis of achalasia.

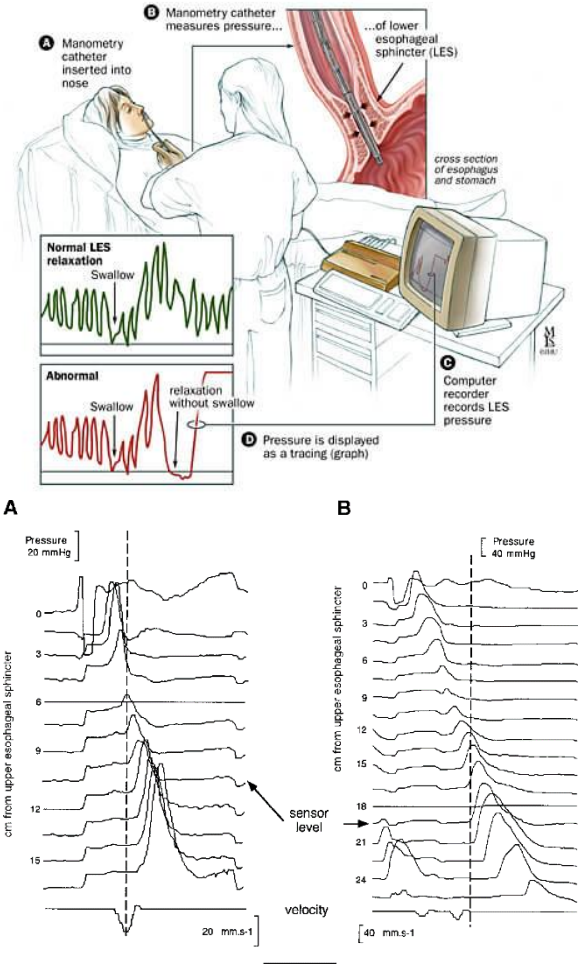


Figure 106 Recording of the luminal pressure within the esophagus.

MANAGEMENT

MANAGEMENT OF UNCOMPLICATED DISEASE

Treatment is aimed at reducing the reflux and prevent damage to the lining of the esophagus. Consultation is important and should include an advice against weight loss, smoking, excessive consumption of alcohol, tea or coffee. The doctor should also advice the patient to avoid anything that increases

the intra-abdominal pressure during sleep such as wearing tight abdominal clothing's and heavy weight lifting 2 hours after a meal. Advice concerning food should include the avoidance of large meals late at night, to avoid meals within 2 hours before sleeping, to include meals that contain less fat. Tilting the bed has been shown to have an effect that is similar to taking an H_2-receptor antagonist.

Most sufferers from GERD do not consult a doctor and do not need to do so. They self-medicate with over- the-counter medicines such as simple antacids, antacid alginate preparations and H_2-receptor antagonists and metoclopramide (a dopamine antagonist that stimulates gastric emptying and increases small bowel transit as well as enhancing contraction of the esophageal sphincter).

The primary goal of surgical management is to establish the competency of the cardia. The most commonly performed anti-reflux surgery is the Nissen's fundoplication (figure 5). The procedure can be performed as open or laparoscopically. Indications for surgery are shown in the table below.

Indications for anti-reflux surgery
• Persistence of symptoms after 8-12 weeks of medical treatment • Recurrence of symptoms • presence of complications • presence of mechanically defective LOS on manometric study

MANAGEMENT OF COMPLICATED DISEASE

Surgery is the treatment of choice for severe complicated disease. Complications should be treated accordingly.

COMPLICATIONS

BARRETT'S ESOPHAGUS

Barrett's esophagus is a condition in which the epithelial lining of the esophagus is replaced by that is

similar to the lining of the intestine, a process is called **intestinal metaplasia.** Barrett's esophagus is uncommon.

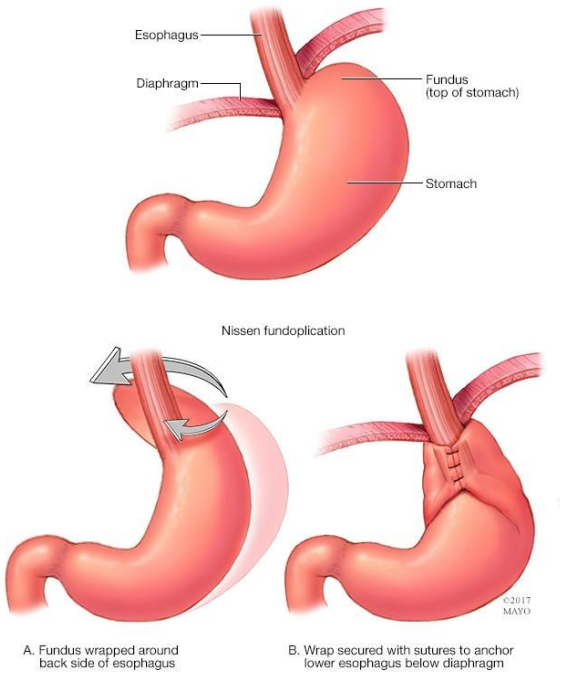


Figure 107 fundoplication

No signs or symptoms are associated with Barrett's esophagus, once Barrett's esophagus is diagnosed, there's a greater risk of developing esophageal cancer (may progress to high grade dysplasia, and finally to cancer, this happens only in about 1-5% of patients with Barrett's). Endoscopy must perform during the workup of GERD to allow taking biopsies to look for dysplasia. The intestinal metaplasia and the length of the Barrett's segment are important risk factors for the development of carcinoma.

Currently, there are no medications to reverse Barrett's esophagus. it appears that treating the underlying GERD may slow the progress of the disease and prevent complications by reducing acid reflux and strengthening the LES.

STRICTURES

Reflux-induced strictures usually occurs mainly in the late middle aged and elderly, but may present in children. Its occur in the lower esophagus. It's important to distinguish between benign reflux induced strictures and carcinoma in the lower esophagus. They are usually 1-4 cm in length and occur due to submucosal fibrosis. . Peptic strictures generally respond well to dilatation and long-term

treatment with a PPI. As most patients are elderly, anti-reflux surgery is not usually considered.

OTHER COMPLICATIONS

The risk of metaplasia and malignant transformation is increased with GERD. Pulmonary complications may also occur.

MOTOR ESOPHAGEAL DISORDERS

The most common motility disorders of the esophagus are shown in the table below

Motor disorders of the esophagus
Primary motor abnormality • Achalasia • Diffuse esophageal spasm • Nonspecific motor abnormality Secondary motility disorders • scleroderma • systemic lupus erythematosis

ACHALASIA

Achalasia is a primary motor esophageal disease that involves a failure of the lower esophageal sphincter to

relax, resulting in a progressive obstruction of the esophagus with dilatation of the lower esophageal segment. The disease occurs at any age, but usually after 30 years. Both sexes are equally affected.

ETIOLOGY

Achalasia is thought to be due to a partial or complete degeneration of the myenteric plexus of Auerbach. This may result in the loss of the inhibitory effect on smooth muscles resulting in a failure of relaxation of the LES. Most of the cases are without an identifiable cause (primary achalasia).

Infestation with the protozoon, *Trypanosoma cruzi,* which occurs in South America (Chagas' disease), also causes degeneration of the myenteric plexus, leading to a motor disorder of the esophagus that is indistinguishable from achalasia (secondary achalasia).

CLINICAL FEATURES

There is *progressive* dysphagia over several years, often for both solids and liquids. There may also be retrosternal pain.

Other common symptoms include weight loss, halitosis and regurgitation of undigested food, which can lead to aspiration pneumonia, particularly at night (nocturnal choking), resulting in bouts of coughing and recurrent chest infections.

INVESTIGATIONS

An erect chest X-ray might demonstrate a widened mediastinum, A fluid level behind the heart. The gastric air bubble is usually absent as a result of incomplete emptying of the esophagus.

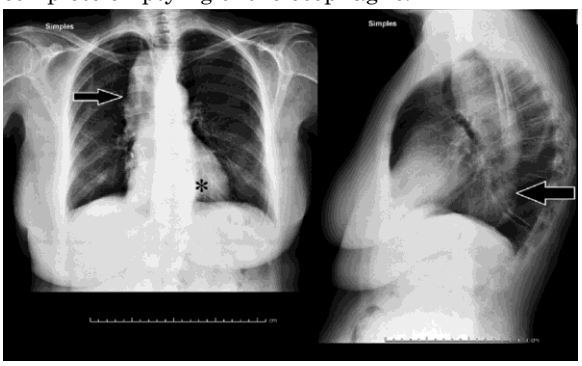


Figure 108 an erect chest X-ray for a patient with achalasia showing a widening in the mediastinum and the absence of a gastric bubble.

A barium swallow will show dilatation of the esophagus, leading to a tapered narrowing at the lower end (figure 7). endoscopy is essential to exclude other causes of lower esophageal narrowing, in particular carcinoma.

Using manometry, The demonstration of a high-pressure non-relaxing lower esophageal sphincter, with poor or absent peristalsis of the esophageal body, is diagnostic of achalasia.

TREATMENT

Treatment involves either balloon dilatation of the lower oesophageal sphincter or surgical myotomy (Hellers myotomy; division of the muscles over the lower esophagus and proximal stomach).

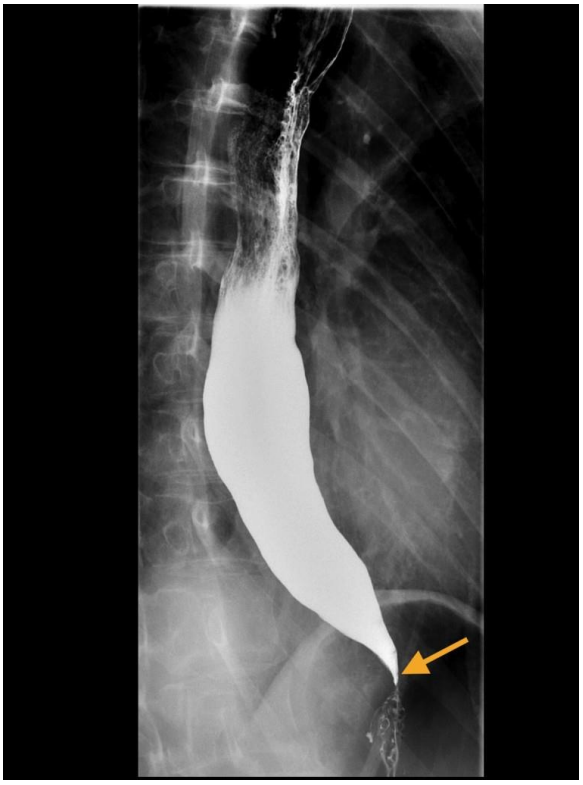


Figure 109 Barium swallow demonstrating achalasia

DIFFUSE ESOPHAGEAL SPASM

This disorder tends to occur in middle-aged to elderly patients. Complaints are of *intermittent* dysphagia and retrosternal pain, which can mimic angina. The symptoms are caused by repetitive irregular peristalsis of the esophageal body.

Barium swallow shows a characteristic corkscrew esophagus (figure 8), caused by the contracted muscle indenting the lumen, esophageal manometry confirms the diagnosis. Medical treatment includes calcium channel blockers and proton pump inhibitors. Surgical treatment involves a long **myotomy.**

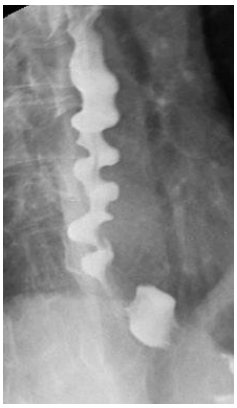
CAUSTIC INJURIES OF THE ESOPHAGUS

Figure 110 corkscrew esophagus typical of diffuse esophageal spasm.

a caustic injury of the esophagus is one which results from chemical reaction that results from ingestion of harmful chemicals. Approximately 10% of caustic ingestions result in severe injury requiring treatment.

ETIOLOGY

in Accidental caustic lesions occur mainly in chil-



dren, and in general, rather small quantities of caustics are taken. In adults or teenagers, the swallowing of caustic liquids is usually deliberate, during suicide attempts, and greater quantities are swallowed.

The vast majority of caustic chemicals are acidic or alkaline substances. Alkalies are more frequently swallowed accidentally than acids, because strong acids cause an immediate burning pain in the mouth, uncommon caustic agents, may have important toxic concerns including hydrogen fluoridephenol, zinc chloride and mercuric chloride.

PATHOLOGY

The severity and extent of the injury is determined by the amount of the substance involved, the duration of the contact (i.e. proportional), the type of the substance (usually its physical properties, especially the pH; strong acids pH< 2, strong bases pH>12), the ability of the substance to penetrate the tissue (alkalies dissolve tissue and thus penetrate deeply while acids cause coagulative necrosis) and its titrable reserve.

Injury occurs rapidly after alkaline ingestion, within minutes of contact. The most severely injured tissues are those that first contact the alkali, that is the squamous epithelial cells of the oropharynx, hypopharynx, and esophagus. Tissue edema occurs immediately and may persist for 48 hours, stricture formation primarily depends upon burn

depth, more severe burns may also be associated with esophageal perforation.

CLINICAL FEATURES

The patient usually presents with a history of ingestion of chemicals, complaints consist of pain in the mouth, retrosternal chest pain or/and abdominal pain, hypersalivation, dyspnea, odynophagia and dysphagia. Bleeding can occur, and frequently the patient vomits.

If strictures are to occur, the complaint of dysphagia reappears and is due to fibrosis and retraction, resulting in narrowing of the esophagus. On examination, the oropharynx may be inflamed. Signs of severe injury on examination are shown in the table below.

Signs of severe esophageal injury

- **Altered mental status**
- **peritoneal signs**
- **stridor**
- **hypotension**
- **shock**
- **Subcutaneous air (surgical emphysema)**

MANAGEMENT

CONSERVATIVE

Patients should be resuscitated and given appropriate opiate analgesia. They should also be encouraged to drink water to dilute the corrosive. Thereafter, the esophagus should be rested and the patient kept NPO and commenced on intravenous fluids and antibiotics. Vomiting should be actively discouraged, as this can cause further burns and risks perforation, if the patient's oropharynx is severely inflamed, an artificial airway (tracheostomy) might be required.

INTERVENTIONAL

The key to management is early endoscopy by an experienced doctor to inspect the whole of the esophagus and stomach. Air insufflation should be kept to a minimum to avoid esophageal perforation. Deep ulcers and the recognition of a grey or black eschar signify the most severe lesions with the greatest risk of perforation. Minor injuries with only edema of the mucosa resolve rapidly with no late sequelae. These patients can safely be fed. With more severe injuries, a feeding jejunostomy may be appropriate until the patient can swallow saliva satisfactorily. Severe mucosal injury should treated by steroid for 3 weeks, followed by regular dilatation (to prevent stricture formation).

ESOPHAGEAL CANCER

Esophageal cancer is the ninth most common cancer in the world. It is an extremely aggressive tumor with a poor survival rate. Only 5-10% of those diagnosed will survive for 5 years. In general, it is a disease of mid to late adulthood, and is 3 times more common in females.

ETIOLOGY AND EPIDEMIOLOGY

The incidence of esophageal cancer varies geographically more than that of any other cancer. Factors which increase the risk of developing esophageal cancer are shown in the table below.

Risk factors for developing esophageal cancer.

Diet

- **consumption of food at high temperature.**
- **pickled vegetables**
- **preserved meat, salted dry fish**
- **food contaminated with fungi**

Alcohol

Corrosive stricture

Achalasia

Tobacco (smoking, chewing)

Barrett esophagus(the detection of Barret's esophagus on endoscopy and biopsy increases the future risk of cancer by >40x compared to individuals without Barret's esophagus)

PATHOLOGY

The esophagus is lined by stratified squamous epithelium. Historically the majority of esophageal malignancies were squamous carcinomas which usually affect the upper two thirds. The minority were adenocarcinomas occurring in the lower third of the esophagus, probably derived from metaplastic intestinal mucosa, i.e. Barrett's esophagus(transformation of distal esophageal epithelium from squamous to a specialized columnar epithelium capable of further neoplastic progression) . currently adenocarcinoma makes up 60-70% of new cases.

CLINICAL FEATURES

Dysphagia that progresses from solids to liquids is one of the most common presentations. odynophagia, regurgitation and cough (from aspiration pneumonitis) are other forms of presentation.

Occasionally, patients may present with metastatic disease, including enlarged cervical lymph nodes, hoarseness from recurrent laryngeal nerve involvement, and chest pain from mediastinal invasion. Other general features of malignancy include weight loss, anorexia, and lassitude.

INVESTIGATIONS

Even if the diagnosis is made initially by barium swallow (Figure 9), it must always be confirmed by endoscopy and biopsy. For this reason, endoscopy is the best first-line investigation for anyone with dysphagia. A barium swallow will show a filling defect in the esophagus, stenosis of the esophageal canal or/and shouldering effect.

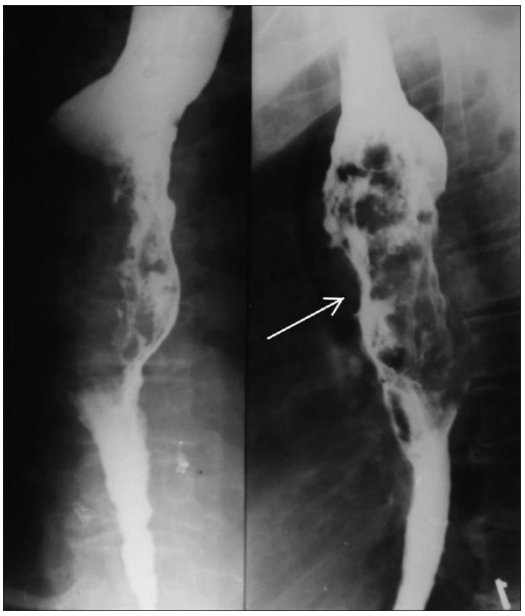


Figure 111 A-Barium swallow in a patient with a carcinoma of the mid-esophagus (filling defect, arrow). B-esophageal cancer causing stenosis of the esophageal canal and shouldering appearance.

Thereafter, investigations are aimed at accurate staging of the disease (see the table below). Local T (tumor) stage and N (nodal) spread are best assessed by endoscopic ultrasonography, M (metastases) stage can be assessed with chest X-ray (lung secondaries), abdominal ultrasound (liver metastases and ascites), CT of the chest and abdomen (lung and liver metastases, distant lymphadenopathy) and laparoscopy (peritoneal metastases). Some units employ thoracoscopy and radioisotope scanning, searching for secondaries in the pleura and bone, respectively.

MANAGEMENT

Management depends on the stage of the disease and risk of mortality and morbidity with each modality of treatment. Treatment should include a multidisciplinary team (radiologist, oncologist, surgeon).

SURGICAL TREATMENT

The choice of surgical operation depends on the extent and location of the tumor.

CURATIVE SURGERY

Patients with disease confined to the esophagus and who are fit for surgery should be considered for resection. A certain proportion will be found to have more extensive disease at operation, but with better preoperative staging, this figure should be small, resection should be associated with removal of regional- lymph nodes with restoration of continuity.

Lesions above the carina (the tracheal bifurcation) are usually dealt with by a three stages Esophagectom**y** known as the **McKeown operation.** If the tumor arises lower in the esophagus, a two-stage **Ivor Lewis** operation is usually performed. Both of these operations require the chest to be opened through a thoracotomy. Trans hiatal esophagectomy has been gaining popularity as it avoids opening the chest.

PALLIATIVE TREATMENT

Palliative surgery (resection, bypass) can be used but the risks must be weighed against the benefits of these major surgeries.

Endoscopic dilatation may provide short-term relief and must be repeated at ever-shortening intervals, laser ablation can be carried out endoscopically and provides very good palliation of dysphagia, but does require to be repeated at regular intervals of 1-2 months. Perforation can result. Alternatively, the esophagus can be **intubated** by inserting a cloth-covered metal stent through the lesion.

staging of esophageal cancer	
T0	No evidence of primary tumor
Tis	High grade dysplasia (carcinoma in situ)
T1	Tumor invades the lamina propria
T2	Tumor invades muscularis externa
T3	Tumor invades the adventitia
T4	Tumor invades adjacent structures
N0	No regional lymph nodes metastasis
N1	Regional lymph nodes metastasis

M0	No distant metastasis		
Ml	Distant metastasis		
Stage	description		
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage IIA	T2	N0	M0
	T3	N0	M0
Stage IIB	T1	N1	M0
	T2	N1	M0
Stage III	T3	N1	M0
	T4	Any N	M0
Stage IV	Any T	Any N	Ml

ABDOMINAL WALL ANATOMY AND HERNIAS

ANATOMY

ABDOMINAL WALL

The Superficial layer of the abdominal wall is formed of fatty connective tissue, in the lower region of the -anterior part of the abdominal wall, below the umbilicus, it forms two layers: a superficial fatty layer and a deeper membranous layer.

The superficial fatty layer of superficial fascia (Camper's fascia) is continuous with the superficial fascia throughout other regions of the body. The deeper membranous layer of superficial fascia (Scarpa's fascia) continues into the thigh to fuse with the fascia lata and it is firmly attached to the linea alba and the symphysis pubis. It continues into the anterior part of the perineum where it is firmly attached and is referred to as the superficial perineal fascia (Colles' fascia).

The anterior abdominal wall has five muscles; -the external oblique, internal oblique, the transversus abdominis muscle, the rectus abdominus and in some people the pyramidalis. The transversalis fascia lines the inner aspect of the transversus abdominis muscle covers the peritoneum.

The rectus abdominis and pyramidalis muscles-are enclosed in an aponeurotic tendinous sheath (the rectus sheath) formed by a unique layering of the aponeuroses of the external and internal oblique, and transversus abdominis muscles. Deep to the transversalis fascia is a layer of connective tissue, the extraperitoneal fascia separates the transversalis fascia from the peritoneum containing varying amounts of fat.

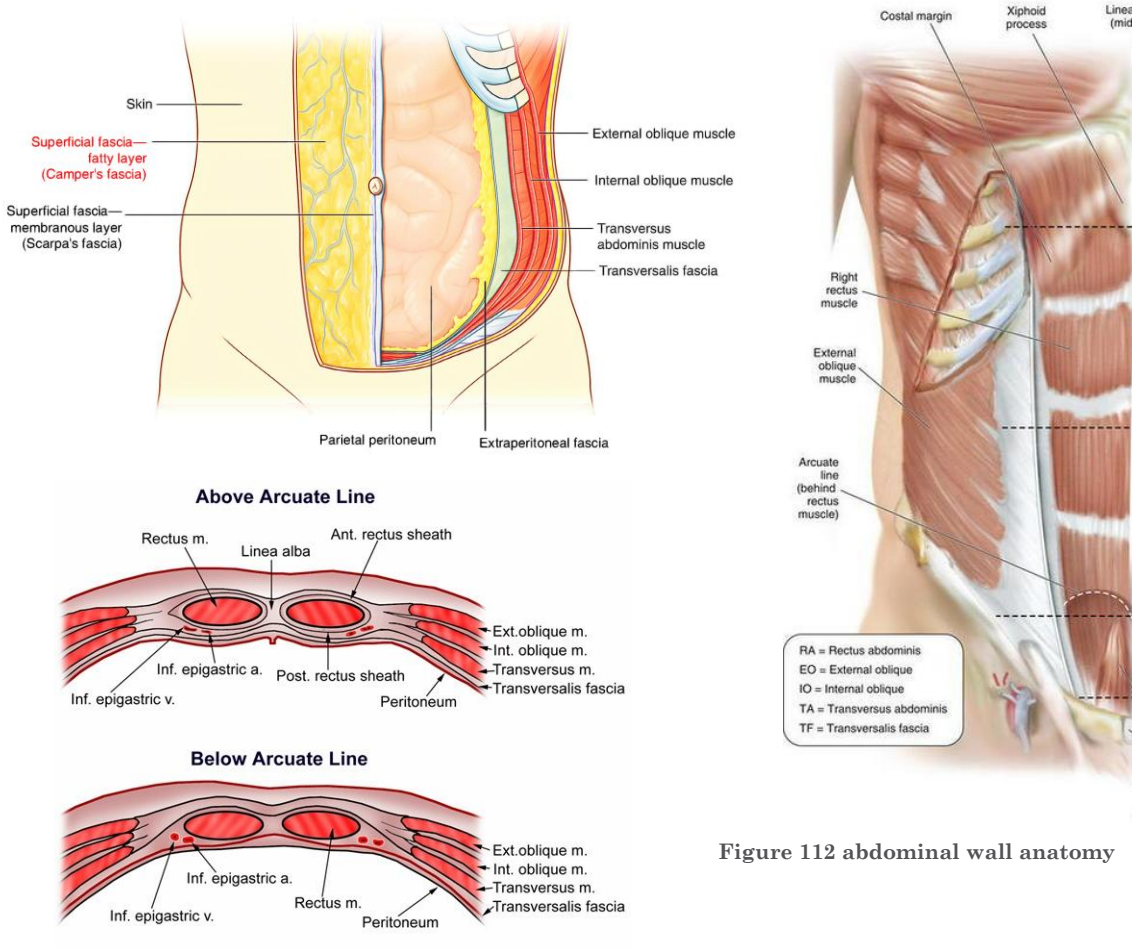


Figure 112 abdominal wall anatomy

At a point midway between the umbilicus and the pubic symphysis, there is no posterior wall of the rectus sheath and the anterior wall of the sheath consists of the aponeuroses of the external oblique, the internal oblique, and the transversus abdominis muscles. From this point inferiorly, the rectus abdominis muscle is in direct contact with the transversalis fascia. Marking this point of transition is an arch of fibres; the arcuate line. The lateral edge of the rectus sheath is referred to as linea semilunaris (spigelian line); it is the site for spigelian hernia.

The skin and muscles of the anterolateral abdominal wall are supplied by T7 to T12 and L1 spinal nerves. Branches of L1 (the iliohypogastric nerve and ilioinguinal nerve), which originate from the lumbar plexus supply the skin on and structures within the lower abdominal wall.

THE INGUINAL CANAL (FIGURE 2)

The inguinal canal is a slit-like passage that extends in a downward and medial direction, just above and parallel to the lower half of the inguinal ligament. It begins at the deep inguinal ring and continues for approximately 4 cm, ending at the superficial inguinal ring. The following is a detailed description of inguinal canal anatomy;

- Anterior wall; The anterior wall of the inguinal canal is formed along its entire length by the aponeurosis of the external oblique muscle. It is also reinforced laterally by the medial fibres of the internal oblique muscle.

- Posterior wail; The posterior wall of the inguinal canal is formed along its entire length by the transversalis fascia. It is reinforced along its medial one-third by the conjoint tendon (combined insertions of the transversus abdominis and internal oblique muscles).

- Roof; The roof (superior wall) of the inguinal canal is formed by the arching fibres of the transversus abdominis and internal oblique muscles.

- Floor; The floor (inferior wall) of the inguinal canal is formed by the medial one-half of the inguinal ligament.

The contents of the inguinal canal are the genital branch of the genitofemoral nerve, the spermatic cord in men and the round ligament of the uterus in women. Additionally, in both sexes, the ilio-inguinal nerve passes through part of the canal, exiting through the superficial inguinal ring with the other contents.

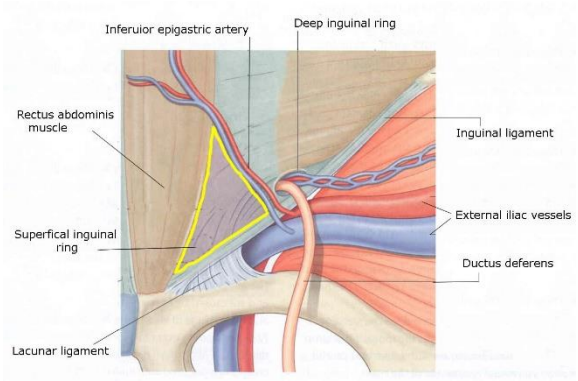


Figure 2 THE INGUINAL CANAL

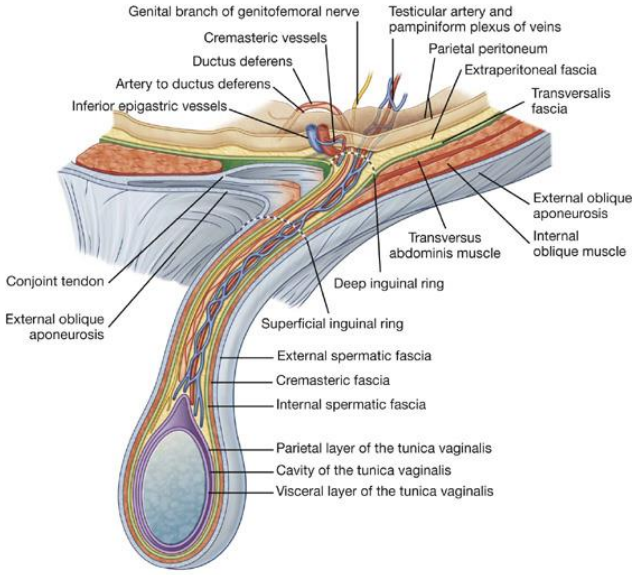


Figure 3 the inguinal canal and spermatic cord

ABDOMINAL WALL HERNIAS

A hernia is a protrusion of a viscus or part of a viscus through an abnormal opening in the walls of its containing cavity. Abdominal wall hernias are the most common variety.

As a rule, a hernia consists of three parts - the sac, the coverings of the sac and the contents of the sac. The sac is a diverticulum of peritoneum, consisting of mouth, neck, body and fundus (figure 1).

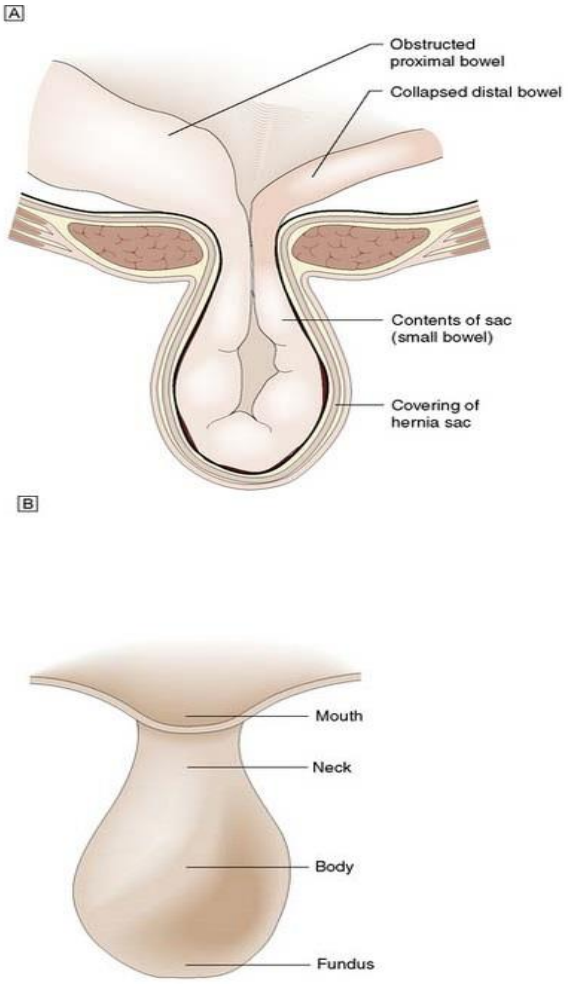


Figure 113 anatomy of a hernia

Coverings are derived from the layers of the abdominal wall through which the sac passes. The contents can be intestines (enterocele), omentum (omentocele), a portion of the bladder or an ovary with or without the corresponding fallopian tube. When the contents contain only a portion of the circumference of the intestine, it is referred to as Richter's hernia (figure 2). When the content is a Meckel's diverticulum, the hernia is referred to as Littre's hernia. When there is more than one loop of bowel present in the sac, the condition is referred to as maydl's hernia. A sliding hernia is one in which the contents form part of the wall of the sac.

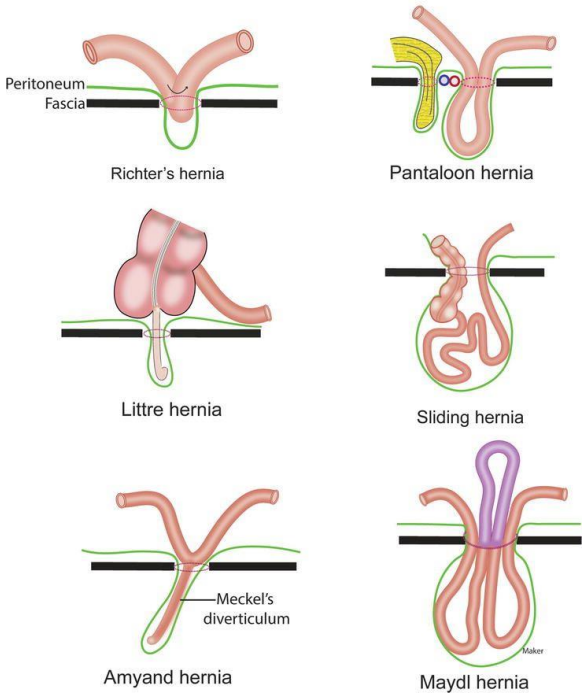


Figure 114 some types of hernias

Irrespective of the site, hernias can be classified as the following;

- Reducible; The hernia either reduces itself when the patient lies down or can be reduced by the patient or the surgeon.
- Irreducible; In this case the contents cannot be returned to the abdomen but there is no evidence of other complications. It is usually due to adhesions between the sac and its contents or overcrowding within the sac. The term 'incarceration' is often used to indicate that the lumen of that portion of the colon occupying a hernial sac is blocked with feces.
- Obstructed; This is an irreducible hernia but has good blood supply.
- Strangulated; A hernia becomes strangulated when the blood supply of its contents is seriously impaired, rendering the contents ischemic.

According to the site, hernias can be uni- or bilateral, left or right and localized to one of the sites depicted in figure 3.

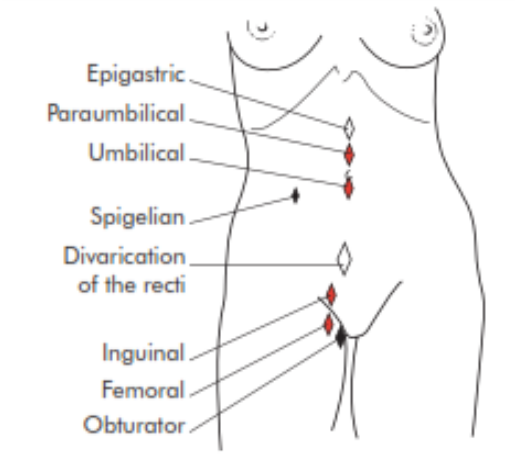


Figure 115 sites for abdominal wall hernia

INGUINAL HERNIAS

Inguinal hernias are the most common form of abdominal wall hernias. According to the site of exit, inguinal hernias can either be direct or indirect. The anatomical distinction is shown in figure 7. Indirect hernias exit through the internal inguinal ring, while direct hernia push through the fascia transversalis in a defect that can be bounded by the hesselbach triangle (see later).

INDIRECT INGUINAL HERNIAS

These represent the most common form of inguinal hernias. They are more common in children (refer to pediatric surgery lectures (inguinoscrotal conditions)). In adult males, 65% of inguinal hernias are indirect and 55% are right-sided. The hernia is bilateral in 12% of cases.

ETIOLOGY

An indirect hernia may occur in a congenital preformed sac - the remains of the processus vaginalis, which is usually obliterated soon after birth, persistence of the processus vaginalis can allow the abdominal Viscera to easily herniate through the internal inguinal ring (figure 8). Causes of increased intra-abdominal pressure (e.g. chronic cough, constipation) may further weaken the internal ring and cause viscera to herniate.

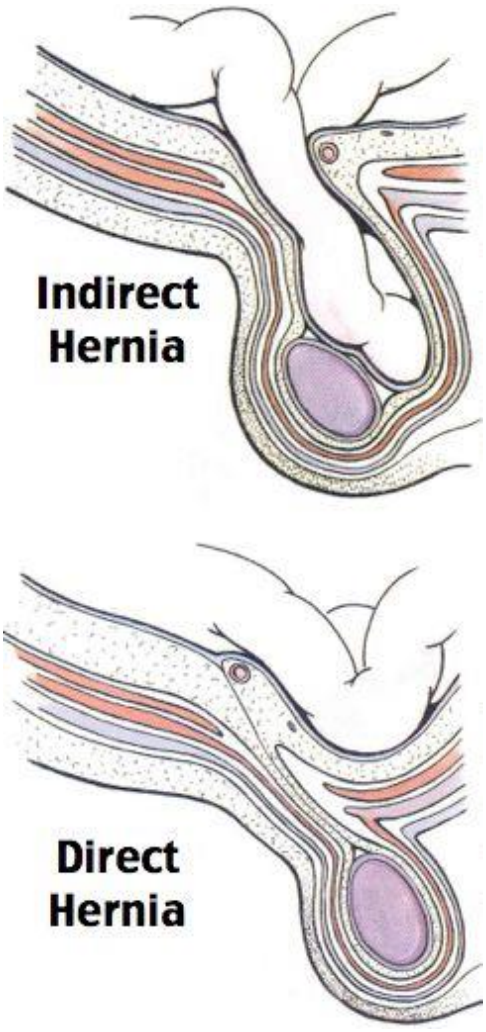


Figure 116 A indirect hernia, B direct hernia

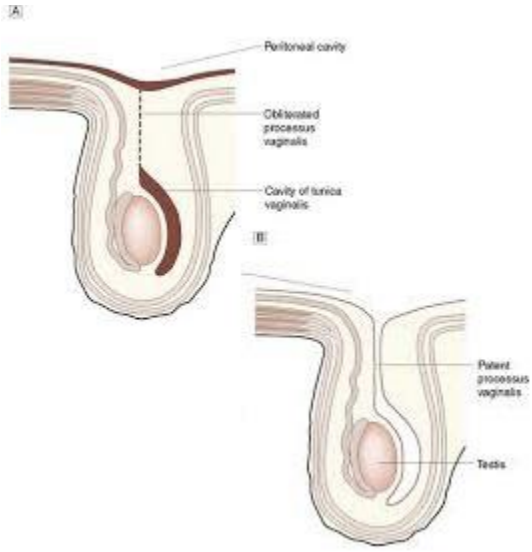


Figure 117 persistence of the processus vaginalis

SURGICAL ANATOMY

Indirect hernias exit through the internal inguinal ring. They enter the inguinal canal and pass obliquely in three forms (figure 9);

• bubonocele: in which the sac doesn't extend beyond the external ring (figure 9-A).

• funicular sac: in which there is an obliterated segment that intervenes between the tunica vaginalis and the herniating sac. It may extend beyond the external inguinal ring, (figure 9-B).

• Complete or scrotal: the mass extends beyond the external inguinal ring into the scrotum and is in communication with the tunica vaginalis, (figure 9-C).

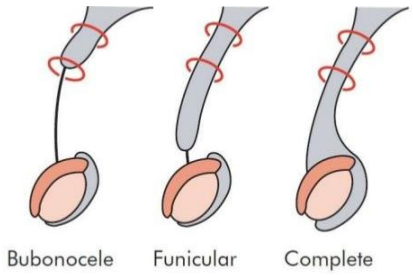


Figure 9 types of oblique

CLINICAL FEATURES

Patients seek medical attention because of a painless bulge in the groin or an annoying heavy feeling or dragging sensation. Usually they are asymptomatic and the hernia is discovered on routine examination.

Examination is diagnostic. Patients with indirect inguinal hernias have a bulging mass above the groin crease (medial and above the pubic tubercle) which, with coughing, increases in size (expansile cough impulse) and is more palpable (palpable cough impulse). The hernia usually reduces spontaneously (when the patient lie down) or by gentle pressure. If the hernia contents are intestines, bowel sounds can be heard on auscultation. To distinguish a direct from an indirect hernia, deep ring occlusive test is used. By pressing firmly on the internal inguinal ring (2 cm above the mid inguinal point), notice whether there is a cough impulse or not. If so, at this point it is probably a direct hernia, while if not it is probably an indirect one.

The differential diagnosis of a groin swelling is shown in the table below.

differential diagnosis of a groin swelling
• **Inguinal Hernia** • **Femoral Hernia** • **Hydrocele** • **Lymph Node** • **Lipoma** • **Saphina Varix** • **Femoral Aneurysm** • **EctopicTestis** • **Psoas Abscess**

MANAGEMENT

Operation is the treatment of choice. The basic operation is inguinal herniotomy, which entails opening the hernial sac, freeing it from the spermatic cord, reducing any contents and then *excising* the hernia! sac after transfixing and ligating its neck. It is employed either by itself or as the first step in a repair procedure (herniorrhaphy). Herniorrhaphy is used in adults whom usually present with a stretched or widened internal ring and the procedure implies tightening the ring and strengthening the posterior wall. Laproscopic or pre-peritoneal approaces are now in use.

A truss may be used when operation is contraindicated or is refused by the patient.

DIRECT HERNIA

In adult males, 35% of inguinal hernias are direct. At presentation, 12% of patients will have a contralateral hernia. May occur in males or females, but males are ten times more likely to get a direct inguinal hernia.

ETIOLOGY

A direct inguinal hernia is always acquired. Predisposing factors are smoking and occupations that involve straining and heavy lifting (or anything that increase the intra-abdominal pressure). Damage to the ilioinguinal nerve (previous appendectomy) is another cause, because of the resulting weakness of the conjoined tendon.

SURGICAL ANATOMY

The sac passes through a weakness or defect of the transversalis fascia in the posterior wall of the inguinal canal. The bulging occurs medial to the inferior epigastric vessels in the area referred to as the inguinal triangle (Hesselbach's triangle, figure 10), which is bounded laterally by the inferior epigastric artery, medially by the rectus abdominis muscle and inferiorly by the inguinal ligament.

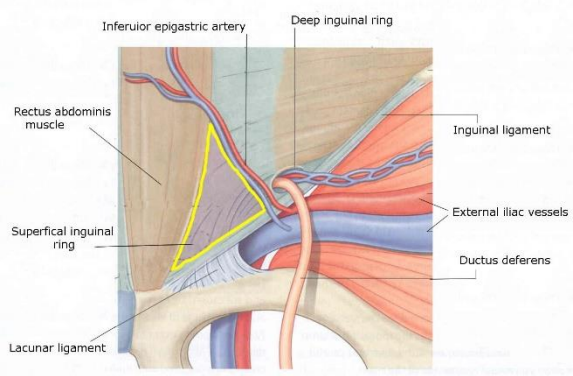


Figure 10 view of the abdominal wall showing the inguinal triangle (Hesselbach's triangle), the site for direct inguinal Hernia.

The hernia can descend through the external inguinal ring but the fascia transversalis cannot stretch sufficiently to allow it to descend down into the scrotum. As the neck of the sac is wide, direct inguinal hernias do not often strangulate and are almost always reducible.

CLINICAL FEATURES

Patients present with a diffuse bulging mass in the inguinal region. The mass is located above the groin- crease, increases in size and is more palpable with coughing. It is usually reducible. The deep ring occlu¬sive test will show that the mass still have a positive cough impulse after pressure on the deep ring.

MANAGEMENT

The principles of repair of direct hernias are the same as those of an indirect hernia, with the exception that the hernia sac can usually be simply inverted after it has been dissected free and the transversalis fascia reconstructed in front of it. Reconstruction of the transversalis fascia is done by overlapping repair or synthetic mesh insertion.

FEMORAL HERNIA

Femoral hernia is the third most common type of primary hernia. It accounts for about 20% of hernias in women and 5% in men. it is the most liable to become strangulated, mainly because of the narrowness of the neck of the sac and the rigidity of the femoral ring. Strangulation is the initial presentation of 40% of femoral hernias.

ETIOLOGY

In both sexes, femoral hernia is assumed to be acquired. It is likely that the portal of the femoral canal (see below) is larger in women, possibly as a result of a smaller muscle bulk of iliopsoas and pectineus. Increased intra-abdominal pressure and other factors related to pregnancy may also be important in females since the incidence of femoral hernia is higher in multiparous than nulliparous women.

SURGICAL ANATOMY

The femoral canal occupies the most medial compartment of the femoral sheath. A femoral hernia projects through the femoral ring and passes down the femoral canal. The ring is bounded laterally by the femoral vein, anteriorly by the inguinal ligament, medially by the lacunar ligament, and posteriorly by the superior ramus of the pubis and the reflected part of the inguinal ligament (pectineal ligament of Astley Cooper).

As the hernia enlarges, it passes through the saphenous opening in the deep fascia of the thigh (the site of penetration of the long saphenous vein to join the femoral vein) and then turns upwards to lie in front of the inguinal ligament.

CLINICAL FEATURES

The incidence of femora! hernia rises with age. The right side is affected twice as often as the left and in 20% of cases the condition is bilateral.

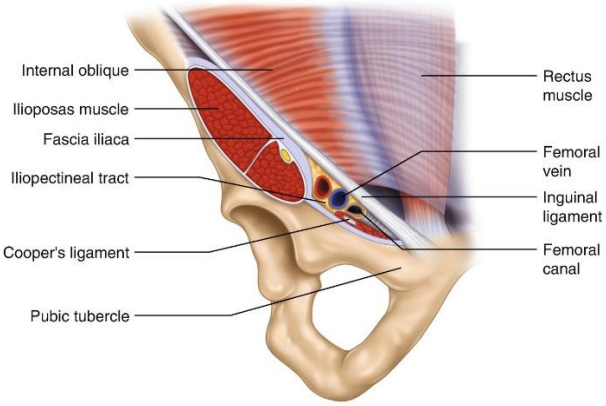


Figure 11 anatomy of the femoral ring the site of femoral hernia

Patients are usually asymptomatic but are likely to present with a strangulated hernia. Most patients present with a groin swelling or mass. The mass is differentiated from inguinal hernia by its location below the groin crease (below and lateral to the pubic tubercle; figure 12). A femoral hernia is frequently difficult or impossible to reduce because of its J-shaped course and the tight neck of the sac.

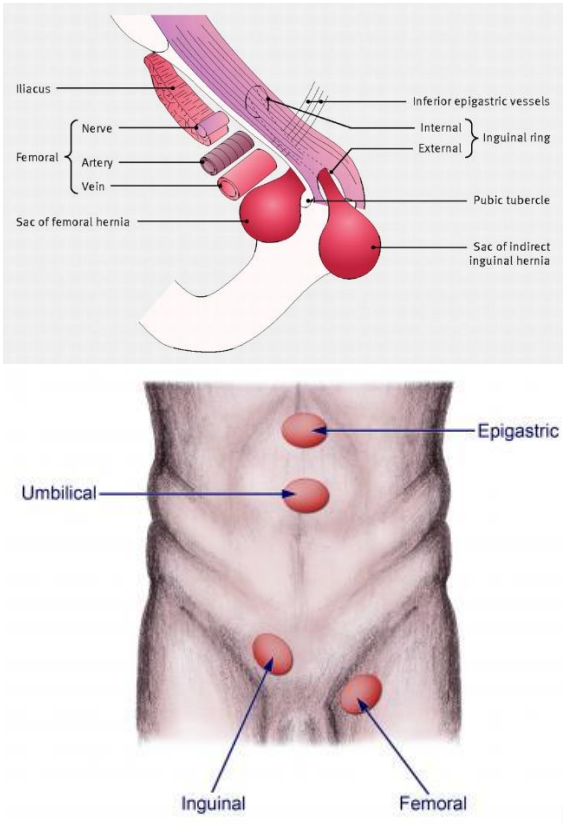


Figure 118 the essential differential diagnosis of the femoral and inguinal hernia

The differential diagnosis of a femoral hernia is the same as that for groin swellings.

MANAGEMENT

A femoral hernia is particularly likely to obstruct and strangulate, and therefore surgical intervention is indicated. The aim of the operation is to excise the sac and obliterate the femoral ring by suturing the inguinal ligament to the pectineal ligament. The femoral canal can be approached from below the inguinal ligament **(Lockwood approach),** through the inguinal canal **(Lotheissen approach),** or from above by entering the rectus sheath and displacing the rectus abdominis medially **(McEvedy approach).**

VENTRAL HERNIAS

EPIGASTRIC HERNIA

Epigastric hernias protrude through the linea alba above the level of the umbilicus. It is likely that an epigastric hernia is the direct result of a sudden strain tearing the interlacing fibres of the linea alba. The herniation may consist of extraperitoneal fat (fatty hernia of the linea alba) or may be a protrusion of peritoneum containing omentum (true epigastric hernia).

Most of the cases are asymptomatic. Patients may present with epigastric discomfort. It is repaired by closing the defect with interrupted non absorbable sutures.

UMBILICAL HERNIA

This is more likely to occur in infants and children. The hernia is often symptomless but increases in size on crying and assumes a classical conical shape. Conservative treatment is indicated under the age of 2 years. Over 95% of these hernias close spontaneously in the first 3 years of life. Persistence after the third birthday is an indication for elective repair. Surgery involves excision of the hernial sac and closure of the defect in the fascia of the abdominal wall.

PARAUMBILICAL HERNIA

In adults the hernia does not occur through the umbilical scar but is a protrusion through the linea

alba, just above or sometimes just below the umbilicus.

ETIOLOGY

Women are more affected than men. Paraumbilical hernia tend to occur in obese multiparous women with flabby abdominal muscles.

CLINICAL FEATURES

The hernia gradually enlarges, the covering tissues become stretched and thin, and eventually loops of bowel may become visible under parchment-like skin, with a tendency to sag downwards. The sac may be irreducible because of adhesions that form between omentum and loops of bowel.

Symptomatically, a large umbilical hernia causes a dragging pain because of its weight. Often there are transient attacks of intestinal colic because of partial intestinal obstruction. The skin may become reddened, excoriated and ulcerated, and rarely an intestinal fistula may even develop.

TREATMENT

If the hernia is untreated it increases in size and more and more of its contents become irreducible. Eventually, strangulation may occur. Thus, operation should be advised in nearly all cases. For small defects **umbilical Herniorrhaphy (mayo's repair)** is performed. Paraumbilical hernioplasty is performed in the case of very large primary umbilical hernias. Additional lipectomy is performed when there is a large, pendulous, fat-laden abdominal wall.

INCISIONAL HERNIA

Incisional hernias occur after 3-5% of all abdominal operations, predisposing factors which may increase the risk of having a hernia after a surgical incision are " shown in the table below.

Predisposing factors for incisional hernia	
• **Cough**	• **Early return to strenuous work and activity**
• **Infection**	• **Malnutrition and some mineral and vitamins deficiencies**
• **Haematoma**	• **Chronic systemic diseases**
• **Seroma**	• **Difficult micturition**
• **Irradiation**	• **Early Post Operative pregnancy**
• **Malignancies**	
• **Chemo Therapy**	
• **Steroids**	
• **Constipation**	

Surgical repair is usually advised. Some patients prefer to wear an abdominal support to control the hernia. The skin wound is excised and flaps are elevated to expose the aponeurosis. The sac can be invaginated or excised, and the edges of the defect may be repaired with overlapping sutures, but repair with the insertion of a synthetic mesh is preferred

OTHER FORMS OF ABDOMINAL WALL HERNIA

A **Spigelian hernia** occurs through the linea semilunaris at the outer border of the rectus abdominis muscle. Treatment is surgical, as the hernia is liable to strangulate.

Both indirect and direct hernias may occur on the same side (pantaloon or saddle-bag hernia), with sacs straddling the inferior epigastric vessels.

PERITONEUM

The peritoneal cavity is lined by the parietal peritoneum, a mesothelial lining. This lining is called the visceral peritoneum where it is reflected onto the enclosed abdominal organs (figure 1). As opposed to the parietal peritoneum, the visceral peritoneum is poor in nerves, blood vessels and lymphatics. The peritoneal surface area is a semipermeable membrane with an area comparable to that of the cutaneous body surface (~2 m2). The circulation of peritoneal fluid is directed toward lymphatics in the under surface of the diaphragm. There, particulate matter including bacteria up to 20 µm in size is cleared via stomas in the diaphragmatic mesothelium and lymphatics and discharged mainly into the right thoracic duct.

This upward movement of peritoneal fluids is responsible for the occurrence of many subphrenic abscesses. Normally, there is less than 50 ml of free peritoneal fluid (a transudate).

PERITONITIS

Peritonitis is an inflammatory or suppurative response of the peritoneal lining the abdominal wall and the covering of the abdominal viscera to direct irritation. Primary peritonitis is uncommon (< 1%), although in childhood it can account for up to 15% of acute abdominal emergencies. Secondary peritonitis results from bacterial contamination originating from within viscera or from external sources (e.g., penetrating injury).

ETIOLOGY AND BACTERIOLOGY

Most cases of peritonitis are caused by an invasion of the peritoneal cavity by bacteria, so that when the term 'peritonitis' is used without qualification, bacterial peritonitis is implied.

PRIMARY BACTERIAL PERITONITIS

primary bacterial peritonitis is a much rarer condition that occurs in otherwise healthy people in the absence of surgery or trauma and is the result of primary infection of the peritoneal lining by streptococcal organisms, usually in children and adult females. Other causes include pneumococci and Hemophilus species.

SECONDARY BACTERIAL PERITONITIS

Bacteria implicated in secondary bacterial peritonitis are shown in the table below.

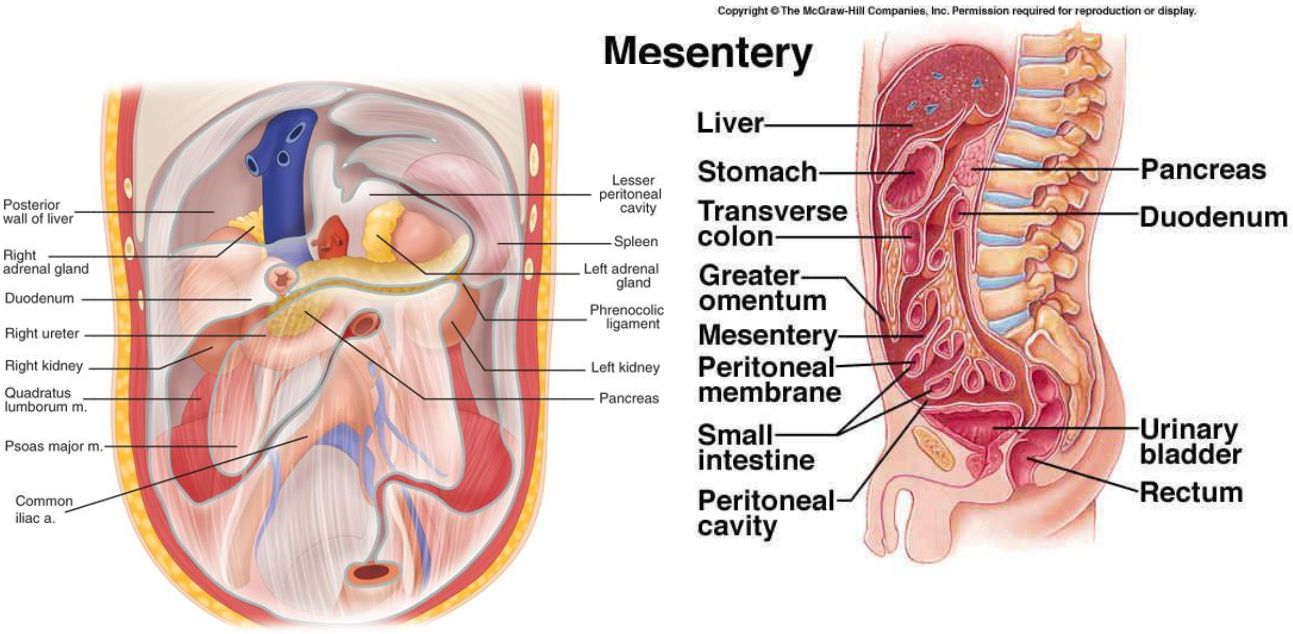


Figure 119 peritoneal cavity and peritoneal membranes

Causes of bacterial peritonitis

Gastrointestinal source

- **Gram positive aerobes; *Streptococci, Staphylococcus***
- **Gram negative anaerobes; *Bacteroides***
- **Gram positive anaerobes; *Staphylococcus, Clostridium***
- **Gram negative aerobes; *Escherichia coli, Klebsiella pneumonia, pseudomonas aeurigonosa***

Other sources

- ***Chlamydia***
- **Gonococcus**
- **β-Haemolytic streptococci**
- **Pneumococcus**
- ***Mycobacterium tuberculosis***

The number of bacteria within the lumen of the gastrointestinal tract is normally low until the distal small bowel is reached, whereas high concentrations are found in the colon. The biliary and pancreatic tracts are normally free from bacteria.

Bacteroides are commonly found in peritonitis. These Gram negative, non-sporing organisms, although predominant in the lower intestine, often escape detection because they are strictly anaerobic and slow to grow on culture media unless there is an adequate carbon dioxide tension in the anaerobic apparatus. In many laboratories, the culture is discarded if there is no growth in 48 hours. Since the widespread use of

metronidazole (Flagyl), Bacteroides infection have greatly diminished.

PATHOGENESIS

Infecting organisms may reach the peritoneal cavity via a number of routes;

- Gastrointestinal perforation; e.g. perforated ulcer,
- diverticular perforation
- Exogenous contamination; e.g. drains, open surgery, trauma
- Transmural bacterial translocation (no perforation); e.g. inflammatory bowel disease, appendicitis, ischemic bowel
- Female genital tract infection; e.g. pelvic inflammatory disease
- Hematogenous spread (rare); e.g. septicaemia
- Perforation; perforated viscus
- Lymphatics

Even in patients with non-bacterial peritonitis (e.g. acute pancreatitis, intraperitoneal rupture of the bladder or hemoperitoneum), the peritoneum often becomes infected by transmural spread of organisms from the bowel, and it is not long (often a matter of hours; 6-12 hours) before a bacterial peritonitis develops.

Peritoneal infection is usually caused by two or more bacterial strains. Gram-negative bacteria contain endotoxins (lipopolysaccharides) in their cell walls that have multiple toxic effects on the host, primarily by causing the release of tumor necrosis factor (TNF) from host leukocytes (initiating SIRS). Systemic absorption of endotoxin may produce endotoxic shock with hypotension and impaired tissue perfusion. Other bacteria such as Clostridium welchii produce harmful exotoxins.

TNF mediates the release of plasminogen activator inhibitor produced by inflamed peritoneal mesothelial cells, which can lead to persistence of fibrin. Fibrin clots segregate bacterial deposits, a source of endotoxins that contribute to sepsis, but segregation may also inadvertently shield bacteria from bacteria- clearing mechanisms.

PATHOLOGY

The term localized peritonitis implies the containment of the infection after it has been translocated to the peritoneal cavity. This is usually due to collection of the infection at certain favourable anatomic sites (the subphrenic spaces, the pelvis and the peritoneal cavity proper (supracolic and an infracolic compartment by the transverse colon and transverse mesocolon), and the paracolic gutters, figure 2). The formation of fibrin clots and the aggregation of intestinal loops also limits the spread of the infection. The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection.

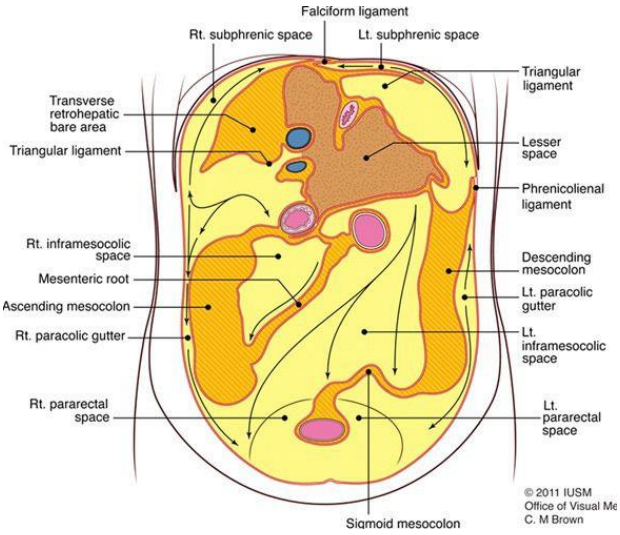


Figure 120 the peritoneal compartments

Diffuse (generalized) peritonitis occurs when the spread of bacterial contamination is faster or stronger than the mechanisms of localization. This can occur in sudden gush of infected fluid from a distended viscus (pistol-shot perforation), young children (small greater omentum), pathogenic strains and immunodeficiency.

CLINICAL FEATURES

Localised peritonitis is bound up intimately with the causative condition, and the initial symptoms and signs are those of that condition. When the peritoneum becomes inflamed, the temperature, and especially the pulse rate, rise. Abdominal pain increases and usually there is associated vomiting. The most important sign is guarding and rigidity of the abdominal wall over the area of the abdomen that is involved, with a positive 'release' sign (rebound tenderness). If inflammation arises under the diaphragm, shoulder tip ('phrenic[7]) pain may be felt. In cases of pelvic peritonitis arising from an inflamed appendix in the pelvic position or from salpingitis, the abdominal signs are often slight; there may be deep tenderness of one or both lower quadrants alone, but a rectal or vaginal examination reveals marked tenderness of the pelvic peritoneum. Patients may suffer diarrhoea in this case. Infrequently, localised peritonitis becomes diffuse. Conversely, in favourable circumstances, diffuse peritonitis can become localised, most frequently in the pelvis or at multiple sites within the abdominal cavity.

DIFFUSE (GENERALIZED) PERITONITIS

Diffuse (generalized) peritonitis may present in differing ways dependent on the duration of infection.

EARLY

Abdominal pain is severe and made worse by moving or breathing. It is first experienced at the site of the original lesion and spreads outwards from this point. Vomiting may occur. The patient usually lies still. Tenderness and rigidity on palpation are found typically when the peritonitis affects the anterior abdominal wall. Infrequent bowel sounds may still be heard for a few hours but they cease with the onset of paralytic ileus. the pulse rises progressively but, if the peritoneum is deluged with irritant fluid, there is a sudden rise. The temperature changes are variable and can be subnormal.

LATE

If resolution or localization of generalized peritonitis does not occur, the abdomen remains silent and increasingly distends. Circulatory failure ensues, with cold, clammy extremities, sunken eyes, dry tongue, thready (irregular) pulse, drawn and anxious face (Hippocratic facies; Figure 3) and diminished urine output. The patient finally lapses into unconsciousness. With early diagnosis and adequate treatment, this condition is rarely seen in modern surgical practice.

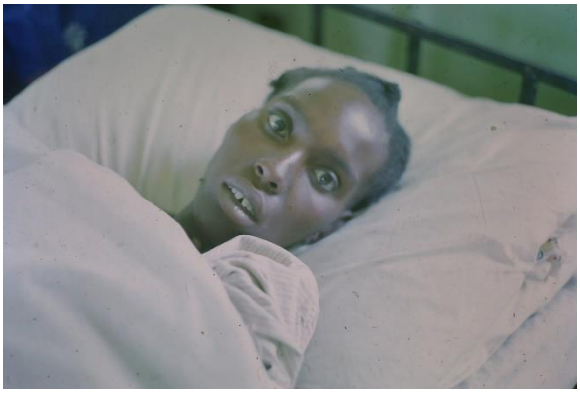


Figure 121 Hippocratic face in terminal defused peritonitis

INVESTIGATIONS

Diagnostic work up in a patient with suspected peritonitis is shown in the table above.

Diagnostic aids in patients with peritonitis

Blood

- **CBC and differential**
- **Blood grouping (for cross and matching)**
- **Blood culture**
- **Blood urea nitrogen, creatinine, electrolytes (kidney function test)**
- **Serum amylase**
- **Arterial blood gases**

Urine

- **Urine Analysis**
- **Urine output**

Radiology

- **Abdominal X-ray**
- **ultrasound or CT of the Abdomen**

ECG monitoring and oximetry

- Blood tests; In stable and unstable patients, blood is drawn for CBC, cross matching, urea and electrolytes (to assess kidney function) and serum amylase. A complete blood count may reveal leucocytosis with neutrophilia. Serum amylase may help in the diagnosis of acute pancreatitis (but is raised in perforated DU).
- Urine; all patients with this presentation should be catheterized by a urinary catheter and hourly urine output is measured. A urine sample is take for urine analysis to check for urinary tract infections.
- **Radiology;** radiograph of the abdomen may confirm the presence of dilated gas-filled loops of bowel (consistent with a paralytic ileus), an *'erect'* film to demonstrate free air under the diaphragm ultrasound and computerised tomography (CT) scanning are increasingly used to identify the cause of peritonitis in *stable patients.*

TREATMENT

In case of doubt, *early surgical intervention* is the gold standard and is to be preferred to a 'wait and see' policy. This rule is particularly true for previously healthy patients and those with postoperative peritonitis. Caution is required in patients at high operative risk because of comorbidity or advanced age.

- **Correction of circulating volume and electrolyte** imbalance; Patients are frequently hypovolemic with electrolyte disturbances. The plasma volume must be restored and electrolyte concentrations corrected. Central venous catheterization and pressure monitoring may be helpful, particularly in patients with concurrent disease. Plasma protein depletion may also need correction as the inflamed peritoneum leaks large amounts of protein. If the patient's recovery is delayed for more than 7-10 days, intravenous nutrition is required.
- Gastrointestinal decompression; A nasogastric tube is passed into the stomach and aspirated. Intermittent aspiration is maintained until the paralytic ileus has resolved. Measured volumes of water are allowed by mouth when only small amounts are being aspirated. If the abdomen is soft and not tender, and bowel sounds return, oral feeding may be progressively introduced. It is important not to prolong the ileus by missing this stage.
- **Urinary catheterization;** This is important for hourly urinary output monitoring and should be inserted under complete aseptic conditions to prevent the high risk of septicaemia and bacteraemia.
- **Antibiotic therapy;** Administration of antibiotics prevents the multiplication of bacteria and the release of endotoxins. As the infection is usually a mixed one, initial treatment with parenteral broad-spectrum antibiotics active against aerobic and anaerobic bacteria should be given.
- Analgesia; when diagnosis is made, analgesia with opiates is given as soon as possible. Freedom from pain allows early mobilization and adequate physiotherapy in the postoperative period, which help to prevent basal pulmonary collapse, deep vein thrombosis and pulmonary embolism.
- Vital system support; patients might be given oxygen in high concentrations. Mechanical ventilation might be needed in respiratory failure.

Renal and cardiac status should be monitored by vital signs, CVP, urine output and regular KFTs and corrected accordingly (with diuretics and inotropes). An attending nephrologist, cardiologist and pulmonologist should be on call.

If the cause of peritonitis is amenable to surgery, operation must be carried out as soon as the patient is fit for anaesthesia. In operations for general peritonitis it is essential that, after the cause has been dealt with, the whole peritoneal cavity is explored with the sucker and, if necessary, mopped dry until all seropurulent exudate is removes. the use of a large volume of saline (1-2 litres) containing dissolved antibiotic (e.g. tetracycline) has been shown to be effective.

PROGNOSIS AND COMPLICATIONS

With modern treatment, diffuse peritonitis carries a mortality rate of about 10%. The systemic and local complications are shown in the table below

complications of peritonitis
Systemic
• **endotoxic shock**
• **Bronchopneumonia/respiratory failure**
• **Renal failure**
• **Bone marrow suppression**
• **Multisystem failure**
Local (abdominal}
• **Adhesional small bowel obstruction**
• **Paralytic ileus**
• **Residual or recurrent abscess**
• **Portal pyemia/liver abscess**

SUBPHRENIC ABSCESSES

Subphrenic abscess is the collection of pus in the subphrenic space. The symptoms and signs of subphrenic infection are frequently non-specific and it is well to remember the aphorism, *'pus somewhere, pus nowhere else, pus under the diaphragm'.*

CLINICAL FEATURES

Patients often complain of constitutional symptoms. There is sometimes epigastric fullness and pain, or pain in the shoulder on the affected side, because of irritation of sensory fibres in the phrenic nerve, referred along the descending branches of the cervical plexus. Persistent hiccoughs may be a presenting symptom.

A swinging pyrexia is usually present. If the abscess is anterior, abdominal examination will reveal some tenderness, rigidity or even a palpable swelling. Sometimes the liver is displaced downwards but more often it is fixed by adhesions. Examination of the chest is important and, in the majority of cases, collapse of the lung or evidence of basal effusion or even an empyema is found.

INVESTIGATIONS

A number of the following investigations may be helpful;

- Blood tests usually show a leukocytosis and raised C-reactive protein.
- A plain radiograph sometimes demonstrates the presence of gas or a pleural effusion. On screening, the diaphragm is often seen to be elevated (so called 'tented' diaphragm) and its movements impaired.
- Ultrasound or CT scanning is the investigation of choice and permits early detection of subphrenic collections.
- Radiolabelled white cell scanning may occasionally prove helpful when other imaging techniques have failed.

MANAGEMENT

If skilled help is available it is usually possible to insert a percutaneous drainage tube under ultrasound or CT control. The same tube can be used to instil antibiotic solutions or irrigate the abscess cavity.

SPECIAL TYPES OF PERITONITIS

- Postoperative; Following an anastomotic dehiscence, the general condition of a patient is usually more serious than if the patient had suffered leakage from a perforated peptic ulcer with no preceding operation. The patient is ill with raised pulse and peripheral circulatory failure. Local symptoms and signs are less definite. Abdominal pain may not be prominent and is often difficult to assess because of normal wound pain and postoperative analgesia. Usually most investigations are equivocal. The

principles of treatment do not differ from those of peritonitis of other origin. Antibiotic therapy alone is inadequate; it must be dealt with by re-exploration.

- In patients on treatment with steroids; Pain is frequently slight or absent. Physical signs are similarly vague and misleading (softish abdomen because muscles are week)
- In children; The diagnosis can be more difficult, particularly in the preschool child. History from the parents and quiet child examination is rewarding.
- In patients with dementia; Such patients can be -fractious and unable to give a reliable history. Abdominal tenderness is usually well localized, but guarding and rigidity are less marked because the abdominal muscles are often thin and weak.

BILE PERITONITIS

Unless there is reason to suspect that the biliary tract was damaged during operation, it is improbable that bile as a cause of peritonitis will be thought of until the abdomen has been opened. The common causes of bile peritonitis are shown in the table below.

Causes of bile peritonitis
• **Perforated cholecystitis**
• **Post cholecystectomy**
• **Following other operations/procedures**
• **Leaking duodenal stump post gastrectomy**
• **Following liver trauma**

there are signs of diffuse peritonitis. After a few hours. a tinge of jaundice is not unusual (due to absorption of bile). Laparotomy (or laparoscopy) should be undertaken with evacuation of the bile and peritoneal lavage.

MECONIUM PERITONITIS

Any congenital intestinal obstruction may be complicated by meconium peritonitis, an aseptic peritonitis, developing late in intrauterine life or immediately after birth as a result of intestinal perforation. Typically, the baby is born with a firm, distended, discolored abdomen and signs of intestinal obstruction. A plain abdominal radiograph may show dilated intestinal loops and areas of calcification in the liver and spleen. Occasionally, the cause of the intestinal perforation resolves spontaneously before birth but most neonates with meconium peritonitis will need early surgery. The prognosis in these patients is poor.

PNEUMOCOCCAL PERITONITIS

Primary pneumococcal peritonitis may complicate nephrotic syndrome or cirrhosis in children. Otherwise healthy children, particularly girls between 3 and 9 years of age, may also be affected, and it is likely that the route of infection is sometimes via the vagina and fallopian tubes. At other times, and always in males, the infection is blood-borne and secondary to respiratory tract or middle ear disease. The prevalence of pneumococcal peritonitis has declined greatly and the condition is now rare.

CLINICAL FEATURES

The onset is sudden and the earliest symptom is pain localized to the lower half of the abdomen. The temperature is raised to 39 °C. or more and there is usually frequent vomiting. After 24-48 hours, profuse diarrhea is characteristic. There is usually increased frequency of micturition. The last two symptoms are caused by severe pelvic peritonitis. On examination, abdominal rigidity is usually bilateral but is less than in most cases of acute appendicitis with peritonitis.

DIFFERENTIAL DIAGNOSIS

A leucocytosis of 30 000 /µl (30 x 109 /I) or more with approximately 90% polymorphs suggests pneumococcal peritonitis rather than appendicitis. Even so, it is often impossible to exclude perforated appendicitis. The other condition that can be difficult to differentiate from primary pneumococcal peritonitis in its early stage is *basal pneumonia.* An unduly high respiratory rate and the absence of abdominal rigidity are the most important signs supporting the diagnosis of pneumonia, which is usually confirmed by a chest radiograph.

TREATMENT

After starting antibiotic therapy and correcting dehydration and electrolyte imbalance, early surgery is required unless spontaneous infection of pre-existing ascites is strongly suspected, in which case a diagnostic peritoneal tap is useful. Laparotomy or laparoscopy may be used. Should the exudate be odourless and sticky, the diagnosis of pneumococcal peritonitis is practically certain, but it is essential to perform a careful exploration to exclude other pathology. Assuming that no other cause for the peritonitis is discovered, some of the exudate is aspirated and sent to the laboratory for microscopy, culture and sensitivity tests. Thorough peritoneal lavage is carried out and the incision closed.

IDIOPATHIC STREPTOCOCCAL AND STAPHYLOCOCCAL PERITONITIS IN ADULTS

idiopathic streptococcal and staphylococcal peritonitis in adults is fortunately rare. In streptococcal peritonitis, the peritoneal exudate is odourless and thin, contains some flecks of fibrin and may be bloodstained. In these circumstances pus is removed by suction, the abdomen closed with drainage and nonoperative treatment of peritonitis performed. The use of intravaginal tampons has led to an increased incidence of *Staphylococcus aureus* infections: these can be associated with 'toxic shock syndrome' and disseminated intravascular coagulopathy.

FAMILIAL MEDITERRANEAN FEVER (PERIODIC PERITONITIS)

Familial Mediterranean fever (periodic peritonitis) is characterized by abdominal pain and tenderness, mild pyrexia, polymorphonuclear leukocytosis and, occasionally, pain in the thorax and joints. The duration of an attack is 24-72 hours, when it is followed by complete remission, but exacerbations recur at regular intervals. Most of the patients have undergone appendectomy in childhood. This disease, often familial, is limited principally to Arab, Armenian and Jewish populations; other races are occasionally affected. Mutations in the *MEFV* (Mediterranean fever) gene appear to cause the disease. This gene produces a protein called pyrin, which is expressed mostly in neutrophils but whose exact function is not known. Usually, children are affected but it is not rare for the disease to make its first appearance in early adult life, with cases in women outnumbering those in men by two to one. Exceptionally, the disease becomes manifest in patients over 40 years of age. At operation, which may be necessary to exclude other causes but should be avoided if possible, the peritoneum - particularly in the vicinity of the spleen and the gall bladder - is inflamed. There is no evidence that the interior of these organs is abnormal.

Colchicine therapy is used during attacks and to prevent recurrent attacks.

ACUTE TUBERCULOUS PERITONITIS

Tuberculous peritonitis sometimes has an onset that so closely resembles acute peritonitis that the abdomen is opened. Strawcoloured fluid escapes and tubercles are seen scattered over the peritoneum and greater omentum. Early tubercles are greyish and translucent. They soon undergo caseation and appear white or yellow and are then less difficult to distinguish from carcinoma.

Occasionally, they appear like patchy fat necrosis. On opening the abdomen and finding tuberculous peritonitis, the fluid is evacuated, some being retained for bacteriological studies. A portion of the diseased omentum is removed for histological confirmation of the diagnosis and the wound closed without drainage.

CHRONIC TUBERCULOUS PERITONITIS

The condition presents with abdominal pain (90% of cases), fever (60%), loss of weight (60%), ascites (60%), night sweats (37%) and abdominal mass (26%).

infection originates from a tuberculous mesenteric lymph node, tuberculosis of the ileocaecal region, a tuberculous pyosalpinx or blood-borne infection from pulmonary tuberculosis, usually the 'miliary' but occasionally the 'cavitating' form.

STOMACH AND DUODENUM

ANATOMY

STOMACH

The stomach is an easily distensible viscus partly covered by the left costal margin. The greater and lesser curvatures correspond to the long and short borders of the stomach respectively, and the organ can be further divided anatomically into four distinct areas based on the microscopic mucosa! appearance: namely, the cardia, fundus, corpus (body) and antrum. The stomach is limited at its proximal end by the gastroesophageal junction just below the lower esophageal sphincter (It's not a true sphincter, It's a functional sphincter). Distally, the stomach is limited by the pylorus, a true anatomical sphincter. It is composed of greatly thickened inner circular muscle and helps to regulate the emptying of stomach contents into the duodenum.

DUODENUM

The duodenum is divided into four parts, which are closely applied to the head of the pancreas. The first part is approximately 5 cm in length.

The bulb is the first 1 cm of the first part of the duodenum. The second part has on its medial wall the ampulla of Vater. The third and fourth parts pass behind the transverse mesocolon into the infracolic compartment.

BLOOD SUPPLY

The stomach has an extensive blood supply derived from the celiac axis. The lesser curvature of the human stomach is supplied by the right gastric artery inferiorly and the left gastric artery superiorly, which also supplies the cardiac region. The greater curvature is supplied by branches from the splenic and gastroduodenal arteries, namely the right and left gastroepiploic artery. The fundus of the stomach, and also the upper portion of the greater curvature, is supplied by the short gastric arteries, which arise from the splenic artery.

Blood supply to the duodenum is derived from both the celiac axis (via the gastroduodenal artery which gives the superior pancreaticoduodenal artery) and branches from the superior mesenteric artery (inferior pancreaticoduodenal artery). The veins from the stomach and the duodenum accompany the arteries and drain into the portal venous system.

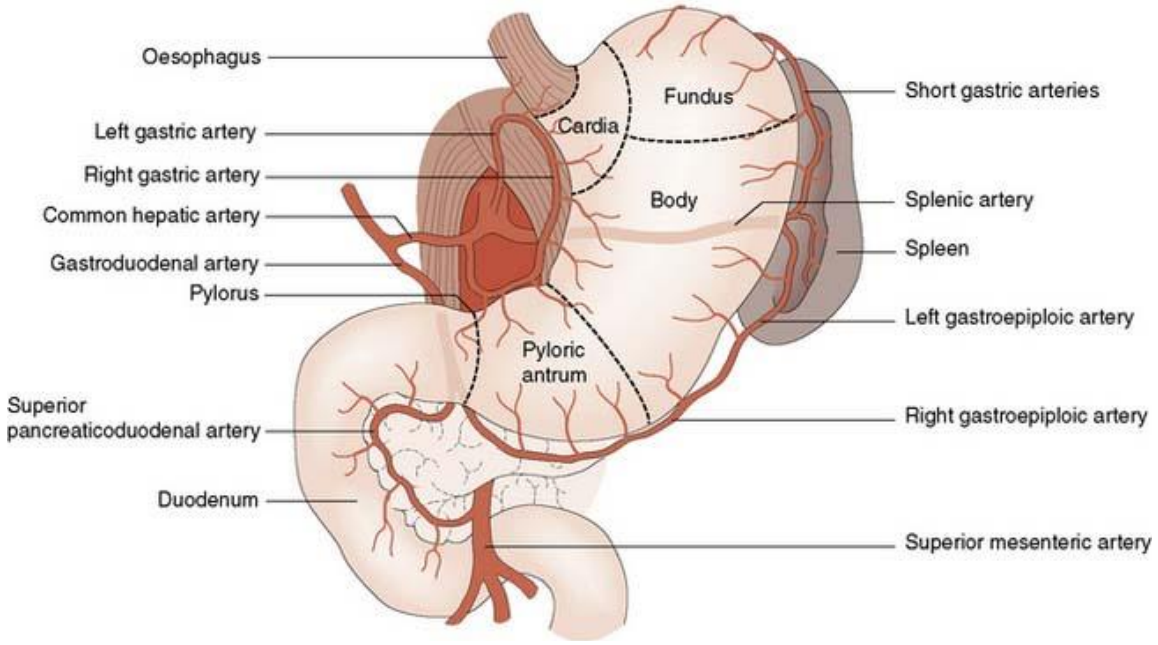


Figure 122 Anatomy, including arterial blood supply to the stomach and proximal duodenum

LYMPHATICS

The perigastric lymph nodes are found within the wall of the stomach. The paragastric lymph nodes are found in the lesser and greater curvatures and accompany the arteries; drainage is to nodes around these vessels, namely celiac and superior mesenteric, which are also known as the paraaortic lymph nodes.

NERVE SUPPLY OF THE STOMACH AND DUODENUM

The parasympathetic nerve supply to the stomach is derived from A-the **anterior** (Left) vagal trunk which gives two branches : Hepatic branch and The anterior nerve of latarjet.
B- **posterior** (Right) vagal trunks which gives celiac branch and the posterior nerve of latarjet.

The parasympathetic nerve supply of the stomach is responsible for the motor fibres to the stomach wall, inhibitory fibres to the pyloric sphincter and secretomotor fibres to the glands of the stomach. Sympathetic fibres accompany the gastric arteries from the celiac ganglion. These provide motor fibres to the pyloric sphincter.

The duodenum receives a sympathetic and parasympathetic supply from the celiac and superior mesenteric **plexuses.**

HISTOLOGY AND PHYSIOLOGY

Shallow depressions called gastric pits (figure 2-A) open onto the gastric surface, each gastric pit communicates with several gastric glands, which extend deep into the underlying lamina propria. The gastric pit is dominated by **mucus secreting cells.** The alkaline mucus (contains bicarbonate) is produced by all regions of the stomach and serves as a lubricant, and a protection against acid and digestive enzymes. The production of mucus is promoted by the action of local prostaglandins. The gastric glands are dominated by two types of cells; (1) parietal cells and (2) chief cells.

The parietal cells in the stomach are responsible for the production of acid. Acid secretion by these cells is stimulated by acetylcholine, released by the vagus nerve, and gastrin from the antrum (see below). Acetylcholine acts on neuroendocrine cells; **ECL (enterochromaffin like)** cells, located close to the parietal cells. On stimulation, these cells release histamine, which has a paracrine action on the parietal cell, stimulating acid production and secretion (figure 2). **Somatostatin, and secretin** inhibit acid secretion. Parietal cells also produce the **intrinsic** factor (necessary for B_{12} absorption)

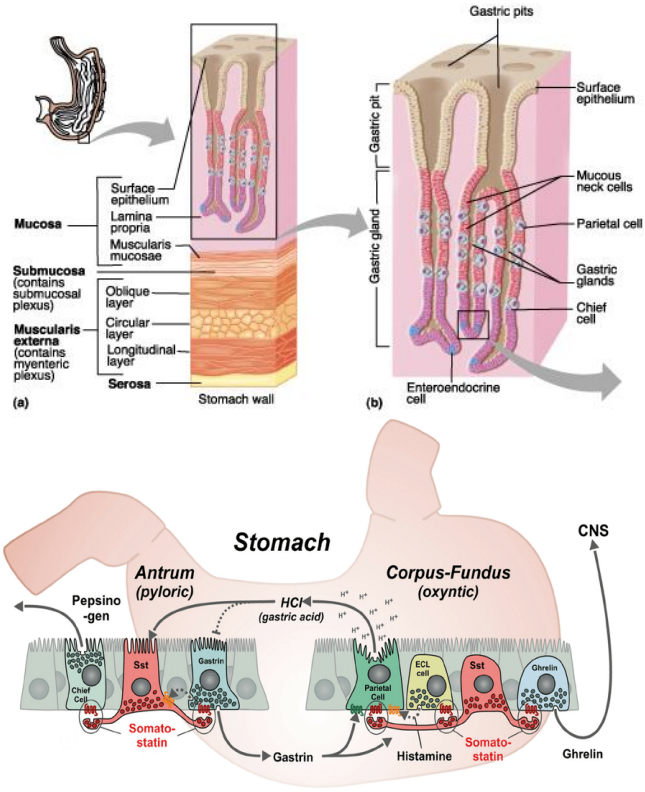


Figure 123 A-stomach lining and the gastric pit B-The parietal cell in relation to the mechanism of gastric acid secretion.

Pepsin is a proteolytic enzyme produced in its precursor form, **pepsinogen, by** the **chief cells,** Pepsinogen production in chief cells is stimulated by acetylcholine from the vagus nerve. The precursor is then converted to its active form, pepsin, by the acid contents of the stomach.

Gastrin is released by G cells in response to the presence of food in the stomach. The production of gastrin is inhibited by acid, hence creating a negative-feedback loop.

Classically, gastric secretion has been divided into three phases:

- Cephalic (neural) phase; Signals arise in the central cortex or appetite centers, triggered by

the sight, smell, taste and thought of food, and travel down the vagus nerves to the stomach.
- Gastric phase; Food (in particular protein digestion products) causes the release of acid, mainly through stimulation of G cells and the release of gastrin, during this face the acidification of the stomach stimulates the D-cells to release somatostatin which inhibits gastric acid secretion.
- Intestinal phase; The presence of undigested food in and the acidification of the duodenum triggers inhibits stomach acid secretion by release of secretin.

PEPTIC ULCER DISEASE

Peptic ulcer disease means the presence of mucosal ulcerations in the stomach or duodenum. It affects areas of mucosa exposed to acidic gastric contents.

The pathogenesis of peptic ulcer involves disturbance in the balance between the acid secretion and pepsin from the stomach and the mucosal barrier (A thick layer of mucus) on the other hand. The stimuli to the parietal cell for acid secretion are neural (Via the vagus nerve) and humoral (Gastrin and histamine) .

The normal stomach mucosa is adapted to contain the acid produced by the parietal cell . When the mucosal defense is compromised or absent, the acid causes musical ulceration. Ulcer also happens where acid attacks mucosa not specialized to deal with It like the duodenum; specially the first part.

GASTRIC ULCERATION

Gastric ulcer may be benign or malignant. Malignancy was once thought to be a complicating factor of benign gastric ulceration. It is now realized that malignant change in a benign ulcer is rare, and that *such ulcers are in fact probably malignant from the outset.* Gastric ulcers generally run a chronic course.

PATHOLOGY

Gastric ulcerations can take place in the following sites;

- Lesser curvature; accounts for more than 50-60% of gastric ulcers, and are located 2 cm from the incisura angularis. When these are found in the stomach they are single. But when associated with duodenal ulcers they are called multiple (accounts for 20-25%). Ulcers may occur close to the gastroesophageal junction.
- Pre pyloric (pyloric canal); These account for 20% of all gastric ulcers.
- Greater curvature; Giants ulcers on the greater curvature account for 5% of gastric ulcers, but are significant because they have a 12-15% incidence of malignancy.

ETIOLOGY

Although the exact etiology of gastric ulcers is obscure, there are many predisposing factors (see the table below). *Helicobacter pylori* is found in approximately 75% of cases of gastric ulcers, It infects the antrum region of the stomach. The role of NSAIDs as anti-inflammatory agents' centers on their inhibition of protective prostaglandin synthesis and are implicated in 30% of gastric ulcers.

Predisposing factors for gastric ulcers
• **Infection with *Helicobacter pylori***
• **Chronic use of NSAlDs**
• **Gastritis**
• **Gastric stasis**
• **Co-existing duodenal ulcer**
• **Duodenogastric reflux**
• **Increased acid/pepsin concentration**
• **Chronic alcohol use**
• **Smoking**
• **Steroid use**
• **Infection**

HELICOBACTER PYLORI:

It's a spiral shaped , Gram negative, motile rod that is able to penetrate the viscid mucus layer lining the stomach. Its potent urease activity splits any urea in the vicinity, producing ammonia and thus neutralizing the Ph in the local milieu surrounding the organism. Ingestion of h-pylori result in chronic gastritis.

Pathogenesis if H-pylori:

1-Increases fasting & postprandial gastrin

2-Increases pepsinogen secretion

3-Decreases gastric mucosal resistance

4-Suppresses somatostatin release

5-Releases tissue damaging cytotoxins that possess protease and phospholipase activity allowing them to attack and damage the mucosal barrier.

There's a Non-cytotoxic-producing strain of H-pylori which explain asymptomatic carriage of organism.

Ingestion of H-pylori result in chronic gastritis. long standing chronic gastritis leads to gastric atrophy & increased risk of metaplasia & the earlier H. pylori acquired, the greater is risk of Ca

DIAGNOSIS OF H-PYLORI:

1. Rapid Urease breath test: -
   - Used for screaming & to test for H. pylori eradication following treatment, expensive
2. Serology:-
   - Sensitive, specific & used for epidemiological surveys
3. Endoscopic antral biopsy for
   - Rapid Urease test (CLO)
   - Culture & sensitivity (Gold standard investigations)
   - Histological examination

TREATMENT OF H-PYLORI:

A-MEDICAL TREATMENT:

A 2-weeks course of antimicrobial therapy combined with acid reduction therapy will eradicate H-pylori. Acid reduction is usually afforded by a proton pump inhibitor (e.g.Omeprazole, lanzoprazole) and the antimicrobial therapy is based in either clarithromycin or amoxicillin, together with metronidazole. The combination of two antibiotic is recommended because of the high incidence of antibiotic resistance .

PPI is given for at least 1 month, while antibiotics should be at least given for 2 weeks. After 12 weeks re-endoscopy is used to see wither the ulcers have healed. If not, malignancy is suspected and further biopsies are taken.

Such protocol will eradicate H-pylori in 90% of patients

*Bisthmus is included in some regimens

B-SURGICAL TREATMENT:

Either Gastrectomy or vagotomy (Check Dr.Emad slides and record to study the types of gastrectomy and vagotomy they're easily explained.)Surgical treatment is Indicated in case of :Failure of medical treatment and Development of complications. The principle of the surgery is to reduce acid and pepsin secretion.

CLINICAL FEATURES OF GASTRIC ULCER

Patients with gastric ulcers most commonly presents in old ages (Forties to fifties) , it occurs predominantly in Men about 1:3 for men to women however this six preponderance for men is less marked than in duodenal ulcer as men is 80% more likely to have than women .

The patient will present with epigastric pain that is aggravated by food and relieved by vomiting. For this reasons these patients avoid food which can end in anorexia and cachexia. Reflux of acid can cause retrosternal pain. Patients may also presents with symptoms of upper gastrointestinal bleeding, including hematemesis (either coffee ground colored or fresh blood), or melena. If the bleeding is chronic, the patient may have signs and symptoms of anemia.

INVESTIGATIONS

1-The golden standard for diagnosing gastric ulcers is the use of fibro-optic gastroscopy, which also allows the gastroenterologist to obtain biopsies. This has replaced the use of barium studies as a mean of

diagnosis of gastric ulcers. The gastroenterologist should always take at least 12 biopsies if any ulcer is detected as there is a risk that these ulcers might be malignant. Biopsy may also allow detection of H.pylori (As mentioned before) by using the rapid urease test. In the latter, the biopsy specimen taken from the antrum is placed in a gel containing urea. Ammonia released by the action of the H. pylori-derived urease is detected and causes a color change-in most kits, from yellow to pink/red.

2- is the case of H-pylori (The other tests that were mentioned before)

3-Faceal occult blood examination is often positive is the presence of ulcer.

MANAGEMENT

Medical treatment has replaced the surgical treatment

In H. pylori-negative patients on NSAIDs, in whom the use of these drugs cannot be avoided altogether, the least damaging agents should be used (e.g. selective COX- 2 inhibitors), an antisecretory agent should be prescribed preferably selective histamine (H2) receptor antagonists.

SPECIAL FORMS OF GASTRIC ULCERS

Stress ulceration refers to erosions or ulceration of the stomach occurring in stress circumstances. Cushing's and Curling's ulcers are special forms of stress ulceration that occur following central nervous injury and burns, respectively. Curling's ulcer is caused by hypoperfusion of gastric mucosa secondary to plasma loss in extensive burns. Other forms of gastric ulcers may occur in erosive and hemorrhagic gastritis. These conditions are treated by proton pump inhibitors (PPI)

DUODENAL ULCERS

Duodenal ulcers are the other variety of peptic ulcer disease and imply ulcers in the duodenum.

At least 80% of peptic ulcers happen in the duodenum.

PATHOLOGY

Duodenal ulcers usually occur in the first part of the duodenum and 50% occur on the anterior wall. Of interest is that which affects the posterior wall of the bulb of the duodenum, as this is a common cause of massive upper gastrointestinal bleeding which occurs secondary to erosion of the gastroduodenal artery, as it passes at that site.

Postbulbar ulcers are commonly associated with zollinger Ellison syndrome caused by a gastrin-secreting tumor (gastrinoma) [Increase the secretion of gastrin which stimulates the parietal cells to secret more acid] , which is normally found in the pancreas but may occasionally be found in the duodenum or stomach. When there is involvement of both the anterior and the posterior wall of the bulb the ulcer is called a kissing ulcer.

ETIOLOGY AND PATHOGENESIS

The main pathogenic predisposing factor is exposure of the duodenal mucosa to increased amounts of gastric acid. This can be due to increased secretory capacity, or an abnormal emptying. The former may be caused by increased basal secretion, abnormal response to a meal, or increased levels of gastrin (e.g. zollinger Ellison syndrome). The association of H.pylori infection with duodenal ulcers is well established. H. pylori is detected in 95% of patients with duodenal ulceration. It infects the mucosa of the antrum of the stomach, where it causes an inflammatory response. This gastritis stimulates the gastrin-producing (G) cells of the antrum to increase gastrin production.

The use of NSAIDs may also be responsible for the small number of H. pylori-negative duodenal ulcers. Inhibition of prostaglandins synthesis by the use of NSAIDs can increase stomach output acidity and cause mucosal ulceration. Alcohol intake plays a role too and there is also an association between smoking and duodenal ulcerations.

Predisposing factors for duodenal ulcers

- **Infection with *Helicobacter pylori***
- **Chronic use of NSAIDs**
- **Smoking**
- **Zollinger-ellison syndrome**

CLINICAL FEATURES

As in gastric ulcers the patients most commonly present with epigastric pain. Unlike gastric ulcers and gall bladder diseases, the pain of duodenal ulcers is *relieved* by food. Patients with duodenal ulcer disease may try to eat to relief the pain, and on presentation they may be overweight, on the contrary to those with gastric ulcers, whom try to avoid food and thus are cachexic. Nausea, vomiting, and upper gastrointestinal bleeding are also associated with duodenal ulcers.

The pain is characterized by chronicity and periodicity. Chronicity is due to either ignorance of the condition or self-management by some educated individuals. Periodicity means disappearance of pain for weeks or months to return again. This periodicity may be related to the spontaneous healing of the ulcer.

INVESTIGATIONS

Fiberoptic upper endoscopy is used to diagnose duodenal ulcers, but unlike gastric ulcers, there is no need for obtaining a biopsy, since all duodenal ulcers are benign, barium meal are rarely used to diagnose duodenal ulcers. Detection of *H.pylori* is mainly by breath urea test.

MANAGEMENT

Eradication therapy is used for *H.pylori* positive patients. Healing is monitored by symptomatic improvement. Other agents that may be used to supplement antisecretory agents (PPI or H_2 antagonists) include bismuth compounds, sucralfate, prostaglandin analogues and antacids. Surgery is only indicated for complications. **Complications are either : bleeding, perforation, penetration or obstruction.**

BLEEDING PEPTIC ULCERS

Bleeding peptic ulcers are caused by erosion of an artery passing below the site of the ulcer. In the case of duodenal ulcers, the gastroduodenal artery is most commonly involved in posterior bulbar ulcers. Hemorrhage is the most serious complication and is associated with 40% mortality rate. Further discussion on the diagnosis and management of bleeding peptic ulcers is made in 'the upper gastrointestinal bleeding' seminar.

PERFORATION

The incidence of duodenal ulcer perforation is decreasing, probably due in part to improvements in the medical management of duodenal ulcers. Perforation usually occurs in acute ulcers on the anterior wall of the duodenum especially the bulb.

CLINICAL FEATURES

The presentation with perforation may be the first manifestation in 25% of patients with duodenal ulcers. Typically, the pain is sudden in onset and of extreme severity; indeed, the patient can often recall the exact moment of the onset of the pain. Subphrenic irritation may be indicated by referred pain in one or both shoulders usually the right one. The pain is aggravated by movement and the patient lies rigidly still. There's nausea but only occasionally vomiting. Sometimes, there's accompanying hematemesis or melaena.

Examination reveals a patient in severe pain, cold and sweating with rapid shallow respirations. In the early stages(hours), there may be no clinical evidence of true shock: The pulse is steady and the BP is normal; the temperature is either normal or a little depressed. The abdomen is rigid and silent, although in some instances an occasional bowel sound may be heard. Liver dullness is diminished in about half the cases owing to escape gas into the peritoneal cavity. Rectal examination may reveal pelvic tenderness.

In the delayed case, after 12 hours or more the features of generalized peritonitis with paralytic ileus become manifest; the abdomen is distended, effortless vomiting occurs and the patient is extremely toxic and is oligemic shock.

INVESTIGATIONS

Fiberoptic endoscopy and barium meal are contraindicated as they may cause leak of fluids (contrast or luminal fluids secondary to pneumatic pressure preceding endoscopy) into the peritoneal cavity, increasing the possibility of adhesions and intestinal obstruction after peritonitis. However, the use of water soluble contrast (gastrografin) is useful in showing leaks and the presence of a crater. In 80% of cases of perforation, an erect chest X-ray will

demonstrate free air under the diaphragm (figure 3), although the absence of free air does not exclude a perforation.

CT is more sensitive in the detection of free intra-peritoneal gas and can exclude common differential diagnosis such as pancreatitis when doubt exists

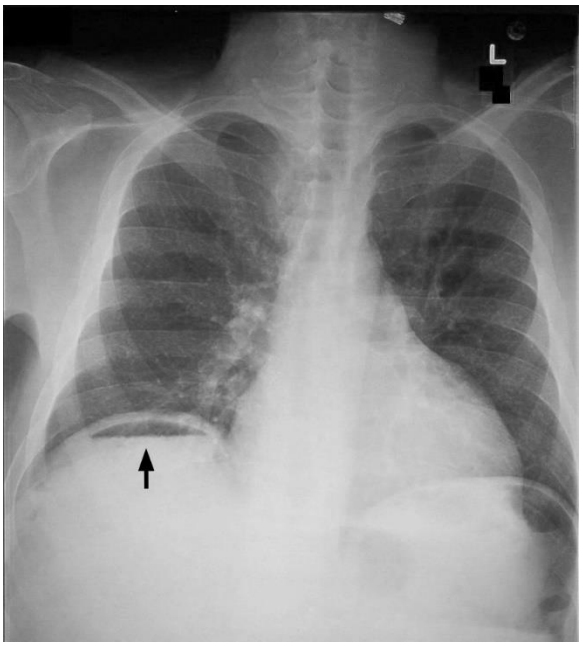


Figure 124 erect chest x-ray showing air under diaphragm (arrow).

MANAGEMENT

The initial management, as for other causes of peritonitis, consists of resuscitation, oxygen, intravenous fluids and antibiotics and the passage of a nasogastric tube. Adequate analgesia and antiemetics should be given as necessary, intravenous H2-blocker or PPI commenced. A urinary catheter enables close monitoring of urine output. In most patients, surgery is indicated. Surgery can be either open or laparoscopic. Surgery usually involves simple closure, whereby the ulcer is under-run with sutures or plugged using an omental patch (figure 4). A biopsy should always be taken in gastric ulcers.

DIFFERENTIAL DIAGNOSIS:

Perforated appendicitis, acute cholecystitis, acute pancreatitis, MI.

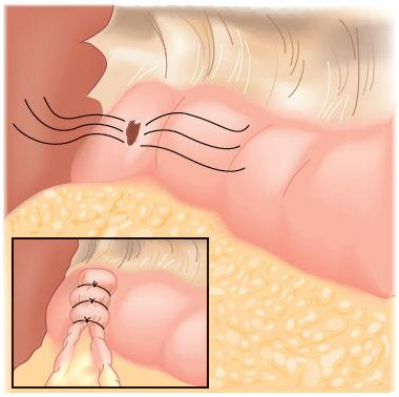


Figure 125 closure of a duodenal perforation with an omental patch.

OBSTRUCTION

Healing and fibrosis of prepyloric and duodenal ulcers may result in obstruction and cause signs and symptoms of gastric outlet obstruction, including *non-bilious* vomiting that becomes increasingly *projectile*. The most prominent feature on examination is the presence of a succession splash (i.e. an audible splashing noise when the patient is gently rocked from side to side).

Patients are often hypokalemic with metabolic alkalosis, and may show the presence of a glass hour appearance on barium meal. When gastric outlet obstruction is suspected, a large-bore nasogastric tube is passed. Often, large volumes of non-bilious gastric contents can be aspirated and undigested food may be recognized. After resuscitation definitive treatment include endoscopic balloon dilatation.

	Gastric ulcer	Duodenal Ulcer
Relived by	Lying down or vomiting	Eating
Duration	Few weeks	1-2 months
Vomiting	Common (to relieve the pain)	Uncommon
Appetite	Pt. afraid to eat	Good
Diet	Avoid fried food	Good , eat to relieve the pain
Weight	wt. Loss	No wt. loss

GASTRIC CARCINOMA

Adenocarcinoma is the most common malignancy affecting the stomach. It accounts for 9096 of malignant tumors found within the stomach, lymphomas, carcinoids and gastrointestinal stromal tumors filling the rest.

A-Common Primary

- Adenocarcinoma (95%), it arises from columnar glandular epithelium.
- Lymphoma (4%),
- Malignant GIST (1%)

B-Rare Primary

- Carcinoid (Arises from the G-cells) , Angiosarcoma, Carcinosarcoma, and Squamous cell carcinoma

C-Secondary From:

- Melanoma , Breast(Blood born)
- Colon or Pancreas (Direct ext.)
- Ovary (By peritoneal seeding)

ETIOLOGY AND EPIDEMIOLOGY

The disease is more common in japan when compared to the united states and Europe. Risk factors for developing gastric cancer are shown below.

Risk factors for developing gastric cancer

- **Infection with *Helicobacter pylori***
- **Pernicious anemia**
- **Gastric polyps**
- **Gastric surgery**
- **Gastroduodenal reflux**
- **Smoking**
- **Diet (spirits, N-nitroso compounds, excessive salt intake)**

Risk factors for developing Gastric cancer:

A-**Predisposing Factors**: Smoking, Alcohol, previous pernicious anemia, Atrophic gastritis (Achlorhydria) , Gastric resection , obesity , gastric polyp ,gastroduodenal reflux, Ménétrier's disease.

B-**Environmental factors:** H-plylori infection, low socioeconomic state, Diet Rich (in pickled vegetables, salted fish , excessive dietary salt ,smocked meat, high nitrates)

C-**Genetic**: Family history, Blood group A , HNPCC(Hereditary non-polyposis colon cancer)

CLINICAL PRESENTATION

1-Asymptomatic

2-Early: Vague epigastric discomfort / indigestion . Pain is constant, nonradiating, unrelieved by food digestion

3-More advanced: Weight loss,anorexia,fatigue, emesis

4-Symptoms dependent on location: Proximal, distal, diffuse**.**

5-GI bleeding, obstruction

SIGNS

Patients usually are pale from chronic loss of blood and may have other signs of anemia. Non-metastatic effects of malignancy are seen, particularly migratory thrombophlebitis (Trousseau's sign) and deep venous thrombosis. Patients may have signs of gastric outlet obstruction.

Metastatic lymph nodes may be palpable, most notably in the left supraclavicular fossa (Virchow's node, Troisier's sign). The Left anterior axillary lymph node, is another site often involved by metastatic gastric disease (Irish nodes). Metastasis to the liver may present with Jaundice and a Palpable liver mass. Ascites may result from portal hypertension. A blummer's shelf' can be felt on digital rectal examination when there is transcelomic spread to the anterior wall of the rectum. the Sister Mary Joseph nodule refers to a palpable nodule bulging into the umbilicus as a result of metastasis of gastric cancer in the pelvis or abdomen.Palpable ovarian mass (Krukenberg's tumor).

CLASSIFICATION OF GASTRIC CANCER

There're two types of classification: Bormann's classification and Lauren's classification

BORMANN'S CLASSIFICATION

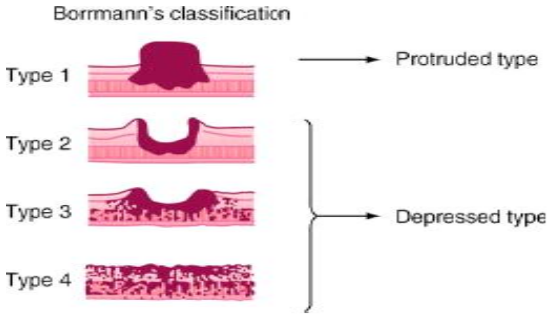
Gross classification into

Polypoid type (type 1)

ulcerative type (type 2)

infiltrative ulcerative (type 3)

diffuse infiltrative type (type 4)



LAUREN'S SYSTEM

It classified gastric carcinoma to diffuse and intestinal type:

Intestinal	Diffuse
• **Environmental** • **Gastric atrophy, intestinal metaplasia** • **Men > women** • **Increasing inc. w/ age** • **Gland formation** • **Hematogenous Spread** • **Microsatellite instability** • **APC gene mutations** • ***p53, p16* inactivation** • ***APC*, adenomatous polyposis coli** • **More distal and localized** • **Better prognosis 5 yr surgery overall 20%**	• Women > men • Younger age group • Poorly differentiated, signet ring cells • Transmural / lymphatic spread • Decreased E-cadherin • *p53, p16* inactivation • Poorer prognosis 5 yr surgery overall <10% • More proximal

Spread patterns of gastric cancer:

Direct invasion, Lymph node dissemination, Blood spread, Transperitoneal colonization

DIAGNOSIS

A full blood count may show anemia due to obvious or occult bleeding. The carcinoembroyonic antigen (CAE) is detected in the blood of these patients. The diagnosis of gastric carcinoma is made by fiber-optic endoscopy which has replaced the use of barium meal. A biopsy is taken for grading of the tumor. Staging is done by CT scan or MRI. the staging of gastric carcinoma is shown below , it mainly depends on the Lymphatic involvement.

MANAGEMENT

If there is no metastasis, surgery is indicated and is curative and involves total or near-total gastrectomy. When there is metastasis, the role of surgery become limited to. palliation and involves the relief of obstruction by gastrojejunostomy and suturing of ulcers that cause bleeding in addition to the use of chemotherapy or radiotherapy. Analgesia constitute an important part of the management. celiac block can be used to relieve the pain to avoid dependence on opiates.

Stages of gastric cancer according to TNM classification	
Tis	Carcinoma in situ; Intraepithelial tumor without invasion of lamina propria
T1	Tumor invades lamina propria or submucosa
T2	Tumor invades muscularis externa or subserosa
T3	Tumor penetrates serosa (visceral peritoneum) without invasion of adjacent structures
T4	Tumor invades adjacent structures
N0	No regional lymph node metastasis
N1	Metastasis in 1 to 6 regional lymph nodes
N2	Metastasis in 7 to 15 lymph nodes
N3	Metastasis in more than 15 regional lymph nodes
M0	No distant metastasis
M1	Distant metastasis
Stage	TNM
IA	$T_1N_0M_0$
IB	$T_1N_1M_0$, $T_2N_0M_0$
II	$T_1N_2M_0$, $T_2N_1M_0$
IIIA	$T_2N_2M_o$, $T_3N_1M_0$, $T_4N_0M_0$
IIIB	$T_3N_2M_0$
IV	$T_4N_{1-3}M_o$,$T_{1-3}N_3M_0$ Any T,N with M_2

THE COMPLICATIONS OF PEPTIC ULCERATION

The common complications of peptic ulcer are perforation, bleeding and stenosis.

PERFORATED PEPTIC ULCER

A perforated ulcer is a condition in which an untreated ulcer can burn/burst through the wall of the stomach (or other areas of the GI tract), allowing digestive juices and food to leak into the abdominal cavity.

It is the second most common complication of peptic ulcer but nowadays a more common indication for operation than bleeding

EPIDEMIOLOGY

Despite the widespread use of gastric antisecretory agents and eradication therapy, the incidence of perforated peptic ulcer has changed little. However, there has been a considerable change in the epidemiology of perforated peptic ulcer in resource-rich countries over the last two decades. Previously, most patients were middle aged, with a ratio of 2:1 of male:female. With time there has been a steady increase in the age of the patients suffering this complication and an increase in the numbers of females, such that perforations now occur most commonly in elderly female patients. NSAIDs appear to be responsible for most of these perforations.

perforated gastric ulcers have much higher mortality than perforated duodenal ulcers

the most common site of perforation is the anterior aspect of the duodenum

CLINICAL FEATURES

Classically, the abdominal pain caused by a peptic perforation develops very suddenly in the upper abdomen. Most patients can accurately time the dramatic onset of symptoms. The natural history of such an episode can be divided into three phases:

1) Chemical peritonitis/contamination.

Initially, the perforation leads to chemical peritonitis, with or without contamination with micro-organisms. (Note that the presence of acid sterilizes gastroduodenal contents; it is when gastric acid is reduced by treatment or disease (e.g. gastric cancer) that bacteria and fungi are present in the stomach and duodenum). Spillage of gastroduodenal contents is usually diffuse but may be localized in the upper abdomen by adhesions or the omentum. Spillage along the right gutter into the right lower quadrant, mimicking acute appendicitis (valentino's sign)

- General signs: pale, sweating, subnormal temperature and rapid weak pulse
- Local signs:

A) Board like rigidity, guarding, epigastric tenderness

B) Decreased liver dullness (air under diaphragm)

C) Shifting dullness

D) Decreased bowel sound due to paralytic ileus (late sign)

2) Intermediate stage. (stage of illusion)

After 6 to 12 hours many patients obtain some spontaneous relief of the pain. This is probably due to the dilution of the irritating gastroduodenal contents by the ensuing peritoneal exudate (production of large amount of alkaline fluid and bringing antibodies)

3) Intra-abdominal infection (septic peritonitis)

after 12 to 24 hours intra-abdominal infection supervenes. The exact point in time in the individual patient when contaminating micro-organisms become invasive-infective, is unknown. Therefore, you should consider any perforation operated upon with a delay of more than 12 hours as infection rather than contamination. The pain usually increases with fever, anorexia , headache ,repeated vomiting and distension .

on examination the patients usually have fever, tachycardia , and toxemia

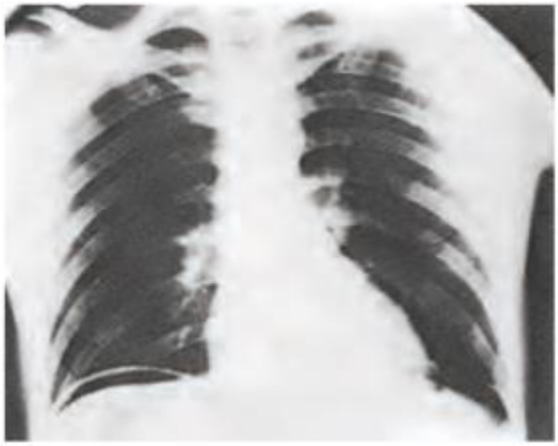
the abdomen shows generalized rigidity and tenderness.

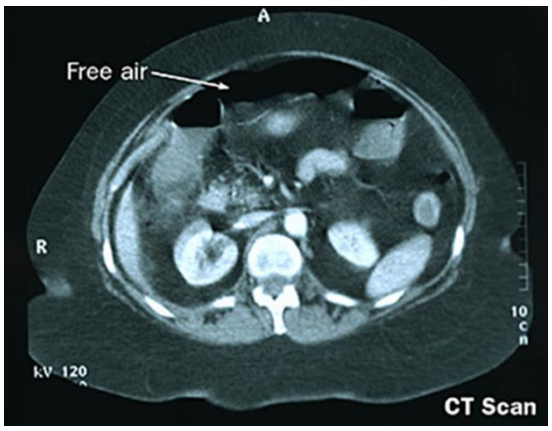
Neglected patients may present a few days after the perforation in septic shock. . Untreated perforation can lead eventually to an early "septic "death from peritonitis or the development of an intra-abdominal abscess.

Elderly patients who is taking NSAIDs will have a less dramatic presentation, perhaps because of the use of those potent anti-inflammatory drugs, the board-like rigidity seen in the abdomen of younger patients may also not observed in them.

INVESTIGATIONS

An erect plain chest radiograph will reveal free gas under the diaphragm in excess of 50% of cases with perforated peptic ulcer (Figure) but CT imaging is more accurate (see below).(absence of free air does not exclude a perforation)





All patients should have serum amylase performed, as distinguishing between peptic ulcer, perforation and pancreatitis can be difficult. Measuring the serum amylase, however, may not remove the diagnostic difficulty. It can be elevated following perforation of a peptic ulcer although, fortunately, the levels are not usually as high as the levels commonly seen in acute pancreatitis. Several other investigations are useful if doubt remains. A CT scan will normally be diagnostic in both conditions .US may show fluid in peritoneum

Fibreoptic endoscopy and barium meal are contraindicated as they may cause leak of fluids (contrast or luminal fluids secondary to pneumatic pressure preceding endoscopy) into the peritoneal cavity, increasing the possibility of adhesions and intestinal obstruction after peritonitis. Gastrografin meal can be used to assess the need for surgery.

TREATMENT

The initial priorities are resuscitation and analgesia. Analgesia should not be withheld for fear of removing the signs of an intra-abdominal catastrophe. In fact, adequate analgesia makes the clinical signs more obvious. It is important, however, to titrate the analgesic dose. Give oxygen , IV fluid and antibiotics , antiemetics and passage of nasogastric tube. A urinary catheter enables close monitoring of urinary output. Following resuscitation, the treatment is principally surgical. Laparotomy is performed, usually through an upper midline incision if the diagnosis of perforated peptic ulcer can be made with confidence. This is not always possible and hence it may be better to place a small incision around the umbilicus to localise the perforation with more certainty. Alternatively, laparoscopy may be used. The most important component of the operation is a thorough peritoneal toilet to remove all of the fluid and food debris. If the perforation is in the duodenum it can usually be closed by several well-placed sutures, closing the ulcer in a transverse direction as with a pyloroplasty. It is important that sufficient tissue is taken in the suture to allow the edges to be approximated, and the sutures should not be tied so tight that they tear out. It is common to place an omental patch over the perforation in the hope of enhancing the chances of the leak sealing. If the perforation is difficult to close primarily it is frequently possible to seal the leak with an omental patch alone, and many

surgeons now employ this strategy for all perforations. When securing the omental patch it is important not to tie the sutures too tight so as to obliterate the omental blood supply. Gastric ulcers should, if possible, be excised and closed, so that malignancy can be excluded. Occasionally a patient is seen who has a massive duodenal or gastric perforation such that simple closure is impossible; in these patients a distal gastrectomy with Roux-en-Y reconstruction is the procedure of choice. All patients should be treated with systemic antibiotics in addition to a thorough peritoneal lavage. In the past, many surgeons performed definitive procedures such as either truncal vagotomy and pyloroplasty or, more recently and probably more successfully, highly selective vagotomy during the course of an operation for a perforation. Studies show that in well-selected patients and in expert hands this is a very safe strategy. However, nowadays, surgery is confined to first-aid measures most commonly, and the peptic ulcer is treated medically as described earlier in this chapter. Following operation, gastric antisecretory agents should be started immediately. H. pylori eradication is mandatory. Perforated peptic ulcers can often be managed by minimally invasive techniques if the expertise is available. The principles of operation are, however, the same; thorough peritoneal toilet is performed and the perforation is closed by intracorporeal suturing. Whatever technique is used, it is important that the stomach is kept empty postoperatively by nasogastric suction, and that gastric antisecretory agents are commenced to promote healing in the residual ulcer.

A great deal has been written about the conservative management of perforated ulcer. Some writers say that virtually all patients can be managed conservatively, whereas most surgeons have difficulty in understanding how a patient who is ill with widespread peritonitis and who has food debris widely distributed through the abdominal cavity will improve without an operation. However, undoubtedly, there are patients who have small leaks from a perforated peptic ulcer and relatively mild peritoneal contamination, who may be managed with intravenous fluids, nasogastric suction and antibiotics. These patients are in the minority. A number of factors have been associated with poor outcome after perforated peptic ulcer, including:

- delay in diagnosis (>24 hours);
- medical comorbidities;
- shock;
- increasing age (>75).

There is little evidence to advocate the conservative management of patients who exhibit any of these characteristics.

Patients who have suffered one perforation may suffer another one. Therefore, they should be managed aggressively to ensure that this does not happen. Lifelong treatment with proton pump inhibitors is a reasonable option especially in those who have to continue with NSAID treatment.

BLEEDING PEPTIC ULCERS

Internal bleeding is the most common complication of peptic ulcers. In recent years, the population affected has become much older and the bleeding is commonly associated with the ingestion of NSAIDs. Diagnosis can normally be made endoscopically, although occasionally the nature of the blood loss precludes accurately identifying the lesion. However, the more experienced the endoscopist, the less likely this is to be a problem.

DUODENAL ULCERS:

The most common site of bleeding from a peptic ulcer is the duodenum. The ulcers causing the bleeding are usually found in the posterior or superior aspect of the first part of duodenum .the gastroduodenal artery, which is commonly the source of major bleeding.

The situation in which there is both an anterior and a posterior duodenal ulcer is referred to as ‘kissing ulcers’.

Anteriorly placed ulcers tend to perforate and, in contrast, posterior duodenal

ulcers tend to bleed, sometimes by eroding into the gastroduodenal artery.

GASTRIC ULCERS:

Large chronic ulcers may erode posteriorly into the pancreas and, on other occasions, into major vessels such as the splenic artery.

MEDICAL AND MINIMALLY INTERVENTIONAL TREATMENTS

Medical treatment has limited efficacy. All patients are commonly started on either an H2-antagonist or a proton pump antagonist, and recent evidence confirms the benefit of proton pump inhibitor administration to prevent rebleeding after endoscopy. Furthermore, meta-analysis of studies suggests that tranexamic acid, an inhibitor of fibrinolysis, may reduce overall mortality.

Therapeutic endoscopy can achieve haemostasis in approximately 70% of cases, with the best evidence supporting a combination of adrenaline injection with heater probe and/or clips. Therapeutic endoscopy will probably never be effective in patients who are bleeding from large vessels and with which the majority of the mortality is associated.

In patients where the source of bleeding cannot be identified or in those who rebleed after endoscopy, angiography with transcatheter embolization may offer a valuable alternative to surgery in expert centres. The risk of significant ischemia following embolization is low because of the rich collateral blood supply of the stomach and duodenum. The surgeon should be mindful that rescue surgery after failed embolization is associated with poor outcome and it may be advantageous to proceed directly to surgery.

SURGICAL TREATMENT

Criteria for surgery are well worked out. A patient who continues to bleed requires surgical treatment. The same applies to a significant rebleed. The only exception applies in expert centers with 24-hour interventional radiology and experience of angiographic embolization where attempts may be made to arrest bleeding and avoid surgery. The surgical team should care for these patients and an operation should not be delayed if any concerns remain. Patients with a visible vessel in the ulcer base, a spurting vessel or an ulcer with a clot in the base are statistically likely to require surgical treatment to stop the bleeding. Elderly and unfit patients are more likely to die as a result of bleeding than younger patients. Ironically, they should have early surgery. A patient who has required more than six units of blood in general needs surgical treatment.

The aim of the operation is to stop the bleeding. The advent of endoscopy has greatly helped in the management of upper gastrointestinal bleeding as a surgeon can usually be confident about the site of bleeding prior to operation. The most common site of bleeding from a peptic ulcer is the duodenum. In tackling this, it is essential that the duodenum is fully mobilized. This should be done before the duodenum is opened as it makes the ulcer much more accessible and also allows the surgeon's hand to be placed behind the gastroduodenal artery, which is commonly the source of major bleeding. Following mobilization, the duodenum, and usually the pylorus, is opened longitudinally as in a pyloroplasty. This allows good access to the ulcer, which is usually found posteriorly or superiorly. Accurate haemostasis is important and can be achieved initially by direct pressure. It is the vessel within the ulcer that is bleeding and this should be controlled using well-placed sutures on a small round-bodied needle that under-run the vessel. The placing of more and more inaccurately positioned sutures is counterproductive. Following under-running, it is often possible to close the mucosa over the ulcer. The pyloroplasty is then closed with interrupted sutures in a transverse direction as in the usual fashion. In a giant ulcer the first part of the duodenum may be destroyed making primary closure impossible. In this circumstance one should proceed to distal gastrectomy with Roux-en-Y reconstruction. The duodenal stump may then be closed using the Nissen technique with T-tube drainage.

The principles of management of bleeding gastric ulcers are essentially the same. The stomach is opened at an appropriate position anteriorly and the vessel in the ulcer under-run. If the ulcer is not excised then a biopsy of the edge needs to be taken to exclude malignant transformation. Sometimes the bleeding is from the splenic artery and if there is a lot of fibrosis present then the operation may be challenging. However, most patients can be managed by conservative surgery. Gastrectomy for bleeding has been widely practiced in the past, but is associated with a high perioperative mortality even if the incidence of recurrent bleeding is less.

Bearing in mind that most patients nowadays are elderly and unfit, the minimum surgery that stops the bleeding is probably optimal (damage control surgery). Acid can be inhibited by pharmacological means and appropriate eradication therapy will

prevent ulcer recurrence. Definitive acid-lowering surgery is not now required. Patients on long-term NSAIDs can be managed as outlined earlier.

GASTRIC OUTLET OBSTRUCTION

The two common causes of gastric outlet obstruction are gastric cancer and pyloric stenosis secondary to peptic ulceration. Previously, the latter was more common. Now, with the decrease in the incidence of peptic ulceration and the advent of potent medical treatments, gastric outlet obstruction should be considered malignant until proven otherwise, at least in resource-rich countries. The term 'pyloric stenosis' is normally a misnomer. The stenosis is seldom at the pylorus. Commonly, when the condition is due to underlying peptic ulcer disease, the stenosis is found in the first part of the duodenum, the most common site for a peptic ulcer. True pyloric stenosis can occur due to fibrosis around a pyloric channel ulcer. However, in recent years the most common cause of gastric outlet obstruction has been gastric cancer. In this circumstance the metabolic consequences may be somewhat different from those of benign pyloric stenosis because of the relative hypochlorhydria found in patients with gastric cancer

CLINICAL FEATURES

In benign gastric outlet obstruction there is usually a long history of peptic ulcer disease. Nowadays, as most patients with peptic ulcer symptoms are treated medically, it is easy to understand why the condition is becoming much less common. In some patients the pain may become unremitting and in other cases it may largely disappear. The vomitus is characteristically unpleasant in nature and is totally lacking in bile. Very often it is possible to recognize foodstuff taken several days previously. The patient commonly complains of losing weight, and appears unwell and dehydrated. When examining the patient, it may be possible to see the distended stomach and a succession splash may be audible on shaking the patient's abdomen.

METABOLIC EFFECTS

These are most interesting, as the metabolic consequences of benign pyloric stenosis are unique. The vomiting of hydrochloric acid results in hypochloraemic alkalosis. Initially the sodium and potassium may be relatively normal. However, as dehydration progresses, more profound metabolic abnormalities arise, partly related to renal dysfunction. Initially, the urine has a low chloride and high bicarbonate content, reflecting the primary metabolic abnormality. This bicarbonate is excreted along with sodium, and so with time the patient becomes progressively hyponatraemic and more profoundly dehydrated. Because of the dehydration, a phase of sodium retention follows and potassium and hydrogen are excreted in preference. This results in the urine becoming paradoxically acidic and hypokalaemia ensues. Alkalosis leads to a lowering in the circulating ionized calcium, and tetany can occur.

Hypochloremia occur due to HCL loss by repeated vomiting

MANAGEMENT

Treating the patient involves correcting the metabolic abnormality and dealing with the mechanical problem. The patient should be rehydrated with intravenous isotonic saline with potassium supplementation. Replacing the sodium chloride and water allows the kidney to correct the acid–base abnormality. Following rehydration, it may become obvious that the patient is also anaemic, the haemoglobin being spuriously high on presentation. It is notable that the metabolic abnormalities may be less if the obstruction is due to malignancy, as the acid–base disturbance is less pronounced. The stomach should be emptied using a wide-bore gastric tube. A large nasogastric tube may not be sufficiently large to deal with the contents of the stomach, and it may be necessary to pass an orogastric tube and lavage the stomach until it is completely emptied. This then allows investigation of the patient with endoscopy and contrast radiology.

Barium meal:

- Dilated stomach.
- Soup Dish appearances or inverted hat appearance (Fluid level + rugae)
- Delayed gastric emptying (Film taken 12-24 hrs. later)

Endoscopy and Biopsy of the area around the pylorus is essential to exclude malignancy. The patient should also have a gastric antisecretory agent,

initially given intravenously to ensure absorption. Early cases may settle with conservative treatment, presumably as the oedema around the ulcer diminishes as the ulcer is healed.

Traditionally, severe cases are treated surgically, usually with a gastroenterostomy rather than a pyloroplasty. Endoscopic balloon dilation can often transiently improve obstructive symptoms, but many of these patients ultimately fail and come to operation. However, this treatment is not devoid of problems. Dilating the duodenal stenosis may result in perforation. The dilatation may have to be performed several times and may not be successful in the long term. Occasionally duodenal stent insertion will be considered in specialist centres.

The standard operation for obstructing PUD is vagotomy and antrectomy. Alternatively vagotomy and gastrojejunostomy should be considered if a difficult duodenal stump is anticipated with resection. HSV and gastrojejunostomy may be comparable to V+A for obstructing ulcer disease,76 and sometimes has appeal because it can be done laparoscopically, and because it does not complicate future resection, if needed. However, potentially curable gastric or duodenal cancers can be missed with this approach.

THE LIVER

ANATOMY (FIGURE 1)

the liver is the largest abdominal organ and is located beneath the right hemidiaphragm. Its triangular in shape. The liver is attached to the under surface of the diaphragm by suspensory ligaments that enclose a 'bare area', the only part of its surface without a peritoneal covering. Grossly, on the anterior surface, the falciform ligament separates the liver into two lobes; right and left. On the posterior surface of the liver, the impression left by the inferior vena cava marks the division between the right lobe and the small caudate lobe. Inferior to the caudate lobe lies the quadrate lobe, sandwiched between the left lobe and the gallbladder.

From a practical standpoint, it is the segmental anatomy of the liver, as defined by the distribution of its blood supply that is important to the surgeon. The portal vein and hepatic artery divide into right and left branches in the porta hepatis (the hepatic doorway). Occluding either branch at surgery produces an easily visible line of demarcation that runs from the gallbladder bed behind and to the left of the inferior vena cava, thus separating the two functional lobes (called hemilivers).

Each hemiliver is further divided into four segments corresponding to the main branches of the hepatic artery and portal vein. In the left hemiliver, segment I corresponds to the caudate lobe, segments II and III to the left lobe, and segment IV to the quadrate lobe. The remaining segments (V-VIII) comprise the right hemiliver.

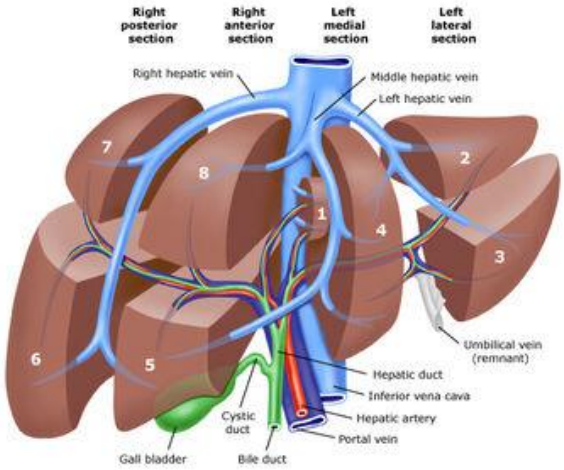


Figure 127 segmental anatomy of the liver

BLOOD SUPPLY

The liver normally receives 100-130 ml of blood per minute per Kg body weight and has a dual blood supply, 75% coming from the portal vein and 25% from the hepatic artery (which arises from the celiac artery), which supplies 50% of the oxygen requirements and 75% of nutrients. The principal venous drainage of the liver is by the right, middle and left hepatic veins, which enter the inferior vena cava.

the hepatic portal system connects the capillaries of the gastrointestinal tract with the capillaries in the liver. Nutrient-rich blood leaves the gastrointestinal tract and is first brought to the liver for processing before being sent to the heart.

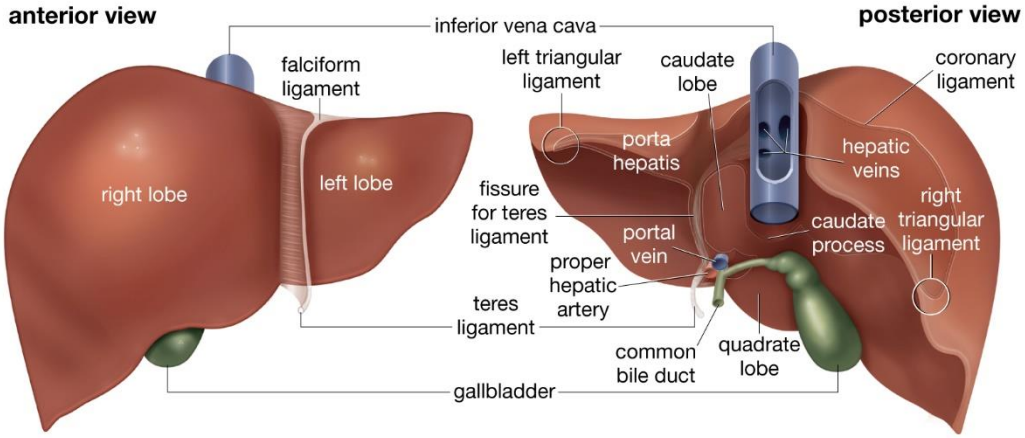


Figure 126 gross anatomy of the liver

HISTOLOGY

The functional unit of the liver is the hepatic acinus (figure 3). Sheets of liver cells (hepatocytes) one cell thick are separated by interlacing sinusoids through which blood flows from the peripheral portal tract into the hepatic acinus to the central branch of the' hepatic venous system. Bile is secreted by the liver cells and passes in the opposite direction along the small canaliculi into interlobular bile ducts located in the portal tracts.

In addition to containing typical endothelial cells, the sinusoidal lining includes a large number of Kupffer cells which are part of the monocyte-macrophage system.

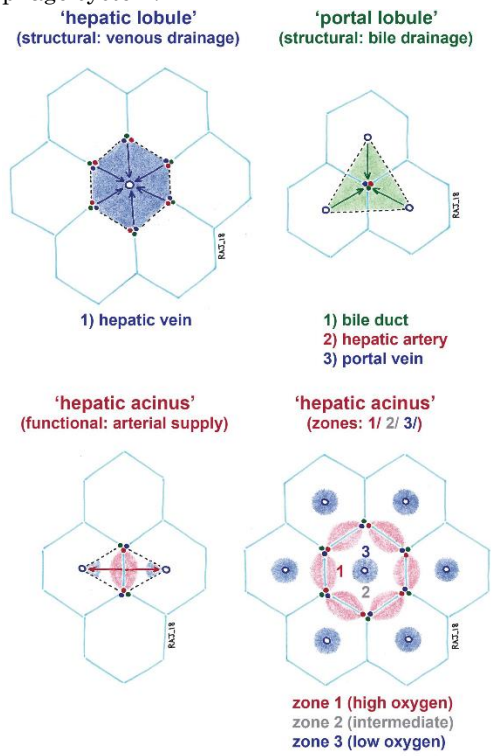


Figure 128 histology of the liver

PHYSIOLOGY

BILE PRODUCTION

Bile is an important fluid as it helps excrete material not excreted by the kidneys and aids in the absorption and digestion of lipids via secretion of bile salts and acids. Bile is produced by hepatocytes and is mainly composed of water, electrolytes, bile salts, bile acids, cholesterol, bile pigment, bilirubin, and phospholipids in addition to other substances. Bile is secreted from hepatocytes into the bile canaliculi where it travels from smaller ducts to the larger ducts eventually ending up in the duodenum or being stored in the gallbladder for storage and concentration as determined by the duct and sphincter of Oddi pressures. Following secretion of bile into the duodenum, it undergoes enterohepatic circulation, where it performs its job in the bowel and bile components that are not excreted are recycled by conversion into bile acids by gut bacteria for reuse by absorption in the ileum and transport back to the liver.

FAT-SOLUBLE VITAMIN STORAGE AND/OR METABOLISM

Most fat-soluble vitamins reach the liver via intestinal absorption in the form of chylomicrons or VLDL.

BILIRUBIN METABOLISM

The liver plays a significant role in the breakdown of heme. Hemolysis takes place in multiple locations throughout the body, including the liver, spleen, and bone marrow.

LIVER ABSCESS

mass filled with pus inside the liver

Liver abscesses can be classified

- Pyogenic liver abscess, which is most often polymicrobial, accounts for 80% of hepatic abscess.
- Amoebic liver abscess due to *Entamoeba histolytica* accounts for 10% of cases.
- Fungal abscess, most often due to *Candida* species, accounts for less than 10% of cases.
- Iatrogenic abscess, caused by medical interventions

PYOGENIC LIVER ABSCESS

Pyogenic Liver Abscess is a pus-filled area in the liver that is caused by pyogenic bacteria.

ETIOLOGY

infection from the biliary system (e.g. ascending cholangitis) is now more common due to the increasing use of radiological and endoscopic intervention. Infection may spread through the portal vein from abdominal sepsis (e.g. appendicitis, diverticulitis), via the hepatic artery from a septic focus anywhere in the body, or by direct spread from a contiguous organ (e.g. empyema of the gallbladder).

Abscess formation may follow blunt or penetrating injury and can be predisposed by alcoholism, metastatic cancer, and diabetes mellitus (weakened kupffer cells?), and in one-third of patients the source of infection is indeterminate (cryptogenic).

Multiple organisms are usually isolated; however, they commonly include *Escherichia coli, Staphylococcus aureus* and *anaerobes.*

CLINICAL FEATURES

- **Classic triad of pyogenic liver abscess**
    - Fever
    - Malaise
    - Right upper quadrant pain
- **Other symptoms**
    - Anorexia and weight loss
    - Nausea and vomiting
- **Physical examination**
    - Jaundice
    - Tender hepatomegaly
    - Intercostal tenderness
    - Epigastric tenderness
    - Decreased breath sounds in right lower lobe of the lung
    - Features of sepsis

The symptoms of pyogenic liver abscess are often non-specific (e.g., fever, weight loss, etc.).

The inflammation may reach the overlying pleura and cause pleural effusion and atelectasis; thus, patients can complain of respiratory symptoms.

INVESTIGATIONS

- Complete blood count: neutrophilic leukocytosis, anemia
- Liver function tests: ↑ alkaline phosphatase (90%), ↑ AST and ALT,
- Inflammatory markers: ↑ ESR and CRP
- Blood culture: positive in ~ 50% of cases

Plain radiographs may show elevation of the diaphragm, pleural effusion and basal lobe collapse. Ultrasonography or CT is used to define the abscess (which is often irregular and thick-walled) and to facilitate percutaneous aspiration for culture. Radioisotope scans and MRI can be also used, but are infrequently considered. ERCP may be useful if biliary obstruction is thought to be responsible.

MANAGEMENT

Treatment for solitary abscesses is percutaneous drainage, Indications for surgical drainage (open/laparoscopic)

- Deep-seated abscess not amenable to percutaneous drainage
- Ruptured abscess
- Thick viscous pus which cannot be drained percutaneously

Antimicrobial therapy should be empiric, based on the etiology of the primary infection. Broad spectrum antibiotics can be used for up to 8 weeks (IV for 2 weeks followed by 6 weeks oral). Multiple small abscesses can be treated with antibiotics alone.

AMEBIC LIVER ABSCESS

Amoebic liver abscess is caused by the protozoa Entamoeba histolytica.

ETIOLOGY

Entamoeba histolytica is a protozoal parasite that infests the large intestine and is endemic in many tropical regions. Trophozoites released by the cyst in the intestine (amebic colitis) may penetrate the

mucosa to gain access to the portal venous system and so spread to the liver. The abscess is large and thin-walled, is usually solitary and in the right lobe, and contains brown sterile pus resembling anchovy sauce.

CLINICAL FEATURES

Amebic liver abscesses are either acute which may resemble Pyogenic abscess or chronic which is defined as being present more than 2 weeks duration.

Patients present with right upper quadrant pain which might be accompanied by anorexia, nausea, weight loss and night sweats. Examination reveals tender enlargement of the liver and jaundice (uncommon). Other features may result from lung and pleural involvement and include basal pulmonary collapse and pleural effusion.

INVESTIGATIONS

Chest x-rays are used to visualise atelectasis and spread of the abscess to the lung. Ultrasound and CT liver scans are used to demonstrate the site and size of the abscess, which often has poorly defined margins. The stools should be examined for amebae trophozoites or cysts. Direct and indirect serological tests (CF, IHA and ELISA) to detect amoebic protein are available.

MANAGEMENT

treatment may be commenced empirically in areas where the problem is endemic. Treatment consists of the administration of metronidazole with chloroquine phosphate and usually results in rapid resolution. Other drugs include dehydroemetine, emetine, and iodoquinol. The abscess should be aspirated by needle puncture, if there is no clinical response within 72 hours, if there was superinfection (treated as pyogen¬ic abscess), or if the abscess is large.

HYDATID DISEASE

This less common infestation is caused in humans by one of two forms of tapeworm. Echinococcus granuloses (mainly affects liver) and Echinococcus multilocular is (mainly affects lung).

LIFE CYCLE

The adult tapeworm lives in the intestine of the dog, from which ova are passed in the stool; sheep or humans serve as the intermediate host by ingesting the ova (figure 5). The condition is most common in sheep-rearing areas. Ingested ova hatch in the duodenum and the embryos pass to the liver (80%in the right lobe) through the portal venous system.

SURGICAL ANATOMY

The wall of the resulting hydatid cyst is surrounded by an adventitial layer of granulation tissue (pericyst) that represents the host immune response. Underneath the pericyst, a laminated hyaline membrane lined by germinal epithelium (endocyst), on which brood capsules containing scolices develop. The hydatid fluid is produced by cells of the germinal layer (figure 4).

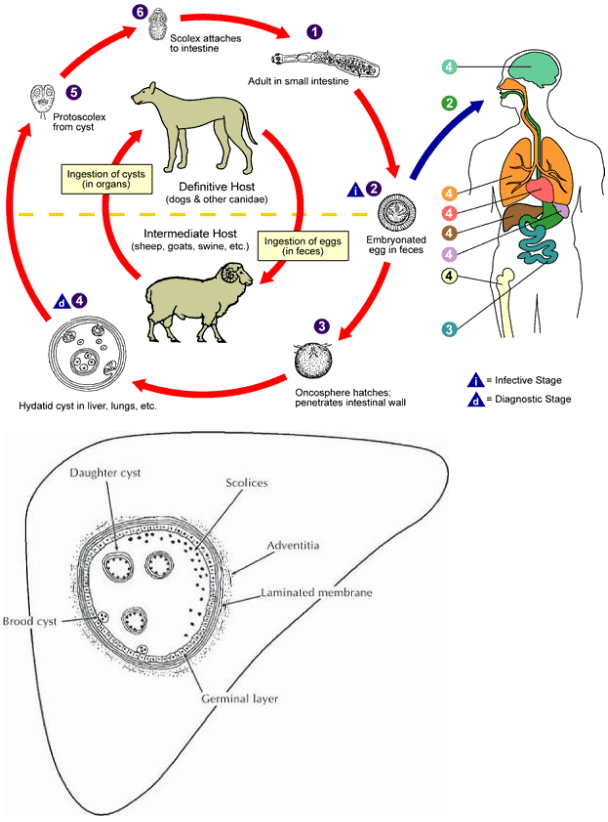


Figure 129 life cycle and structure of echinococcus

CLINICAL FEATURES

The disease may be symptomless, but chronic right upper quadrant pain with enlargement of the liver is the common presentation. The cyst may rupture

into the biliary tree or peritoneal cavity, the latter sometimes causing an acute anaphylactic reaction due to absorption of foreign hydatid protein. Other complications include secondary infection and biliary obstruction (psuedostones) with jaundice.

Chest or pulmonary symptoms may occur secondary to the coexistence of a hydatid cyst in the lung.

INVESTIGATIONS

eosinophilia is common and serological tests (IHA, CF, ELISA), are available to detect the foreign protein. The Casoni test is a skin test used in the diagnosis of hydatid disease. The test involves the intradermal injection of 0.5ml of sterilised fluid from hydatid cysts. A wheal response occurring at the injection site within 20 minutes is considered positive. The test is positive in about 90% of cases.

Hydatid cysts commonly calcify and may be seen on a plan film of the abdomen. Alternatively, they can be detected by ultrasound or CT scan of the liver and are recognizable by their thick wall, which may contain multiple daughter cysts.

MANAGEMENT

Hydatid cyst of the liver must be treated surgically.1,2 Albendazole 10 mg/kg/day for 3–6 weeks before surgery should be given to sterilise the cyst. PAIR method which includes Puncture with a needle, Aspiration of fluid under ultrasound guidance, installation of alcohol or hypertonic saline as scolicidal), then Re-aspiration after leaving the solution for 2 minutes. Large symptomatic cysts are best managed by complete excision, together with the parasites contained within.

TUMORS OF THE LIVER

Hepatic tumors can be benign or malignant, and primary or secondary.

Primary liver cancer is relatively rare while secondary liver cancer is much more common.

Primary tumors may arise from the parenchymal cells, the epithelium of the bile ducts, or the supporting tissues.

BENIGN HEPATIC TUMORS

LIVER CELL ADENOMAS

These are relatively uncommon and are found almost exclusively in women (20-40 years of age). They may be associated with the use of contraceptives containing high levels of estrogen. Pregnancy is also a predisposing factor; this may explain the age risk. The majority present as solitary, well-encapsulated lesions, but malignant transformation has been reported. They may be asymptomatic but generally present with right hypochondrial pain as a result of hemorrhage within the tumor. Superficial tumors may bleed spontaneously and present with symptoms of hemoperitoneum. Adenomas may be detected by ultrasonography or CT. MRI and isotope scan are also used. LFTs and serum α-fetoprotein levels are usually normal. Percutaneous biopsy should be avoided because of the risk of haemorrhage. It's only treated by surgical excision.

This is more common in females. The lesion is generally asymptomatic and may regress with time or on withdrawal of the contraceptive pill. Hyperplasia can be differentiated from adenoma by lobulation and the presence of a central fibrous scar (stellate scar), which is often visible on ultrasound or CT (figure 5). Such lesions do not undergo malignant transformation and do not require excision unless symptomatic.

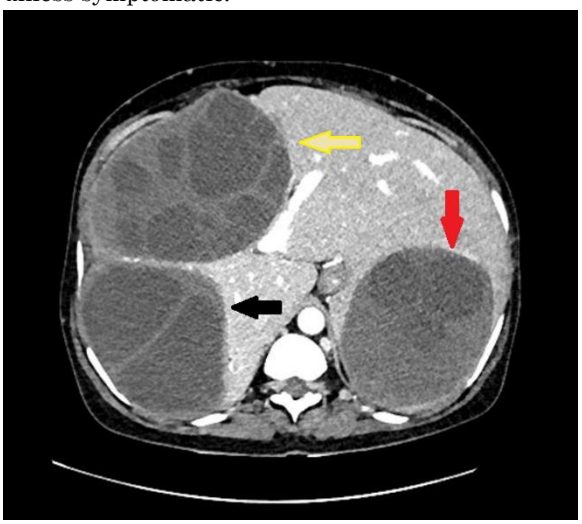


Figure 130 CT scan of the liver using Intravenous contrast enhancement and showing an opaque lesion with multiple lobulations and a central dark scar in the left lobe of the liver, lesion represents focal nodular hyperplasia.

CAVERNOUS HEMANGIOMA

This is the most common benign liver tumor and second in prevalence to metastatic tumors concerning tumors of the liver. Most cavernous hemangiomas are asymptomatic and are detected on ultrasonography as a dense hyperechoic lesion, or are found incidentally at laparotomy. These lesions rarely reach a sufficient size to produce pain, abdominal swelling or hemorrhage. Heart failure occasionally develops, if there is a large arteriovenous communication. Consumptive coagulopathy is also evident in cavernous hemangiomas. Lesions discovered incidentally at laparotomy should be left alone; needle biopsy can be hazardous. ^{99}technetium labelled to RBC's is of use for imaging of the tumor with C.T scan. Large symptomatic lesions can be treated with embolization, laser therapy or corticosteroids but should only be resected by an experienced surgeon.

PRIMARY MALIGNANT TUMORS OF THE LIVER

HEPATOCELLULAR CARCINOMA (HEPATOMA)

Hepatocellular carcinoma (hepatoma) is relatively uncommon in the developed world but is common in Africa and the Far East. In the West, about two-thirds of patients have pre-existing cirrhosis and many others have evidence of hepatitis B or C infection. In Africa and the East, 'aflatoxin' (derived from the fungus, Aspergillus flavus, which contaminates maize and nuts) is an important hepatocarcinogen. Pathogenesis includes activation of oncogenes and mutations in tumor suppressor gene TP53. A clinicopathological variant of the tumor not associated with any of the above-mentioned factors, fibrolamellar carcinoma, is evident in male and females in the age of 20 to 40 years and presents as a single hard scirrhous tumour.

CLINICAL FEATURES

The diagnosis is usually made late in the course of the disease. In non-cirrhotic patients, the tumor may have grown to a considerable size before giving rise to abdominal pain or swelling. In cirrhotic patients, hepatoma may become manifest as sudden deterioration in liver function, often associated with extension of the tumor into the portal venous system (ascites and splenomegaly). Common presenting features include abdominal pain, weight loss, abdominal distension, fever and spontaneous intraperitoneal hemorrhage. Jaundice is uncommon unless there is advanced cirrhosis. Examination may reveal features of established liver disease and hepatomegaly is invariable. Some forms of hepatoma secrets hormones and predispose a paraneoplastic syndrome (e.g cushing syndrome secondary to ACTH secretion).

INVESTIGATIONS

LFTs are generally deranged. Anemia may result from silent hemorrhages. Although early detection of hepatocellular carcinoma in susceptible individuals can be pursued by a policy of serial measurement of α- fetoprotein (an oncofoetal antigen) and ultrasound scanning, this tumour marker is present in only one- third of the white population with hepatocellular carcinoma, compared to 80% of African patients with this disease. CEA (carcinoembryonic antigen) is also detected in patients with hepatoma. Other means of investigation include CT, MRI, PET scan and angiography (highly vascularised tumor).

Percutaneous needle aspiration cytology and needle biopsy for histological confirmation should be reserved for patients who are not being considered for hepatic resection, as these investigations carry a small but significant risk of tumor dissemination and hemorrhage.

MANAGEMENT

- surgery. In some cases surgery may be used to remove cancerous tissue from the liver. However, the tumor must be small and confined.
- Radiation therapy. Radiation therapy uses high-energy rays to kill or shrink cancer cells.
- Chemotherapy. Chemotherapy uses anticancer drugs to kill cancer cells.
- Liver transplantation

In non-cirrhotic patients, large tumors (particularly those of the fibrolamellar type) may be amenable to liver resection. Cirrhotic patients have less hepatic functional reserve, and even those with well- preserved liver function may only tolerate limited segmental resection. embolization should be done before commencing surgery. In cirrhotic patients, multicentricity is common and satellite lesions often surround the primary tumor, so that cure is uncommon.

For advanced tumors, systemic chemotherapy may have palliative value, although response rates of less than 20% are the norm. More encouraging results have been reported following local embolization of these agents plus lipiodol by selective arteriography (chemo-embolization). Radiotherapy and liver transplant are also available options. 5-year survival rate is less than 10%.

CHOLANGIOCARCINOMA

This adenocarcinoma may arise anywhere in the biliary tree, is classified by its location in relation to the liver. Intrahepatic cholangiocarcinoma begins in the small bile ducts within the liver. This is the least common form of the disease, accounting for less than 10 percent of all cases. Perihilar cholangiocarcinoma (also known as a Klatskin tumor) begins in an area called the hilum, where two major bile ducts join and leave the liver. It is the most common form of the disease, accounting for more than half of all cases. The remaining cases are classified as distal cholangiocarcinoma, which begin in bile ducts outside the liver. The perihilar and distal forms of the disease, which both occur outside the liver, are sometimes grouped together and called extrahepatic cholangiocarcinoma.

Risk factors include chronic parasitic infestation of the biliary tree in the Far East (opisthorchis sinensis, chlonorchis spp.), choledochal cysts (caroli disease). Ulcerative colitis, sclerosing cholangitis, and hemochromatosis.

Jaundice, pain and an enlarged liver are the common presenting features, although there may be coexisting biliary infection causing the tumor to masquerade as a hepatic abscess. Resection offers the only prospect of cure but is seldom feasible when cholangiocarcinoma arises in the liver substance.

UPPER GASTRO-INTESTINAL BLEEDING

Upper gastrointestinal bleeding (UGIB) is defined as bleeding derived from a source proximal to the ligament of Treitz (figure 1) which connects the duodenum of the small intestines to the diaphragm and marks the beginning of the jejunum.

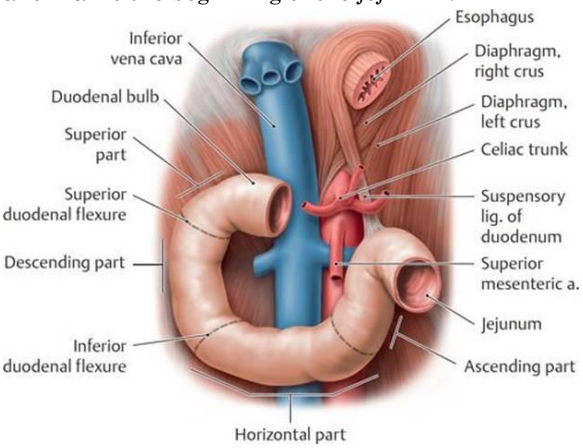


Figure 131 Ligament of Treitz

ETIOLOGY

The causes of upper gastrointestinal bleeding in order of incidence are shown in the table below. Peptic ulcers are the most common cause of UGIB, accounting for about 50% of cases.

causes of upper gastrointestinal bleeding
• **Peptic ulcer disease (35-50%)** • **Gastroesophageal erosions (10-20%)** • **Mallory-weiss tear (5%)** • **Esophageal varices (2-9%)** • **Tumors** • **Vascular lesions (5%)** • **Others**

CLINICAL FEATURES

Upper gastrointestinal bleeding presents with hematemesis and/or melena. Hematemesis is the symptom that describes vomiting of blood either in the form of fresh blood or passage of 'coffeeground' like-material. Melena describes the passage of black tarry stool that has a very characteristic smell Melena results from the digestion of blood by enzymes and bacteria and thus it indicates that blood has been present in the gastrointestinal tract for at least 14 hours. Very rarely, if bleeding is very brisk, upper gastrointestinal bleeding may present as fresh rectal bleeding (rectorrhagia) or Hematochezia, in which case signs of hemodynamic instability are usually present. Slow chronic blood loss may be asymptomatic and detected on rectal examination or by a positive fecal occult blood test (e.g. guaiac).

APPROACH TO THE PATIENT

DIAGNOSIS

UGIB is not a primary diagnosis, but rather a clinical scenario that needs to be dealt with and has a special approach. A patient is said to have UGIB when he presents with the previously mentioned symptoms. The following should be commenced afterward;

PHYSICAL EXAMINATION

• measurement of postural changes in blood pressure and pulse rate; There should be at least 1 minute between the measurement while supine and standing. Patients are said to have postural hypotension when there is more than 20 points drop in systolic blood pressure, postural tachycardia implies a 10 point increase in pulse rate after 1 minute of standing. Postural changes indicate a loss of more than 15-20% of blood volume, when there is an increase in the pulse (while supine) of more than 100 bpm or a fall in blood pressure more than 100 mmHg, this indicates a loss of 30% of blood volume.

• A digital rectal examination is very important to detect the presence of blood in the rectum. This does not mean that the blood is coming from the rectum, but indicates that the patient is bleeding. in UGIB, the blood is usually tarry and black with offensive smell, but brisk bleeding can result in detecting fresh blood.

• Specific features to be looked for include signs of liver disease and portal hypertension (spider nevi, portosystemic shunting and bruising). They are particularly important, as variceal hemorrhage necessitates specific treatment.

INITIAL MANAGEMENT

After the diagnosis of GIB (not UGIB), Whatever the cause, the principles of management are identical. The patient should be first resuscitated and then investigated urgently to determine the cause of the bleeding, this should follow the same principles of treating hemorrhage (see the lecture on hemorrhage);

- 2 wide bore venous cannulas are inserted, one for installing fluids and one for drawing blood for CBC, coagulation profile, urea and electrolytes and cross-matching.
- A central venous catheter is considered when there are signs of cardiovascular instability.
- Bladder catheterization for monitoring urine output
- Insertion of a nasogastric tube to confirm wither there is blood in the stomach (and thus help confirm UGIB). This will help guide the endoscopic therapy but has no specific therapeutic value. A non-bloody nasogastric aspirate may be seen in up to 16% of patients with UGIB. It will also monitor the bleeding and prevent aspiration.

Patients should be replaced with fluids as assessed by the vital signs, urine output and/or central venous pressure and they are better to have blood transfusion, but not if the fluids enough. In 80% of the cases, the bleeding stops spontaneously. In cases of variceal UGIB, some manoeuvres can be lifesaving. These are considered in life threatening cases. For example, a patient with known liver disease and portal hypertension and a life-threatening bleeding, a Sengstaken- Blakemore tube (figure 2) may be inserted before an endoscopy has been carried out.

Results of blood test are important, usually the hemoglobin level does not fall, and only falls after 3 days, because of hemodilution. The hematocrite should be kept more than 30% in older patients and those with coronary artery disease. Iron deficiency anemia is expected in chronic or occult bleeding. Prolonged prothrombin time is an indication for giving fresh frozen plasma. Low platelets are also an indication for platelets transfusion.

DIFFERENTIATION OF UPPER FROM LOWER GIB

The following may help in the differentiation between upper (above the ligament of treitz) and lower sources of GIB;

- Hematemesis indicates an UGIB.
- Melena indicates that blood has been present in the Gl tract for at least 14 hours. Thus, the more proximal the bleeding site, the more likely melena will occur.
- Hematochezia usually represents a lower Gl source of bleeding, although an upper Gl lesion may bleed so briskly that blood does not remain in the bowel long enough for melena to develop. When hematochezia is the presenting symptom of UGIB, it is associated with hemodynamic instability and dropping hemoglobin.
- bloody nasogastric aspirate indicates an upper gastrointestinal source of bleeding.
- hyperactive bowel sounds and an elevated blood urea nitrogen level (due to volume depletion and blood proteins absorbed in the small intestine).

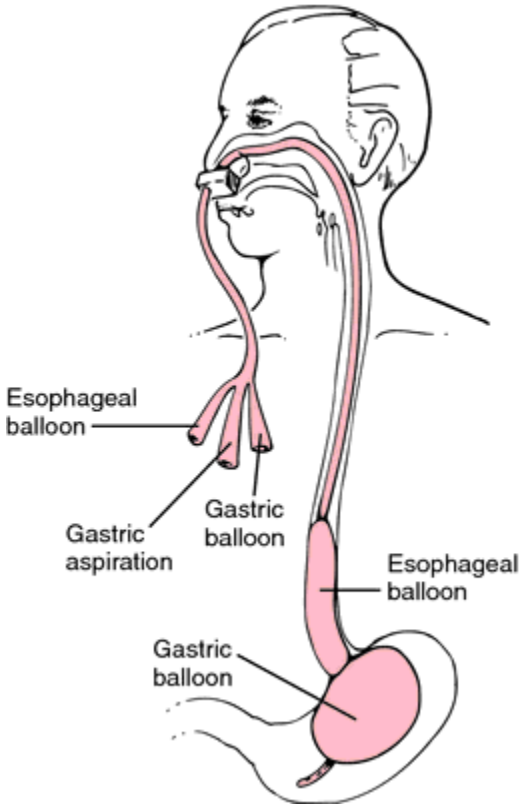


Figure 132 Sengstaken- Blakemore tube

ENDOSCOPY

Endoscopy is the most accurate test to determine the etiology of upper Gl bleeding, and should be performed urgently in patients with hemodynamic instability. It has the marked advantage of providing therapeutic interventions after visualization of the lesion

The management is now considered for each diagnosis made by endoscopy.

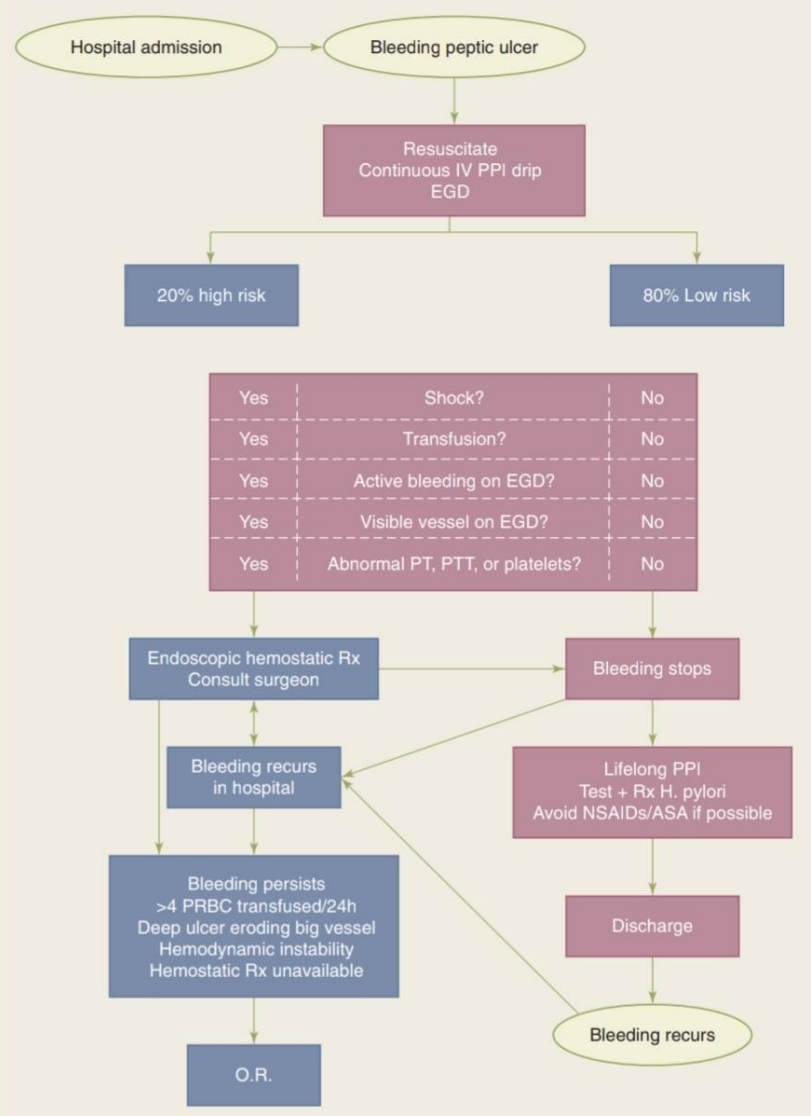
BLEEDING PEPTIC ULCERS

Bleeding peptic ulcers are caused by erosion of an artery passing below the site of the ulcer. In the case of duodenal ulcers, the gastroduodenal artery is most commonly involved in posterior bulbar ulcers. In gastric ulcer most common site is lesser curvature (duodenal ulcer are twice more common than gastric ulcer) . 25% of patients with duodenal ulcers present with upper UGIB as their first manifestation and is the most common cause of mortality, accounting for 40% of deaths.

MANAGEMENT

80-85% of patient spontaniosly stops bleeding

Once the patient is stable, endoscopy is commenced to identify the source of bleeding. Sometimes the bleeding stops spontaneously, but if an active bleeding is detected laser therapy, electro-coagulation, sclerotherapy (e.g. absolute alcohol, 1:10,000 epinephrine), cauterization or arterial banding are used. These manoeuvres can also be applied if endoscopy reveals a nonbleeding visible vessel, because one third of these patients may rebleed. In



contrast, patients with clean-based ulcers have rates of rebleeding approaching zero. If there is no other reason for hospitalization, such patients may be discharged on the first hospital day, following stabilization. Patients without clean-based ulcers (visible vessel, blood, clot) should usually remain in the hospital for 3 days, since most episodes of recurrent bleeding occur within 3 days.

Emergency surgery may be indicated if there is massive bleeding unable to control. Surgery might also be considered when the patient is elderly and unfit, or if more than 4-6 units of blood has been transfused.

A bleeding duodenal ulcer may simply be under-run with sutures, through a duodenotomy (opening of the anterior wall of the duodenum) to gain access to the ulcer or pyloromyotomy. With a bleeding gastric ulcer, the possibility of malignancy must be considered. The ulcer must be biopsied in all cases to determine its nature. In young fit patients, the ulcer should be excised completely by taking a small wedge resection. In elderly patients or those with significant comorbidity, under-running of the ulcer may be preferable, at least in the first instance. if the pathology result confirms malignancy, then the patient should have accurate staging and further treatment as indicated.

Randomized controlled trials document that high-dose constant infusion intravenous omeprazole (a PPI; 80-mg bolus and 8-mg/h infusion), used to raise intra- gastric pH to between 6 and 7 and enhance clot stability, decreases further bleeding (but not mortality), even after the use of appropriate endoscopic therapy in patients with high-risk ulcers (active bleeding, non-bleeding visible vessel, and perhaps adherent clot).

Because ulcers recur (or rebleed) within the 2 years (most likely the first 3 days), thus treatment for eradication of H.pylori and avoiding NSAIDs is very important to decrease the risk of recurrence.

GASTROESOPHAGEAL EROSIONS

Hemorrhagic or erosive gastropathy (e.g., due to nonsteroidal anti-inflammatory drugs (NSAIDs) or alcohol] and erosive esophagitis often cause mild UGIB, but major bleeding is rare. Hemorrhagic and erosive gastropathy, or gastritis, refers to endoscopically visualized subepithelial hemorrhages and erosions. These are mucosal lesions and thus do not cause major bleeding.

Stress-related gastric mucosal injury occurs only in extremely sick patients: those who have experienced serious trauma, major surgery, burns covering more than one-third of the body surface area (curling's ulcer), major intracranial disease (Cushing's ulcer), and severe medical illness (i.e., ventilator dependence, coagulopathy). Significant bleeding probably does not develop unless ulceration occurs. The mortality rate in these patients is quite high because of their serious underlying illnesses. Pharmacologic prophylaxis for bleeding may be considered in the high-risk patients mentioned above. The best data suggest that intravenous H2-receptor antagonist therapy is the treatment of choice, although sucralfate is also effective.

MALLORY-WEISS TEAR

The classic history is vomiting, retching, or coughing preceding hematemesis, especially in an alcoholic patient. It is because of the strenuous and repetitive vomiting; the lesion is induced. It is usually located in the cardia (at gastroesophageal junction) (90%) or in esophagus (10%)

Bleeding from these tears stops spontaneously in 80 to 90% of patients and recurs in only 0 to 5%. endoscopic injection therapy is needed when the tear is actively bleeding and is usually sufficient. Angiographic therapy with (intra-arterial vasopressin) or surgical therapy (under-running with sutures) are needed when the tear continues to bleed despite endoscopic therapy, but are rarely required.

ESOPHAGEAL VARICES

Patients with variceal hemorrhage have poorer outcomes than patients with other sources of UGIB. oesophageal varices are the most common cause of variceal hemorrhage. They are the result of portal hypertension which opens venous collaterals (portosystemic shunts) present in the esophagus (most commonly).

In cases of acute bleeding, vasoconstrictor therapy can be used to restrict portal inflow by splanchnic arterial constriction. octreotide (a somatostatin analogue; 50-µg bolus and 50-µg/h intravenous infusion for 2 to 5 days) may help in the control of acute bleeding. Agents such as somatostatin (250-500

µg/h) and terlipressin, available outside the United States, are also effective. Over the long term, treatment with nonselective beta blockers decreases the rate of recurrent bleeding from esophageal varices. Beta blockers are commonly given along with chronic endoscopic therapy. As mentioned earlier, a Sengstaken-Blakemore tube (refer to figure 2) is used when there is suspicion of variceal hemorrhage with life threatening situation.

Vasoconstrictor therapy is useful until endoscopy is commenced. Ligation is the endoscopic therapy of choice for esophageal varices. Sclerotherapy is the less preferred alternative (it causes strictures).

In patients who have persistent or recurrent bleeding despite endoscopic and medical therapy, more invasive therapy is warranted. Transjugular intrahepatic portosystemic shunt (TIPS, figure 3) decreases rebleeding more effectively than endoscopic therapy, although hepatic encephalopathy is more common and the mortality rates are comparable. In this technique, a guidewire is passed from the jugular vein into the liver and an expandable metal shunt is forced over it into the liver substance to form a channel between the systemic and portal venous systems. It reduces the hepatic sinusoidal and portal vein pressure by creating a total shunt, but without the risks of general anaesthesia and major surgery. TIPS is most appropriate for patients with more severe liver disease and those in whom transplant is anticipated. Patients with milder, well-compressive cirrhosis should probably undergo decompressive surgery (e.g. distal splenorenal shunt).

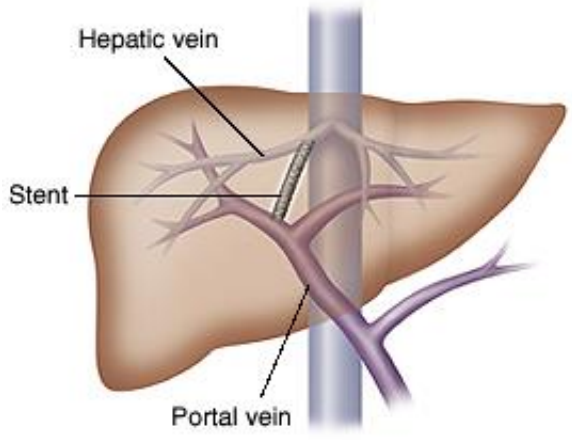


Figure 133 Transjugular intrahepatic portosystemic shunt (TIPS)

Portal hypertension is also responsible for bleeding from gastric varices, varices in the small and large intestine, and portal hypertensive gastropathy and enterocolopathy.

OTHER CAUSES OF UGIB

Tumors may present with UGIB, which can either be acute or chronic, but is usually not severe. They are treated according to the type of tumor.

A dieuflafoy's lesion refers to the presence of an ar¬terio-venous malformation in the stomach. It is cha¬racterized by an unusually large tortuous submucosal artery. If this artery is eroded, significant bleeding may occur, endoscopy can miss the lesion if it is not actively bleeding. If it can be seen during the bleeding, all that may be visible is profuse bleeding coming from an area of apparently normal mucosa.

Treatment options include endoscopic therapy (usual¬ly injection sclerotherapy), angiographic embolization, or operation. At surgery, the lesion may be oversewn or resected.

OBSCURE GIB

Obscure GIB is defined as recurrent acute or chronic bleeding for which no source has been identified by routine endoscopic and contrast x-ray studies. When the presentation is UGIB, The lesion is usually in the small intestines. This may require push enteroscopy (to push through the duodenum with a pediatric endoscope) or video capsule enteroscopy (using specialized capsule). If enteroscopy and video capsule endoscopy are negative or unavailable, a specialized radiographic examination of the small bowel (i.e. enteroclysis) should be performed. 99MTc-labeled red blood cell scintigraphy should be considered. When the patient is young, 99MTc-pertechnetate scintigraphy for diagnosis of Meckel's diverticulum.

The diagnosis of aortic enteric fistula should be considered. There is usually a history aortic graft replacement or a CT scan may show an untreated aortic aneurysm.

PROGNOSIS

The mortality rate of UGIB is ~5 to 10%. Patients rarely die from exsanguinations; rather, they die due to decompensation from other underlying illnesses. The mortality rate for patients under 60 years of age in the absence of malignancy or organ failure is <1%, Thus, The three independent clinical predictors of death in patients hospitalized with UGIB are increasing age, comorbidities, and hemodynamic compromise.

There are scores which calculate the rate of mortality according to these factors. These include the Blatchford risk assessment (designed to be used preendoscopy), and the full Rockall score (after endoscopy). The details are beyond the scope of this lecture.

LOWER GASTROINTESTINAL BLEEDING

lower gastrointestinal bleeding (LGIB) is defined as bleeding derived from a source distal to the ligament of Treitz (figure 1) which connects the duodenum of the small intestines to the diaphragm and marks the beginning of the jejunum. recently, because of the fact that management of bleeding from the small intestines is especial, it came to represent a different entity and the definition is being shrank to the colon and anorectal region. Hemorrhage from the lower gastrointestinal tract accounts for about 20% of all cases of *acute* gastrointestinal bleeding.

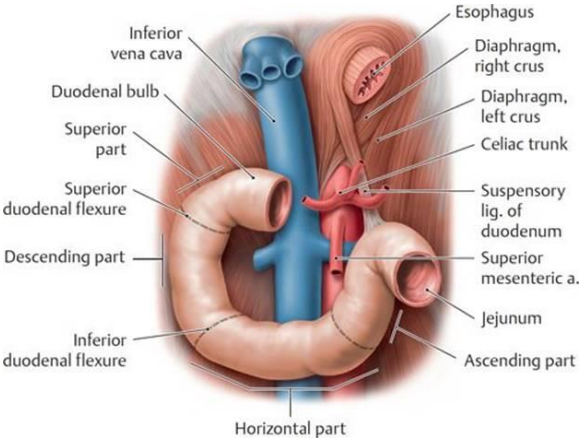


Figure 134 ligament of Treitz

CLINICAL FEATURES

acute LGIB is defined as being of recent duration (arbitrarily designated as less than 3 days) and might result in instability of vital signs, anemia and/or the need for blood transfusion. Chronic LGIB is the passage of blood from the rectum over a period of several days or longer and usually implies that blood loss is intermittent or slow, a patient with chronic bleeding can show occult fecal blood, iron-deficiency anemia, occasional episodes of melena, hematochezia or maroon stools, or small quantities of visible blood per rectum, alternatively, however, LGIB can be subdivided into two categories: clinically overt gastrointestinal bleeding (melena, hematochezia, rectorrhagia) or occult bleeding, identified by an unexplained iron deficiency and/or positive fecal occult blood testing result.

Hematochezia is defined as gross blood seen either on toilet paper after defecation or mixed with stool. Rectorrhagia means the expulsion of fresh red bright blood without stools, while melena is defined as black tarry offensive soft stools resulting from the oxidation of hematin in the gut.

ETIOLOGY

the sources of acute LGIB are shown in the table below.

sources of lower gastrointestinal bleeding
• **Diverticulum**
• **Angiodysplasia**
• **Colitis (ischemic, infectious, chronic IBD, radiation injury)**
• **Neoplasia**
• **postpolypectomy bleeding**
• **Anorectal disease**
• **Small bowel bleeding**

DIFFERENTIATION OF UPPER FROM LOWER

Compared with acute upper gastrointestinal bleeding, patients with acute LGIB are significantly less likely to experience shock. The following may help in the differentiation between upper (above the ligament of Treitz) and lower sources of GIB;

- Hematemesis indicates an UGiB.
- Melena indicates that blood has been present in the Gl tract for at least 14 hours. Thus, the more proximal the bleeding site, the more likely melena will occur.
- Hematochezia usually represents a lower Gl source of bleeding, although an upper Gl lesion may bleed so briskly that blood does not remain in the bowel long enough for melena to

develop. When hematochezia is the presenting symptom of UGIB, it is associated with hemodynamic instability and dropping hemoglobin and this is less likely to occur in LGIB.

- bloody nasogastric aspirate indicates an upper gastrointestinal source of bleeding.
- hyperactive bowel sounds and an elevated blood urea nitrogen level (due to volume depletion and blood proteins absorbed in the small intestine) indicate UGIB.

INITIAL EVALUATION

patients with acute LGIB, who might have signs of hemodynamic instability should be resuscitated thoroughly according to the extent of blood loss. 2 wide bore cannulas are installed, and blood is withdrawn for CBC, cross-match, coagulation profile and urea and electrolytes, proper replacement with I.V fluids or blood products is done, a urinary catheter is important for input-output charts, oxygen is administered as needed.

patients with acute LGIB who were resuscitated and those with chronic LGIB (overt or occult) should be question by history and searched for the cause by examination and proper investigations.

HISTORY

The duration, frequency, and color of blood passed per rectum may help discern the severity and location of bleeding. Characteristically, melena or black, tarry stool, indicates bleeding from an upper gastrointestinal or small bowel source, whereas bright red blood per rectum (rectorrhagia) that passes independently from the passage of stool signifies bleeding from the left colon or rectum. hematochezia (mixed with stool or with paper) indicates the presence of anorectal disease but can be seen with other causes such as major upper gastrointestinal hemorrhage and haemorrhage from the small intestines. anorectal disease (hemorrhoids, fissures) is more likely to present with bleeding of the outlet type; that is, bleeding that passes after the passage of stool and is usually evident on toilet paper.

The past medical history may also help to elucidate a specific bleeding source. Key points include antecedent constipation or diarrhea (hemorrhoids, colitis), the presence of diverticulosis (diverticular bleeding), receipt of radiation therapy (radiation enteritis), recent polypectomy (postpolypectomy bleeding) and vascular disease/hypotension (ischemic colitis). A family history of colon cancer increases the likelihood of a colorectal neoplasm and generally calls for a complete colonic examination in patients with hematochezia. A history of anal pain, pruritus, and fresh red blood on the toilet paper (bleeding of the outlet type) is frequently the expression of anal fissures or haemorrhoids.

EXAMINATION

examination of the anal region of the utmost importance as it may arrest the management at a certain point without further invasive investigation, however, despite presenting features, and findings on physical examination, most patients with LGIB warrant a full examination of the colon, especially if they were above the age of 40 years old.

the anal and perianal region should be inspected thoroughly for the presence of masses that indicate external hemorrhoids or 3rd degree piles, the presence of skin tags on the posterior anoderm indicates the presence anal fissures.

Digital examination is important for detection of pathologies inside the rectum and the distal anal canal. the important of these are divarticulae and neoplasms.

INVESTIGATIONS AND MANAGEMENT

The patient's age affects the clinical approach to LGIB. In children and young patients, cow milk allergy, polyps, Meckel's diverticulum, inflammatory bowel diseases, and anal diseases should be especially considered. Older patients are more likely to have diverticular disease, angiodysplasia, ischemic diseases, colorectal cancer, inflammatory bowel diseases, polyps, nonsteroidal anti-inflammatory drug (NSAID)-induced lesions, and lesions from prior radiotherapy, and may also have anal diseases. Other vascular malformations are infrequent but should occasionally be considered .

Young women could be affected by endometriosis, and when bleeding is episodic or chronic colonoscopy would be better scheduled for menstruation days.

Patients with presumed LGIB may undergo early sigmoidoscopy for the detection of obvious, low-

lying lesions. However, the procedure is difficult with brisk bleeding, and it is usually not possible to identify the area of bleeding. Sigmoidoscopy is useful primarily in patients <40 years with minor bleeding.

Patients with hematochezia and hemodynamic instability (acute LGIB) should have upper endoscopy to rule out an upper Gl source before evaluation of the lower Gl tract, upper endoscopy is also indicated in patients with chronic LGIB suspected to have UGIB.

You can use NGT: if suction is blood this mean UGIB, if bile most likely no UGIB, if clear fluid can't rule out.

Colonoscopy after an oral lavage solution is the procedure of choice in patients admitted with LGIB unless bleeding is too massive or unless sigmoidoscopy has disclosed an obvious actively bleeding lesion, colonoscopy provides both a diagnostic and therapeutic tool.

99MTc-labeled red cell scan allows repeated imaging for up to 24 h reveals bleeding even with a low rate of blood lose(01ml/min).it doesn't localize the lesion it only identifies continued bleeding.

In active LGIB, angiography can detect the site of bleeding (extravasation of contrast into the gut) and permits treatment with intraarterial infusion of vasopressin or embolization. Even after bleeding has stopped, angiography may identify lesions with ab-normal vasculature, such as vascular ectasias or tumours.

Occasionally, the LGIB source is in the small intestine. Push enteroscopy and capsule endoscopy have been used for the assessment of obscure bleeding.

the management of patients with lower Gl bleeding are shown in figure 2.

- (80-90%) of patient stop bleeding spontaneously, stabilize the patient then do colonoscopy

- (10%) continuous bleeding ,can't do colonoscopy and can't determine site of bleeding , so need emergent surgery for total colonectomy

N.B:

- Most common gastrointestinal tract bleeding is UGITB
- Most common MASSIVE LGITB is diverticulosis then angiodysplasia
- Most common light LGIB is anal conditions
- Any massive bleeding in patient >40 you must investigate to rule out colon cancer

OCCULT GIB

Occult GIB is manifested by a positive test for fecal occult blood or iron-deficiency anemia. Evaluation of a positive test for fecal occult blood generally should begin with colonoscopy, particularly in patients >40 years. If evaluation of the colon is negative, many perform upper endoscopy only if iron-deficiency anemia or upper Gl symptoms are present, while others recommend upper endoscopy in all patients since up to 25 to 40% of these patients may have some abnormality noted on upper endoscopy. If standard endoscopic tests are unrevealing, enteroscopy, video capsule endoscopy, and/or enteroclysis may be considered in patients with iron-deficiency anemia.

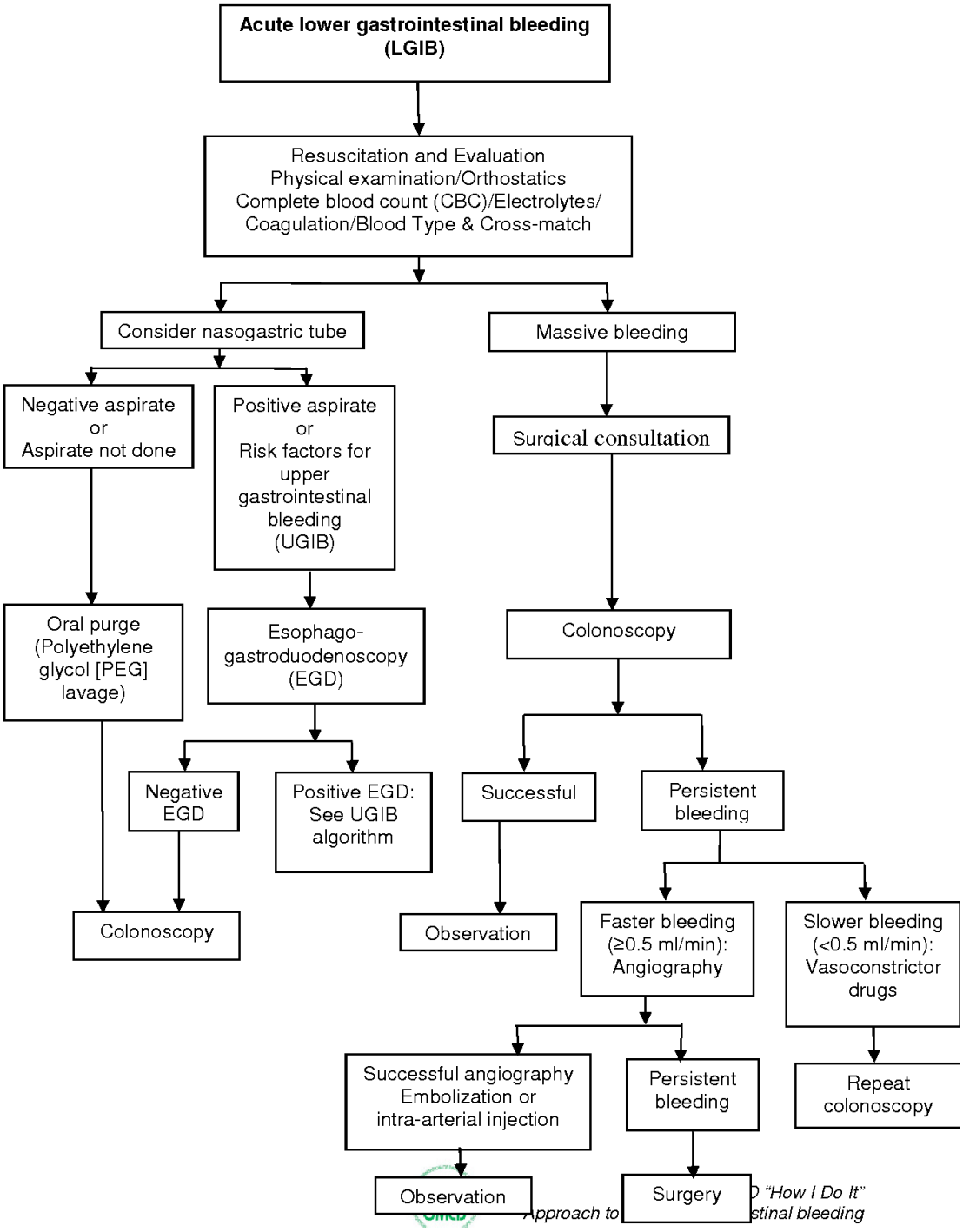


Figure 135 approach to manage a patient with lower GI bleeding

THE GALLBLADDER AND THE BILIARY TRACT

ANATOMY

THE GALLBLADDER

The gallbladder is a pear-shaped structure, 7.5-12 cm long, with a normal capacity of about 20-30 ml (capacity can reach up to 200-300 ml). The anatomical divisions are a fundus, a body and a neck that terminates in a narrow infundibulum (figure 1).

The gallbladder receives its blood supply from the cystic artery which branches from the right hepatic artery. Occasionally this artery arises from the left hepatic, the right gastric or the superior mesenteric artery. The veins drain into the portal venous system. The lymphatic vessels of the gall bladder (subserosa and submucosal) drain into the cystic lymph node of Lund (the sentinel lymph node). Efferent vessels from this lymph node go to the hilum of the liver and to the celiac lymph nodes. The gallbladder is supplied by both sympathetic (celiac plexus) and parasympathetic nerves (vagus nerve).

THE BILIARY TRACT

The cystic duct is about 0.5-4 cm in length. The mucosa of the cystic duct is arranged in spiral folds known as the valves of Heister. Occasionally, an accessory cystic duct is present.

The common hepatic duct is usually less than 2.5 cm(1-2.5cm) long and is formed by the union of the right and left hepatic ducts. The common bile duct is about 7.5 cm long and is formed by the junction of the cystic and common hepatic ducts. It is divided into four parts:

- the supraduodenal portion, about 2.5 cm long, running in the free edge of the lesser omentum;
- the retroduodenal portion;
- the infraduodenal portion (pancreatic), which lies in a groove, but at times in a tunnel, on the posterior surface of the pancreas;
- the intraduodenal portion (intramural), which passes obliquely through the wall of the second part of the duodenum, where it is surrounded by the sphincter of Oddi, and terminates by opening on the summit of the ampulla of Vater (10 cm from the pylorus).

HISTOLOGY

The gallbladder and the biliary tract are covered by columnar epithelium. The gallbladder differs histologically from the rest of the gastrointestinal tract in that it lacks a muscularis mucosa and submucosa. Small ducts (of Luschka) may drain directly from the liver into the body of the gallbladder.

The mucus membrane contains indentations of the mucosa that sink into the muscle coat; Rokitanzky Aschoff sinuses which is found in 40% of normal G.B and in all inflamed, so one of the characters to DX of acute cholecystitis is found these sinuses.

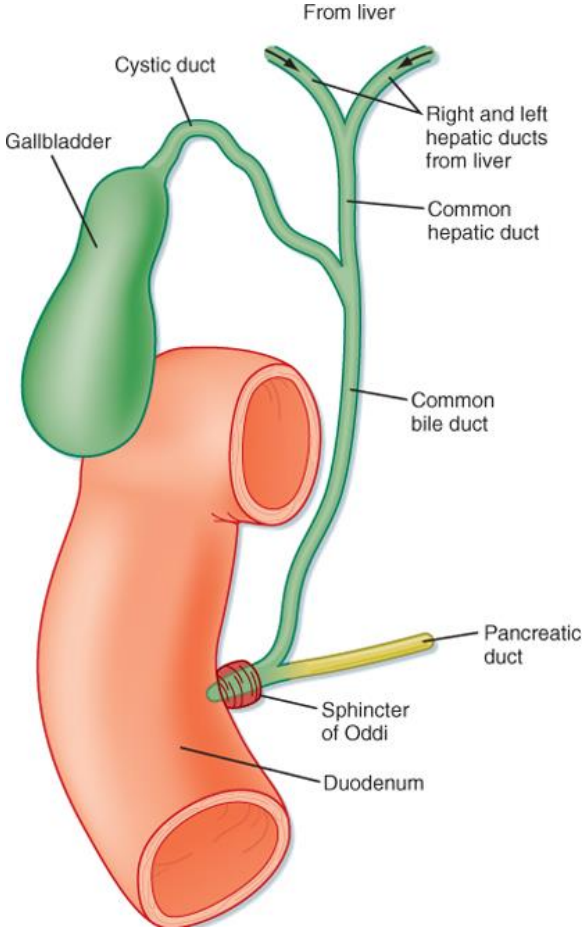


Figure 136 gallbladder and the extrahepatic ducts anatomy

PHYSIOLOGY

The liver produces bile continuously and excretes it into the bile canaliculi. The gallbladder functions as a reservoir for bile. Gallbladder filling is facilitated by tonic contraction of the sphincter of Oddi, which creates a pressure gradient between the bile ducts and the gallbladder . The sphincter of Oddi is about 4 to 6 mm in length and has a basal resting pressure of about 13 mm Hg above the duodenal pressure, the sphincter shows phasic contractions with a frequency of about four per minute and an amplitude of 12 to 140 mm Hg.

Relaxation occurs with a rise in the hormone cholecystokinin (CCK); after a meal, leading to diminished amplitude of phasic contractions and reduced basal pressure, allowing increased flow of bile into the duodenum. The vagus nerve stimulates contraction of the gallbladder, and splanchnic sympathetic stimulation is inhibitory to its motor activity. VIP inhibits contraction and causes gallbladder relaxation. Somatostatin and its analogues are potent inhibitors of gallbladder contraction.

About 80-90% of bile secreted by the liver passes to the gallbladder, where it is stored. The gallbladder in turn can absorb water and thus concentrate bile to about 2-10 times of its original concentration.

CALCULOUS BILIARY DISEASE

Calculous biliary disease is one of the most common problems affecting the digestive tract. Autopsy reports have shown a prevalence of gallstones from 11 to 36%. The hallmark of this disease is the accumulation of stones inside the gallbladder.

CLASSIFICATION

Gallstones form as a result of solids settling out of solution. The major organic solutes in bile are bilirubin, bile salts, phospholipids, and cholesterol. Gallstones are classified by their cholesterol content as either cholesterol stones (10%) or pigment stones (bile) (15%). The most common are mixed stones (cholesterol with ca+2 or bilirubin or... , but the dominant is cholesterol) (75%). Pigment stones can be further classified as either black or brown.

Biliary tract stones are usually secondary, which follows migration of a gallstone to the biliary tract or as a residual after cholecystectomy. Primary biliary tract stones can either be black (which follow a haemolysis disease or liver cirrhosis) or brown (which follow a local infection or obstruction). Biliary tract stones affect about 10% of the population and the incidence appears to be increasing linearly with age.

ETIOLOGY

The prevalence of gallstones is related to many factors, including age, gender, and ethnic background. Certain conditions predispose to the development of gallstones, factors Predisposing the development of stones in the biliary tree and the gallbladder are shown in the table below.

Factors predisposing to biliary stones
• hereditary and ethnic factors
• Gender (F:M = 3 :1)
• Pregnancy (increase progesterone cause stasis of bile→GB stone)
• Obesity (lipid converted into cholesterol)
• Diabetes mellitus (due to neuropathy → decrease contraction)
• Haemolytic disorders
• Liver Cirrhosis
• Vagotomy
• Total parenteral nutrition (TPN) (decrease eating → no contraction → stasis of bile)
• ileum disorder
• short bowel syndrome
• congenital anomalies ***5F: female , fair , forty , fertility . fat**

as has been said earlier. Gallbladder stones form as a result of solids settling out of solution. The major organic solutes in bile are bilirubin, bile salts, phospholipids, and cholesterol.

Cholesterol stones are formed when the bile is supersaturated with cholesterol. Brown pigment stones are formed of unconjugated bilirubin with other calcium containing compounds and form in the bile duct and are related to bile stasis and infected bile. Black pigmented stones are largely composed of an insoluble bilirubin pigment polymer mixed with calcium phosphate and calcium bicarbonate. Black stones accompany haemolysis, usually hereditary spherocytosis or sickle cell disease. For unclear reasons, patients with cirrhosis have a higher instance of pigmented stones.

CLINICAL FEATURES

There is a range of presentations that a patient with calculous biliary disease could exhibit. Most patients will remain asymptomatic from their gallstones throughout life. For unknown reasons some patients progress to a symptomatic stage, with biliary colic caused by a stone obstructing the cystic duct. Symptomatic gallstone disease may progress to complications related to the gallstones. These include acute cholecystitis, choledocholithiasis, gallstone pancreatitis, and gallbladder carcinoma. Less frequently, a complication of gallstones is the presenting picture.

BILIARY COLIC (LAST FOR LESS THAN 6 HOURS)

Patients typically complain of right upper quadrant or epigastric pain, which may radiate to the back. The pain Rapidly increases in intensity which may last several hours followed by gradual decrease. The pain is aggravated by consuming meals, especially those with high fat content. Associated symptoms include dyspepsia, flatulence and food intolerance.

ACUTE CHOLECYSTITIS (LAST FOR MORE THAN 6 HOURS)

Acute cholecystitis is usually caused by a long standing obstruction of the gallbladder, especially at the level of the cystic duct. It is the result of a complex process that begins as a chemical reaction with subsequent development of bacterial infection.

CLINICAL FEATURES

Acute cholecystitis usually begins as an attack of biliary colic, but in contrast to biliary colic, the pain does not subside; it is unremitting and may persist for several days. The pain may radiate to the subscapular area due to irritation of the diaphragm. The patient is often febrile, complains of anorexia, nausea, and vomiting.

On physical exam, the patient is usually febrile with tachycardia and focal tenderness and guarding are usually present in the right upper quadrant. A Murphy's sign; an inspiratory arrest with deep palpation in the right subcostal area, is characteristic of acute cholecystitis.

Untreated, the patient usually recover as the stone is dislodged from the tract or may end up with one of the complications shown in the table below.

Complications of acute cholecystitis

- **Gangrene**
- **Empyema**
- **Perforation**
- **Emphysematous cholecystitis**
- **Cholangitis with jaundice**
- **Hydrops (Mucocele gall bladder); no infection, filled with mucus.**

INVESTIGATIONS

A mild to moderate leucocytosis (12,000 to 15,000 cells/mm^3) is usually present. Serum liver chemistries are usually normal, but a mild elevation of serum bilirubin, less than 4 mg/mL, may be present.

Ultrasonography is the most useful radiologic test for diagnosing acute cholecystitis. In addition, for documenting the presence or absence of stones, it will show the thickening of the gallbladder wall and the pericholecystic fluid (figure 3) oral cholecystography oral administration of a radiopaque compound that is absorbed, excreted by the liver, and passed into the gallbladder. It may visualize stones as filling defects. It has largely been replaced by ultrasonography. Biliary radionuclide scanning (HIDA scan) may be of help in the

atypical case. Lack of filling of the gallbladder after 4 hours indicates an obstructed cystic duct.

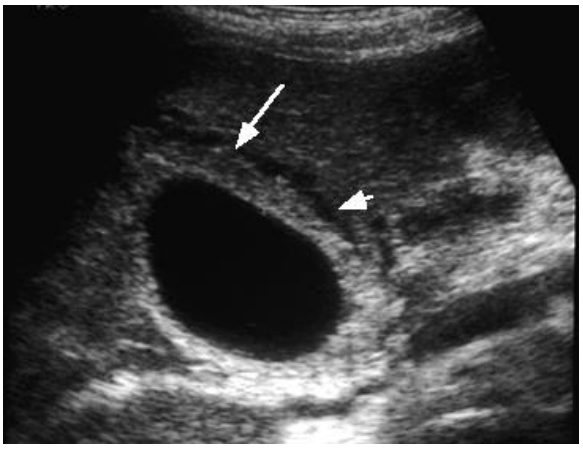


Figure 137 Acute cholecystitis Ultrasound of the right upper quadrant in a patient with acute cholecystitis reveals marked thickening of the gallbladder wall (arrow) with fluid surrounding the distended gallbladder (arrowhead).

MANAGEMENT

Patients who present with acute cholecystitis will need intravenous fluids (kept NPO), antibiotics, and analgesia. The antibiotics should cover gram-negative aerobes as well as anaerobes. Cholecystectomy is the definitive treatment for acute cholecystitis. The timing of cholecystectomy has been a matter of debate in the past. Early cholecystectomy performed within 3 to 5 days of the illness is preferred over elective or delayed cholecystectomy that is performed 4 to 6 weeks after initial medical treatment. Laparoscopic cholecystectomy is the procedure of choice for acute cholecystitis. An open cholecystectomy can be performed instead. Other options include cholecystostomy or percutaneous aspiration.

Other modalities of treatment aim at dissolution of the gallstone and include systemic medical dissolution with Chenodeoxycholic acid and Ursodeoxycholic acid, contact dissolution with Methyl tert-butyl ether (for cholesterol stones) and lithotripsy.

CHOLEDOCHOLITHIASIS

The presence of a stone or multiple stone in the common bile duct is called Choledocholithiasis. As have been discussed earlier, stones in the biliary tree and in this case in the common biliary duct, the stones appear secondary to migration from the gallbladder. Primary Choledocholithiasis may form due to stasis or infection.

CLINICAL MANIFESTATIONS

The pain caused by a stone in the bile duct is very similar to that of biliary colic caused by impaction of a stone in the cystic duct, transient jaundice may be caused by a stone that temporarily impacts the ampulla but subsequently moves away, acting as a ball valve. A small stone may pass through the ampulla spontaneously with resolution of symptoms. Patients may manifest with cholangitis or gallstone pancreatitis.

INVESTIGATIONS

Elevation of serum bilirubin, alkaline phosphatase, and transaminases are commonly seen in patients with bile duct stones.

Commonly the first test, ultrasonography, is useful for documenting stones in the gallbladder (if they are still present), as well as determining the size of the common bile duct. A dilated common bile duct (>8 mm in diameter) on ultrasonography in a patient with gallstones, jaundice, and biliary pain is highly suggestive of common bile duct stones.

Using a side-viewing endoscope, the common bile duct can be cannulated and a cholangiogram performed using fluoroscopy after installing water soluble contrast; this procedure is called Endoscopic Retrograde Cholangiopancreatography (ERCP) (figure 4) This method provides a sensitive way of diagnosis along with the possibility of therapeutic intervention. Once the presence of stones is confirmed by the cholangiogram, the stone can be removed by a dormia basket or balloon catheter after performing sphincterotomy.

Magnetic resonance cholangiopancreatography (MRCP) uses magnetic resonance imaging to visualize the biliary and pancreatic ducts in a non-invasive manner. This procedure can be used to determine if gallstones are lodged in any of the ducts surrounding the gallbladder (PTC) involves cannulation of the biliary tree directly by introducing a water-soluble contrast by needle through the liver substance. It can provide drainage.

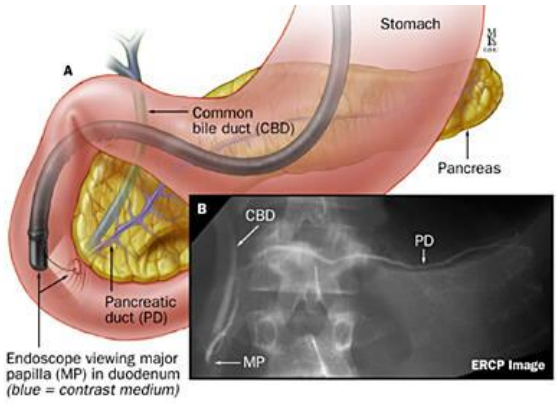


Figure 138 ERCP

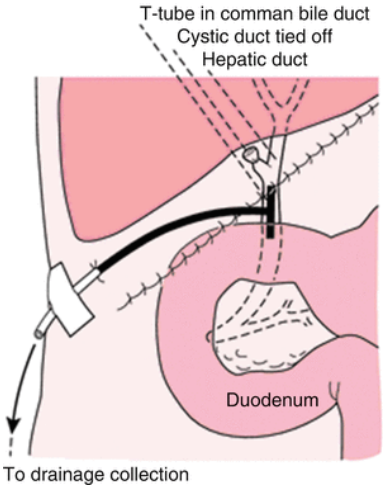


Figure 139. Percutaneous transhepatic cholangiography

TREATMENT

Endoscopic removal of stones has replaced all modalities of treatment. The need for cholecystectomy after endoscopic removal should be based on the presence of stones inside the gallbladder.

Exploration of the bile duct by laparotomy is undertaken much less often with the free availability of ERCP and sphincterotomy. At open surgery, if stones are present in the duct system, the common bile duct is opened longitudinally between stay sutures (choledochotomy) and the stones are extracted with forceps (Desjardins forceps) or a Fogarty balloon catheter. The opening in the common bile duct is closed around a T-tube, the long limb of which is brought out through a stab incision in the abdominal wall (figure 5). This serves as a safety valve to allow the escape of bile if there is a temporary obstruction to flow into the duodenum following duct exploration. It also facilitates the installation of iodine-containing dye to obtain a T-tube cholangiogram some 7-10 days following surgery. If this shows free flow of dye into the duodenum and no residual duct stories, the T-tube can be damned before removal.

CARCINOMA OF THE GALLBLADDER

This is a rare disease and represents about 3-4% of gastrointestinal cancers. The patients are usually female, old, in their sixties or seventies. The majority of cases are adenocarcinoma (90%).

ETIOLOGY

The Etiology of gallbladder cancer is unknown, but seems to be associated closely to gallstone disease; about 70-90% have gallstone disease. Infection may promote the development of cancer as the risk of carcinoma in typhoid carriers is significantly increased. The calcified "porcelain" gallbladder is associated with more than a 20% incidence of gallbladder carcinoma.

METHOD OF SPREAD

The tumor spreads by direct extension into the liver, seeding of the peritoneal cavity, lymphatic and hematogenous spread.

CLINICAL FEATURES

Signs and symptoms of carcinoma of the gallbladder include abdominal discomfort, right upper quadrant pain, nausea, and vomiting. Jaundice, weight loss, anorexia, ascites, and abdominal mass are less common presenting symptoms.

INVESTIGATIONS

The diagnosis is made on ultrasonography and defined by a multidetector row CT scan, with a percutaneous biopsy confirming the histological diagnosis. In selected patients, laparoscopy is useful in staging the disease, as it can detect peritoneal or liver metastases that would preclude further surgical resection. MRI can be used instead.

STAGING

Staging of gallbladder cancer is dependent on histopathological finding and after removing the gallbladder. The UICC staging of gallbladder cancer is based on the depth of invasion.

- Stage I: confined to the mucosa/submucosa.
- Stage II: involvement of the muscle layer.
- Stage III: serosa involvement.
- Stage IV: spread to the cystic node.
- Stage V: invasion of the liver and adjacent organs.

TREATMENT

Treatment of cancer of the gallbladder is surgical. For stages I-III, the best results are reported with cholecystectomy. Initially, the gallbladder' is removed and the diagnosis confirmed by frozen section. If positive and the tumor is stage I-III, a 3-cm resection of surrounding hepatic parenchyma is performed together with lymph node clearance. If the tumor is advanced. some advocate an aggressive approach with right lobectomy and regional node clearance.

Long-term survival is only very rarely encountered after aggressive major resections. Thus, even if respectable, patients with stage ll-IV disease are generally regarded as incurable in view of the uniformly poor prognosis.

In inoperable patients with jaundice and itching, palliation can be achieved by endoscopic/radiological stenting with a metal expanding endoprosthesis or surgically by round ligament segment III bypass. The response of gallbladder cancer to radiotherapy and chemotherapy is poor.

CARCINOMA OF THE BILE DUCTS

The prevalence of carcinomas of the biliary tract is less than that of gallbladder cancer. Contrary to gallbladder cancer, there is a slight preponderance of males. The age at presentation varies but the peak incidence is in the sixth decade.

ETIOLOGY

The Etiology of bile duct cancer is unknown. Bile duct carcinoma is very common in Far East countries where parasitic infestation is endemic *(*Clonorchis sinensis*).* The association with gallstones is much less marked than it is with carcinoma of the gallbladder, although ductal calculi are found in 20-50% of patients who develop cholangiocarcinoma. Bacterial induced endogenous carcinogens derived from bile salts have been implicated and their role is supported by the findings of some epidemiological studies and the higher incidence in typhoid carriers. Cholangiocarcinoma is seen with increasing frequency in certain clinical groups (see the table below).

Associations with bile duct carcinomas
• **Congenital hepatic fibrosis** • **Choledochal cyst** • **Ulcerative colitis** • **Sclerosing cholangitis**

PATHOLOGY

The tumors are best classified into anatomical site of origin.

• Intrahepatic; from the minor hepatic ducts (intrahepatic cholangiocarcinoma). This is discussed in the lecture on the liver.

• Proximal; from the right and left hepatic ducts, hilar confluence and proximal common hepatic duct (Klatskin tumors).

• Middle; from the distal common hepatic duct, cystic duct and its confluence with the common bile duct.

• Distal; from the distal common bile duct, ampullary and periampullary regions.

The gross appearance of extrahepatic cholangiocarcinoma assumes one of three forms: stricture (scirrhous variety), nodular or papillary. All cholangiocarcinoma is slow-growing, locally infiltrative but have a special predilection for perineural spread and do not metastasize beyond the liver.

CLINICAL FEATURES

The main presentation in patients with extrahepatic cholangiocarcinoma is obstructive jaundice, which is progressive and accompanied by itching

and anorexia. Weight loss is not evident until the disease is advanced. Dull upper abdominal pain is a frequent symptom. Some patients present acutely with cholangitis or acute cholecystitis. The duration of symptoms is usually short.

physical examination reveals hepatomegaly. Microcytic hypochromic anaemia is present in patients with papillary tumors at the lower end of the bile duct and in the periampullary region. The feces of these patients have a characteristic silvery appearance due to a combination of steatorrhea and altered blood. A palpable gallbladder is present in patients with distal tumors.

INVESTIGATIONS

CT does not permit sufficiently precise anatomical localization to predict the exact site and respectability of the tumours. The definitive investigation is MRCP. ERCP is indicated for ampullary and periampullary lesions.

MANAGEMENT

Resection (for both intrahepatic and extrahepatic lesions) is the best method of treatment and is indicated for all operable tumors in fit individuals. The percutaneous insertion of iridium-192 wire has been used to provide local irradiation (brachytherapy) with good results. Cholangiocarcinoma of the bile ducts are generally regarded to be unresponsive to chemotherapy. Chemotherapy, radiotherapy and stent insertions for palliation of jaundice are employed for inoperable patients.

SURGICAL JAUNDICE

Jaundice is yellowish discoloration of the skin and mucous membranes, is recognized clinically when serum bilirubin exceeds 50 µmol/L (3.0 mg/dL). it has been found that bilirubin has high affinity for elastin which is found abundantly in the skin and the sclera. Surgical jaundice is a term applied to refer to any form of surgically correctable jaundice. Because most of these occur due to obstruction of the biliary tree, the term is interchangeable with **obstructive jaundice.**

PHYSIOLOGY

Red blood cells are destroyed, mainly in the spleen and small amounts in other reticuloendothelial organs. The destruction is either due to aging or abnormality in shape, where by both of which can increase surface adhesion and entrapment inside splenic sinusoids. Splenic sinusoids have macrophages that engulf and destroy RBCs, thus releasing hemoglobin which is then broken down by an oxidation reaction into globular proteins (reused after destruction to amino acids) and the brown pigment heme. Heme is then oxidatively cleaved by the microsomal enzyme heme oxygenase with the end products being biliverdin, carbon monoxide, and iron, biliverdin is converted it to **bilirubin** by the cytosolic enzyme biliverdin reductase. Bilirubin formed in the reticuloendothelial cells is virtually insoluble in water. This form of bilirubin is termed unconjugated bilirubin. To be transported in blood, bilirubin must be solubilized. This is accomplished by its reversible, non-covalent binding to albumin. Unconjugated bilirubin bound to albumin is transported to the liver, where it, but not the albumin, is taken up by hepatocytes. . In the endoplasmic reticulum, bilirubin is solubilized by conjugation to glucuronic acid. The conjugation of glucuronic acid to bilirubin is catalyzed by bilirubin uridine-diphosphate (UDP) glucuronosyltransferase. The resultant conjugated bilirubin is then transported to the biliary system to be excreted.

The conjugated bilirubin excreted into bile drains into the duodenum and passes unchanged through the proximal small bowel. Conjugated bilirubin is not taken up by the intestinal mucosa. When the conjugated bilirubin reaches the distal ileum and colon, it is hydrolyzed to unconjugated bilirubin by bacterial ß- glucuronidases. The unconjugated bilirubin is reduced by normal gut bacteria to form a group of colorless tetrapyrroles called urobilinogens. About 80 to 90% of these products are excreted in feces, either unchanged or oxidized to orange derivatives called urobilins (e.g. strecobilin). The remaining 10 to 20% of the urobilinogens are passively absorbed, enter the portal venous blood, and are reexcreted by the liver. A small fraction (usually <3 mg/dL) escapes hepatic uptake, filters across the renal glomerulus, and is excreted in urine, this compound along with urochrome give urine its amber yellow color.

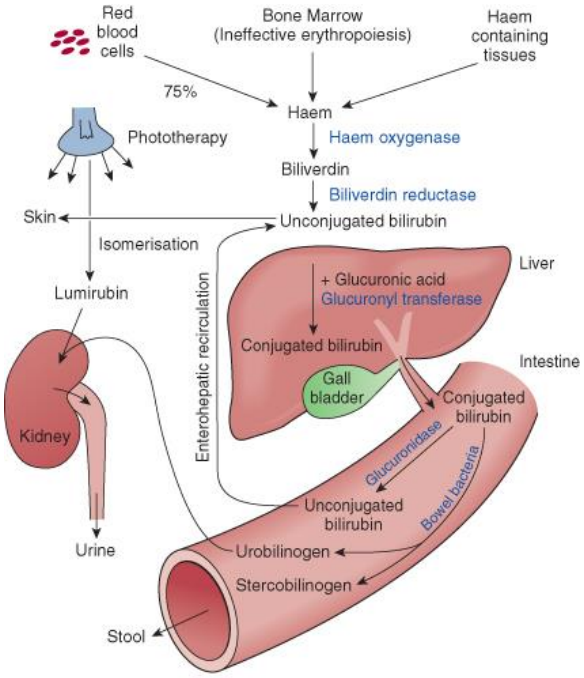


Figure 140 physiology of bilirubin metabolism

SERUM BILIRUBIN

The entities of bilirubin are based on the original van den Bergh reaction. In this assay, bilirubin is exposed to diazotized sulfanilic acid, splitting into two relatively stable compounds that absorb maximally at 540 nm, allowing for photometric analysis by spectroscopy. The direct fraction (termed direct bilirubin) is that which reacts with diazotized sulfanilic acid in the absence of an accelerator substance such as alcohol. *The direct fraction provides an approximate determination of the conjugated bilirubin in serum*. The total serum bilirubin is the

amount that reacts after the addition of alcohol. The indirect fraction (termed indirect bilirubin) is the difference between the total and the direct bilirubin and provides an estimate of the unconjugated bilirubin in serum.

With the van den Bergh method, the normal serum bilirubin concentration usually is 17 µmol/L (<1 mg/dL; each 17 µmol/L is the approximately the

equivalent of 1 mg/dl). However, Total serum bilirubin concentrations are between 3.4 and 15.4 µmol/L (0.2 and 0.9 mg/dL) in 95% of a normal population. Up to 30%, or 5.1 µmol/l (0.3 mg/dl), of the total may be direct-reacting (conjugated) bilirubin.

ETIOLOGY OF SURGICAL JAUNDICE

jaundice may result from excessive destruction of red cells (hemolytic jaundice or pre-hepatic jaundice), from failure to remove bilirubin from the blood stream (hepatocellular jaundice), or from obstruction to the flow of bile from the liver (cholestatic jaundice or posthepatic jaundice).

To the surgeon, the most important type of hemolytic jaundice is that caused by hereditary spherocytosis, in which splenectomy may be necessary. Hemolytic jaundice may also occur after blood transfusion and after operative or accidental trauma, when hematoma formation produces a pigment load that exceeds hepatic excretory capacity.

Hepatocellular jaundice is usually a medical rather than a surgical condition, although its recognition in patients presenting with abdominal pain is important, as surgical intervention may aggravate the hepatocellular injury.

Cholestatic jaundice due to intrahepatic obstruction of bile canaliculi may be a feature of acute and chronic liver disease and can be caused by drugs (e.g. chlorpromazine). This form of jaundice must be differentiated from that due to extrahepatic obstruction, the cause of which has most surgical relevance.

The causes for extrahepatic obstruction are classified as shown in the table on the side. It is important to note that the term cholestatic jaundice applies to both intrahepatic and extrahepatic obstruction, while the term obstructive jaundice is commonly correlated with extrahepatic biliary obstruction.

CLINICAL FEATURES OF SURGICAL JAUNDICE

The term obstructive or cholestatic jaundice is a clinical term that applies to specific features that merit special lines of assessment, obstructive jaundice is the result of impaired bile flow to the duodenum subsequent to the secretion of conjugated bilirubin into the bile canaliculi.

Causes of extrahepatic obstruction
Extramural
Carcinoma of the head of the pancreas
Periampullary tumors
Enlarged lymph nodes
Merizzi's syndrom
Intramural
Biliary atresia
iatrogenic strictures • Surgical • Radiotherapy
Inflammatory strictures
Trauma
Idiopathic
Intraluminal
Common bile duct stones
Parasitic infections • Ascaris lumbrocoides • Hydatid cysts
Biliary tree tumors • Cholangiocarcinoma • Papillomatosis

Obstructive jaundice is likely if there is a history of passage of dark urine (because of excretion of high amounts of conjugated bilirubin) and pale stools (deficiency in urobilins, usually referred to as clay-

colred stool), and if the patient complains of pruritus (owing to an inability to secrete bile salts into the obstructed biliary system, which accumulate in the skin and induce the release of histamine-a pruritic substance from mast cells).

Distinction among the types of jaundice			
	hemolytic	hepatocellular	cholestatic
Stool	Normal	Normal	Pale
urine	Normal	Dark	Dark
Bilirubin	Indirect	Mixed	Direct

EVALUATION

After the patient is suspected with obstructive jaundice, further assessment with history, examination and investigations is important. The aim, is to identify the etiology and to rule out hemolytic, cholestatic and intrahepatic causes of jaundice which have minor surgical relevance.

HISTORY

History as a tool is very important in diagnosing the cause of obstructing jaundice. The table below, presents the clinical scenarios that involves jaundice and with which clinical diagnosis they correlate.

After the exclusion of hemolytic and hepatocellular jaundice during advanced stages of assessment, the differentiation between intrahepatic and extrahepatic cholestatic jaundice is of value. Intrahepatic cholestasis is a consideration if the patient reports use of estrogens, phenothiazrnes, and other drugs that can cause cholestasis.

PHYSICAL EXAMINATION

Jaundice is examined under day light with good exposure. It can be seen on the skin, but is better illustrated on the sclera and the mucous membrane of the conjunctiva. General examination should focus on the presence of wasting associated with weight loss, the presence of lymphadenopathy (look for supraclavicular nodes; troiser's sign) and bruising (secondary to vitamin k deficiency).

differential of the common causes of obstructive jaundice	
Clinical diagnosis	**Scenario**
Biliary stones	Female, middle aged with *Recurrent* or *transient* jaundice with abdominal pain and fatty dyspepsia. There is history of gallbladder stones. There might be an associated cholangitis or pancreatitis.
Biliary tumors	Old man with *progressive* jaundice, pruritus and abdominal discomfort.
Carcinoma of the head of the pancreas	Old man with *progressive* jaundice that is *painless* (i.e. not following the course of jaundice).
Periampullary tumor	recurrent jaundice in a middle, aged man. Associated with anorexia or weight loss.
Iatrogenic strictures	Constant jaundice with history of recent cholycystectomy, gastrectomy, hepatic resection, liver transplant or radiotherapy
Inflammatory strictures	Constant jaundice with history of debilitating toxic episode.

Most commonly Patients have stable vital signs, patients with ascending cholangitis may have high fever.

A full abdominal examination is very important, it can illustrate stigmata of liver disease and detect splenomegaly seen in medical jaundice, in the context of a clinical obstructive jaundice, tenderness in the right lower quadrant is looked for. The same goes for masses. Marked hepatic enlargement (≥6 cm below the inferior costal margin) occurs in some instances of extrahepatic obstruction.

Palpable gallbladders should also be palpated. Because fibrosed gallbladders that contain stones cannot distend when pressure increases in the obstructed biliary tree, Courvoisier's law states that if the gallbladder is palpable in the presence of jaundice, the jaundice is unlikely to be due to stone. In other terms, *any obstructive jaundice with a palpable gallbladder is cancer of the head of the pancreas until proven otherwise.* This because this type of cancer is the most common with this presentation.

Ascites is also important as it might be caused by an underlying malignancy, but is seen in the context of hepatocellular jaundice.

INVESTIGATIONS

A complete blood count is routine and is important to rule out anemia and leukocytosis. with unexplained jaundice, there are a battery of tests that are helpful in the initial evaluation. These include total and direct serum bilirubin, aminotransferases, alkaline phosphatase, albumin, and prothrombin time tests. Enzyme tests (alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase) are helpful in differentiating between a hepatocellular process and *a* cholestatic process. Patients with a hepatocellular process generally has a disproportionate rise in the aminotransferases compared to the alkaline phosphatase. Patients with a cholestatic process have a disproportionate rise in the alkaline phosphatase compared to the aminotransferases.

It is important to order an albumin level and a prothrombin time, to assess liver function. An elevated prothrombin time indicates either vitamin K deficiency due to prolonged jaundice and malabsorption of vitamin K (due to deficient bile acids which are important for absorption of fat soluble vitamins) or significant hepatocellular dysfunction. The failure of the prothrombin time to correct (within 36 hours) with parenteral administration of vitamin K indicates Sever hepatocellular injury.

When the disease is established to be cholestatic in nature, the next step is to determine whether it is intra- or extrahepatic cholestasis, the next appropriate test is an ultrasound. The ultrasound is inexpensive and can detect dilation of the intra- and extrahepatic biliary tree with a high degree of sensitivity (97%) and specificity. Intrahepatic tree diameter is up 2 mm normal, while it is 8 mm in extrahepatic tree. The *absence of biliary dilatation suggests intrahepatic cholestasis,* while the presence of biliary dilatation indicates extrahepatic cholestasis. Ultrasound will also detect gallstones but the sensitivity to detect stones in the common bile duct is much lower (25%; because it is surrounded by gas containing bowel).

Although ultrasonography may indicate extrahepatic cholestasis, it rarely identifies the site or cause of

obstruction. The distal common bile duct is a particularly difficult area to visualize by ultrasound because of overlying bowel gas. Appropriate next tests include computed tomography (CT) and endoscopic retrograde cholangiopancreatography (ERCP). CT scanning is better than ultrasonography for assessing the head of the pancreas and for identifying choledocholithiasis in the distal common bile duct, particularly when the ducts are not dilated.

ERCP is the "gold standard" for identifying choledocholithiasis. It is performed by introducing a side viewing endoscope perorally into the duodenum. The ampulla of Vater is visualized and a catheter is advanced through the ampulla (figure 2). Injection of dye allows for the visualization of the common bile duct and the pancreatic duct. Beyond its diagnostic capabilities, ERCP allows for therapeutic interventions, including the removal of common bile duct stones (by a **dormia** basket or balloon catheter after performing **sphincterotomy),** biopsy of periampullary tumors, and relief of obstructive jaundice by the placement of stents. The investigation may be complicated by acute pancreatitis, and prophylactic antibiotics should be

administered to reduce the risk of cholangitis. Hemorrhage and perforation are less frequent complications.

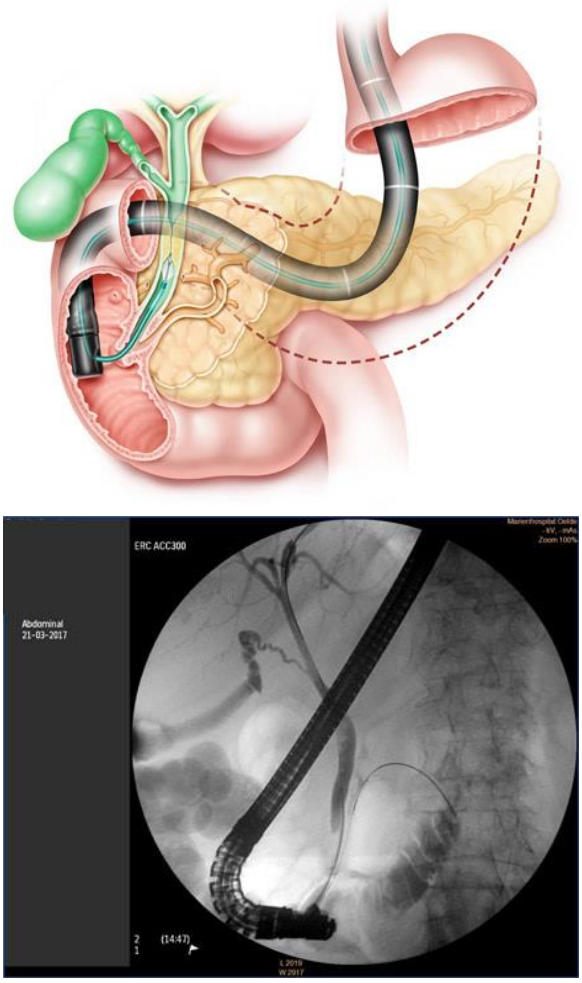


Figure 141 Endoscopic retrograde cholangiopancreatography (ERCP) *A.* A schematic picture showing the side-viewing endoscope in the duodenum and a catheter in the common bile duct. *B.* An endoscopic cholangiography showing stones in the common bile duct. The catheter has been placed in the ampulla of vater .

In patients in whom ERCP is unsuccessful, Percutaneous transhepatic cholangiography (PTC) can provide the same information. It provides a clear outline of the biliary system by the injection of contrast through a slim flexible needle passed subcutaneously into the liver. The technique may cause bleeding or bile leakage and can be complicated by bacteremia and septicemia; coagulation status must be checked and antibiotic cover should be given.

Magnetic resonance cholangiopancreatography is a rapidly developing, noninvasive technique for imaging the bile and pancreatic ducts. It can provide an outline of biliary anatomy and can distinguish between malignant and calculous obstruction, this may replace ERCP as the initial diagnostic test in cases where the need for intervention is felt to be small.

MANAGEMENT

Derangements, including fluid loss should be corrected along with administration of ora! or parental vitamen k, as conservative treatment or prior to the operation. Antibiotics are given as prophylaxis against ascending cholangitis and resultant pyogenic abscess. Preoperative and postoperative mannitol is given to prevent hepatorenal shutdown by bilirubin pigments (accumulating at the renal tubules and causing acute tubular necrosis).

Relief of obstruction can be achieved by removal of the cause. In the case of stones, ERCP with sphincterotomy is curative. Exploration of the bile duct by laprotomy is undertaken much less often with the free availability of ERCP and sphincterotomy.

Any injury in common bile duct may cause bile leak and treat by using T-tube to form fibrosis and remove it after 6 weeks

Resection and anastomosis is needed for short strictures, but stent insertion after intraoperative diagnosis with ERCP can relief obstruction in both short and long strictures.

Carcinoma of the head of the pancreas is treated with the appropriate modality (see the lecture on that subject). The relief of jaundice and pruritus can be achieved depending on the stage of the tumor and the intent of treatment. With curative intent, whippl's procedure is standard, while with palliative surgery, cholecystjejunostomy or choledochojejunostomy is sufficient to bypass the obstruction.

Other tumors and other causes in general might require laparotomy or laparoscopy, with the appropriate indication.

THE SMALL INTESTINES

The small intestine (small bowel) is a tubular structure, with an estimated median length of 6 meters in adults, that consists of three segments lying in series: the duodenum, the jejunum, and the ileum. The anatomy of the duodenum had been discussed in previous lectures. The duodenum is demarcated from the jejunum by the ligament of Treitz.

The jejunum represents the proximal two-fifths. It is mostly in the left upper quadrant of the abdomen and is larger in diameter and has a thicker wall than the ileum. The ileum makes up the distal three-fifths of the small intestine and is mostly in the right lower quadrant. Compared to the jejunum, the ileum has thinner walls. No distinct anatomic landmark demarcates the jejunum from the ileum.

Features which are more characteristic of the proximal than distal small intestine include larger circumference, thicker wall, less fatty mesentery, and longer vasa recta (see later). Both the jejunum and ileum are attached to the retroperitoneum by a broad-based mesentery.

BLOOD SUPPLY AND LYMPHATICS

The distal duodenum, the jejunum, and the ileum derive their arterial blood from the superior mesenteric artery. The branches leave the main trunk of the artery, pass between two layers of the mesentery, and form anastomosing **arcades** as they pass outward to supply the small intestine.

There may be single and then double arcades in the area of the jejunum, with a continued increase in the number of arcades moving into and through the area of the ileum. Extending from the terminal arcade are **vasa recta (straight arteries)** which provide the final direct vascular supply to the walls of the small intestine. The vasa recta of the jejunum tend to be longer.

The venous drainage of the small intestines occurs via the superior mesenteric vein. Lymph drainage occurs through lymphatic vessels coursing parallel to corresponding arteries. This lymph drains through mesenteric lymph nodes to the cisterna chyli; then through the thoracic duct, and ultimately into the left subclavian vein.

INNERVATION

The parasympathetic and sympathetic innervation of the small intestine is derived from the vagus and splanchnic nerves, respectively.

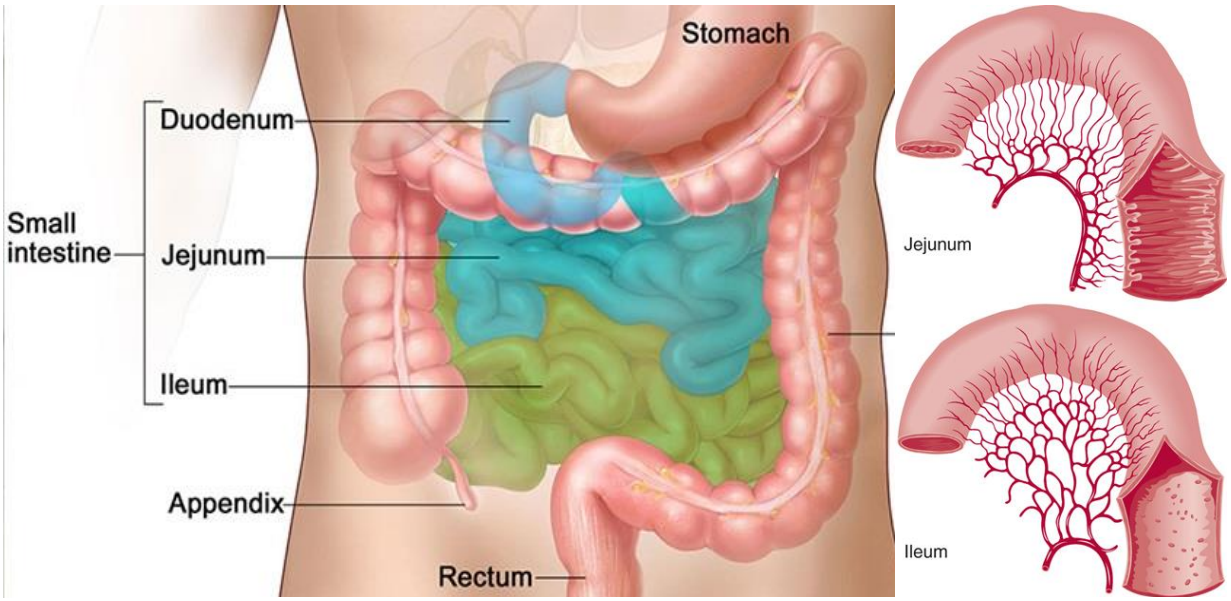


Figure 142 left; anatomy of the intestines, right; arterial arcades and vasa recta supplying the jejunum (A) and ileum (B)

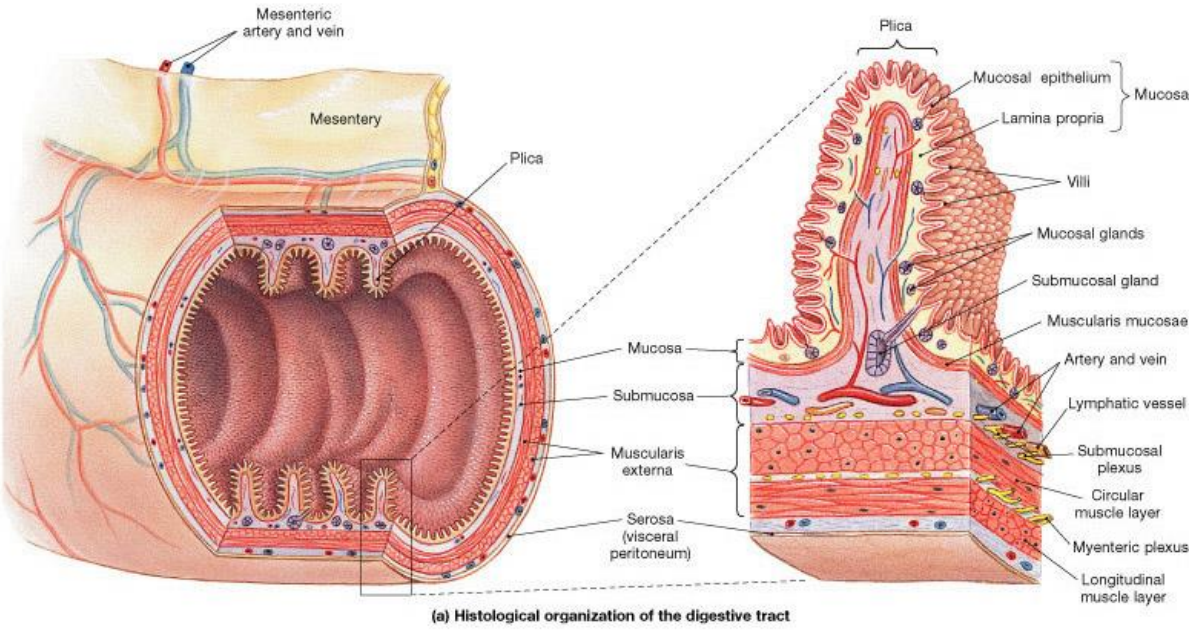


Figure 143 histology of the small intestine

HISTOLOGY AND PHYSIOLOGY

The small intestines has a mucosa, (a muscularis mucosa) submucosa, a muscularis externa and a serosa.

The small intestinal mucosa is organized into mucosal folds known as plicae circulares or valvulae connivances (figure 2) that are visible upon gross inspection. These folds are also visible radiographically and help to distinguish between small intestine and colon (which does not contain them) on abdominal radiographs (figure 3). These folds are more prominent in the proximal intestine than in the distal small intestine.

The small intestine plays the key role in the digestion and absorption of nutrients. Ninety percent of nutrient absorption occurs in the small intestine, and most of the rest occurs in the large intestine. The arrangement of the intestines into plicae and villi increases the total area for absorption by a factor of more than 600, to approximately 2 million cm2.

The intestinal mucosa is further suborganized into villi and crypts. Villi are finger-like projections of epithelium and underlying lamina propria that contain blood and lymphatic (lacteals) vessels that extend into the intestinal lumen. the crypts are located at the base of the villi and extend into the lamina propria of the intestinal mucosa. There function is yet to be identified but it is known to secret enzymes and help in the regeneration of the mucosal epithelium.

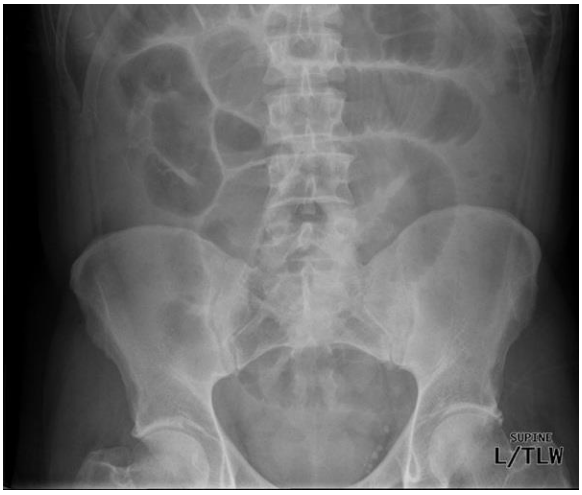


Figure 144 appearance of plicae circulares on a plan abdominal radiograph.

CROHN'S DISEASE

Crohn's disease (or regional enteritis) is a chronic, idiopathic inflammatory disease with a propensity to affect the distal ileum, although any part of the alimentary tract can be involved. It is most

common in North America and northern Europe with an incidence of 5 per 100 000 with women and men being equally affected, but is most commonly diagnosed in young patients during the second and third decades.

PATHOLOGY

This disease may affect any level of the alimentary tract, from mouth to anus, but most commonly located at the terminal ileum. In 50% of cases both small and large intestines are involved, whereas in 25% of cases, the large intestine alone is affected.

When fully developed, Crohn's disease is characterized by sharply delimited and typically transmural involvement of the bowel by an inflammatory process with mucosal damage, the presence of non-caseating granulomas in 40% to 60% of cases and fissuring or ulcers. Edema in the mucosa between the ulcers gives rise to a cobblestone appearance.

Grossly, the affected loops are dull red with areas of thick gray-white exudates and this occurs due to involvement of all intestinal structures (earlier, trans-mural involvement). As a result, the lumen is almost always narrowed (figure 5). Longstanding inflammation leads to progressive fibrosis of the thickened bowel wall, which encroaches on the lumen, producing elongated strictures. The mesenteric lymph nodes are usually enlarged.

A classic feature of Crohn's disease is the sharp demarcation of diseased bowel segments from adjacent uninvolved bowel. When several bowel segments are involved, the intervening bowel is essentially normal (“skip" lesions). Another feature of Crohn's disease that is grossly evident and helpful in identifying affected segments of intestine during surgery is the presence of fat wrapping (the encroachment of mesenteric fat onto the serosal surface of the bowel, figure 5).

ETIOLOGY

There is little doubt that genetic factors are important in the occurrence of crohn's disease. Much recent interest has focused on associations of the disease with . A gene called ***NOD2*** (or ***CARDIS).*** This gene is mutated in as many as 25%ofCrohn's disease patients in some ethnic populations. This

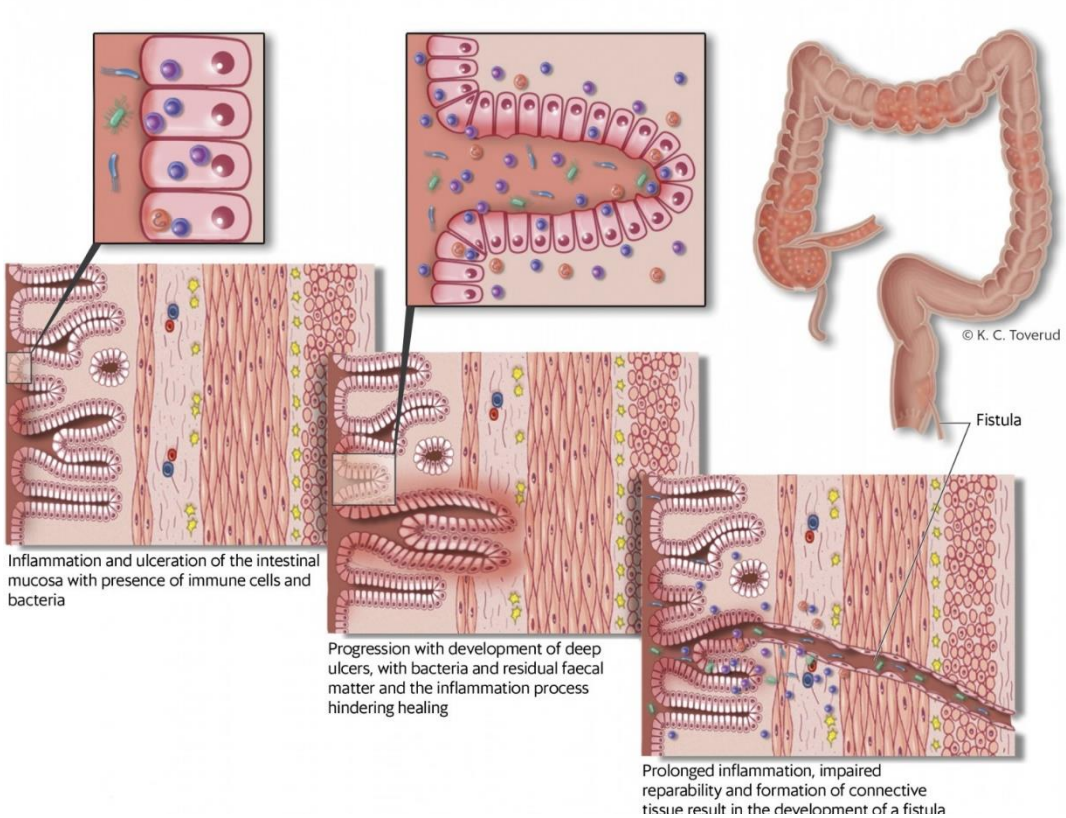
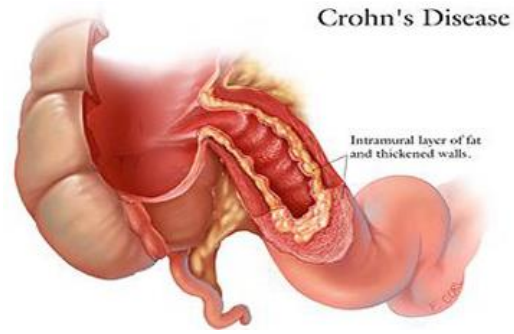


Figure 145 pathological changes in crohn's disease

gene is expressed in Paneth cells (which are found at the depth of the intestinal crypts) and allows identification of certain peptides on the bacterial cell wall. It might be that the mutated gene allows for chronic inflammation against intestinal bacteria to occur in a NOD2 independent pathway.



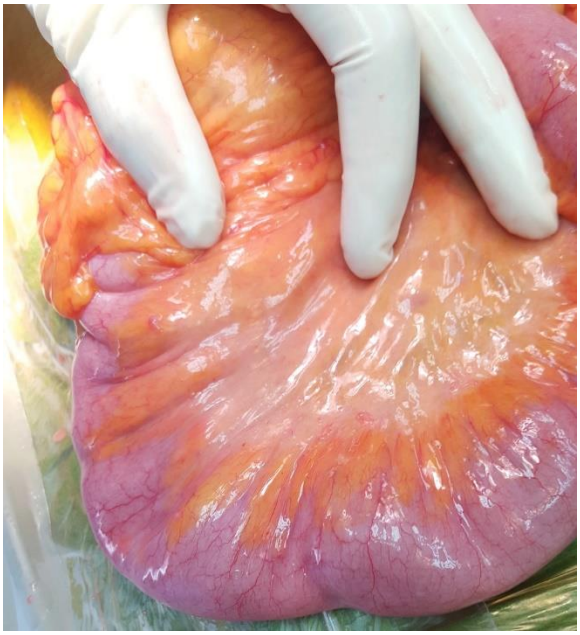


Figure 146 A-Gross appearance of crohn's ileitis B-This intraoperative photograph demonstrates encroachment of mesenteric fat onto the serosal surface of the Intestine (fat wrapping).

CLINICAL FEATURES

Crohn's disease is a Chronic disorder with exacerbations, remissions and a varied clinical presentation. The most common presentation during an exacerbation is intermittent colicky abdominal pain that may mimic the pain of appendicitis. Diarrhea is the second most common symptom and is usually less distressing and less likely to contain. blood.

Other characteristic symptoms of Crohn's disease include weight loss and general malaise.

Examination may reveal a low-grade fever and generalized wasting. On abdominal examination, Tenderness is noticed and an inflammatory mass may be felt, usually at the right iliac fossa and is caused by wrap- ping of the mesentery around the affected bowel loop.

Extra-intestinal manifestations may be present such as eye (iritis, uveitis), joints (polyarthritis, ankylosing spondylitis), skin (erythema nodosum (figure 6)), liver disease (sclerosing cholangitis), pancreas (pancreatitis) and kidney (obstructive uropathy with attendant nephrolithiasis) manifestations.

Crohn's anal disease is discussed in the lecture on anorectal conditions.

COMPLICATIONS

- Intestinal obstruction; intestinal obstruction, may occur due to active disease (edema and narrowing), chronic disease (fibrosis and strictures) or adhesions from previous surgical intervention.
- Fistulae; Fistula formation may occur due to deep ulceration. The fistula may communicate with the peritoneum, other loops of bowel, other viscera (e.g. bladder, vagina) or the skin.
- Hemorrhage; Rare but devastating event is massive intestinal bleeding.
- abdominal abscesses or peritonitis may occur secondary to sub clinical bowel perforation.
- Malignancy; the increased risk for carcinoma is significant.

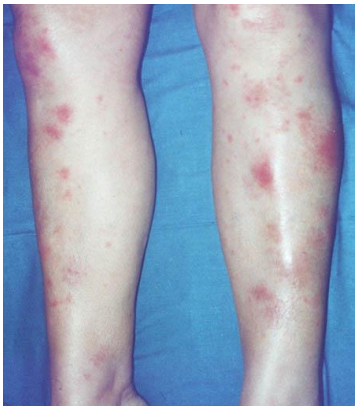


Figure 147 appearance of erythema nodosum in a patient with crohn's disease.

INVESTIGATIONS

The diagnosis of Crohn's disease sometimes can be challenging, it may not be possible to diagnose Crohn's with complete certainty.

The diagnosis of crohn's disease should be based on the clinical features (abdominal pain, diarrhea and weight loss) and the use of the following investigations;

- Barium follow through or enema; at earlier stages of the disease, multiple aphthous ulcers can usually be detected. Later on, networks of ulcers and fissures leading to the typical "cobblestone appearance" of the mucosa may appear. marked narrowing of the terminal ileum from a combination of edema, and (sometimes, but not always) fibrosis; frequently associated with proximal dilatation (string sign of Kantor) is also evident in many cases.
- Ultrasonography and CT scanning may reveal intra-abdominal abscesses and is useful in acute presentations to rule out the presence of other intra-abdominal disorders.
- Proctoscopy, sigmoidoscopy and colonoscopy can be used to detect lesions in the large intestines and Biopsy may reveal occult large intestinal involvement. Esophagogastroduodenoscopy (EGD) is done for disease of the proximal alimentary tract.

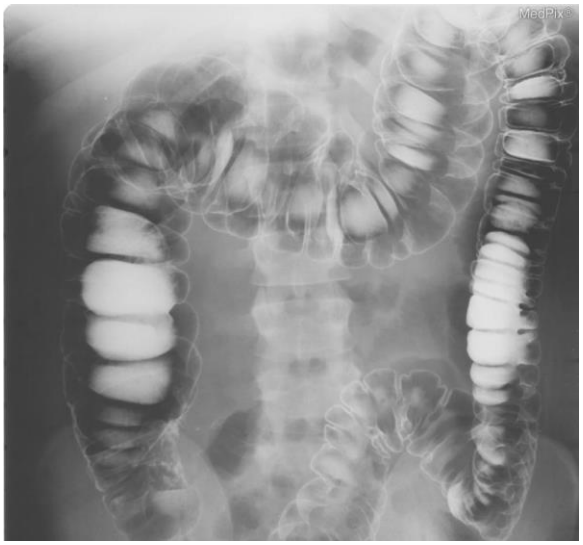


Figure 148 The double contrast barium enema shows loss of the lumen distensibility and a "cobblestone pattern".

MANAGEMENT

There is ho treatment modality that can cure crohn's disease. The aim of medical therapy is relief symptoms and induce remission while surgical therapy is palliative.

MEDICAL THERAPY

The following drugs are used in the treatment of crohn's disease;

- Sulfasalazine; this drug is a combination of 5-**aminosalicylic acid (5-ASA)** and a carrier, **sulfopy**ridine is useful in ulcerative colitis and large bowel Crohn's disease since the active ingredient is released by colonic bacteria. These drugs inhibit the synthesis of inflammatory mediators such as Eicosanoids. The therapeutic effect is achieved by the compound 5-ASA. They are given in a dose of 500 mg every 6 hours daily.
- Steroids can be used in the acute phase (prednisolone 40 mg daily by mouth), but every attempt should be made to avoid long-term steroid therapy in view of the risk of complications.
- Other **immunosuppressant drugs** (e.g. azathioprine, methotrexate, 6-mercaptopurine) are generally indicated when remission cannot be induced with sulfasalazine or steroids or when the disease is severe and multi systemic.
- Antibiotics; are used to treat and prevent infections with gram negative bacteria and include ciprofloxacin or metronidazole.

SURGICAL THERAPY

Indications for surgical therapy in crohn's disease are shown in the table below.

Indications for surgical therapy in crohn's disease
• **Recurrent intestinal obstruction**
• **Bleeding**
• **Perforation**
• **intestinal fistula**
• **malignant changes**

The choice of operation depends on the site and extent of disease. If the disease is limited, resection of

the diseased segment with a small margin of normal tissue may be performed, followed by wide side to side anastomosis. Surgery for multiple small bowel strictures now involves stricturoplasty of each lesion, a technique of enlarging the lumen of diseased bowel- without losing potential absorptive length.

TUBERCULOUS ENTERITIS

Tuberculous enteritis is disease of the intestines that is caused by Infection by Mycobacterium species. Primary tuberculous enteritis, is rare, however, a significant number of patients with pulmonary tuberculosis develop secondary intestinal involvement.

ETIOLOGY

The organism is Mycobacterium tuberculosis. secondary tuberculous enteritis occurs When a patient with pulmonary tuberculosis swallows infected sputurn, Hematogenous spread from active pulmonary or miliary, tuberculosis or the spread of the organism from a contagious organ, Primary tuberculous enteritis occur with the Ingestion of contaminated milk or food.

PATHOLOGY

The commonest site of involvement is the cecum and ileocecal area, and about 87% of patients with tuberculous enteritis have lesions in this area. The next commonest site is the ileum.

The ulcerative type occurs when the virulence of the organism is greater than the host defense. This is seen more frequently in secondary tuberculous enteritis. Once the organism gain entrance to the intestines, it colonises the lymphatics causing transverse ulcers with typical undermined edges. The serosa is usually studded with tubercles. Histology shows caseating granuloma .with giant cells (figure 8). This occurs in about 60% of the cases.

The other variety, called the hypertrophic type, occurs when host resistance is stronger than the virulence of the organism. It is caused by the drinking of infected milk. There is a marked inflammatory reaction causing hyperplasia and thickening of the terminal ileum because of its abundance of lymphoid follicles, thus causing narrowing of the lumen and obstruction and there may be marked mesenteric lymphadenopathy (figure 8). This type exists in 10% of cases.

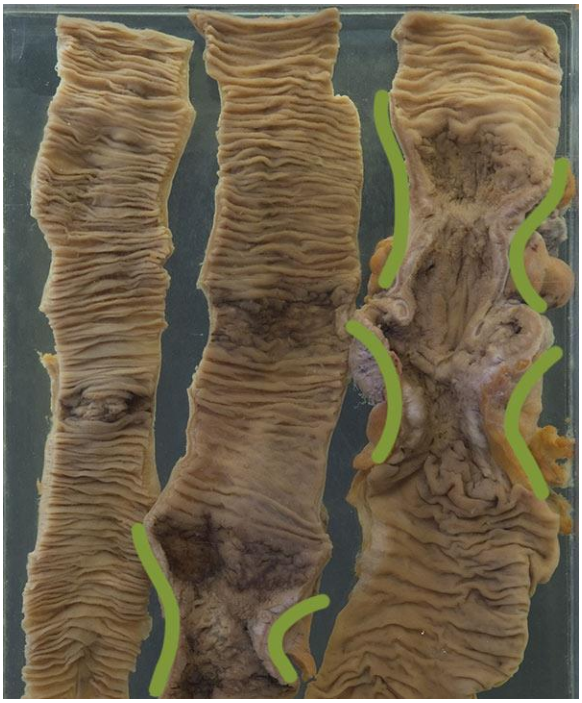


Figure 149 Tuberculous enteritis

An Ulcerohypertrophic type exists in 30% of the cases and is characterized by an inflammatory mass centring around the ileocecal valve with thickened and ulcerated intestinal walls.

CLINICAL FEATURES

Patients can present with subacute intestinal obstruction as is seen in all the pathological varieties of this disease. Diarrhea and abdominal pain, can be evident earlier. When the disease is located at ileocolic region, a right iliac fossa mass can be felt and is caused by inflammation of the intestinal wall and wrapping with the mesentery. This is especially seen in the ulcerohypertrophic type.

If extensive areas of the intestines is involved, malabsorption with resultant weight loss, anemia and other manifestations may become evident.

Patient may also present with symptoms of the original infection such as cough, sputum, hemoptysis, weight loss and night sweats.

INVESTIGATIONS

Raised ESR and CRP, low hemoglobin and a positive Mantoux test are usual. A plain abdominal radiograph is useful in eliciting the calcified mesenteric lymph nodes. A barium follow-through (or small bowel enema) shows strictures of the small bowel, particularly the ileum, typically with a high subhepatic cecum with the narrow ileum entering the cecum directly from below upwards in a straight line rather than at an angle.

Ultrasound and CT scanning are used to visualize the inflammatory mass, the mesenteric lymph nodes and to rule out any other suspected pathology. Laparoscopy reveals the typical picture of tubercles on the bowel serosa, multiple strictures, a high cecum, enlarged lymph nodes, areas of caseation and ascites. It also allows biopsy of the affected lymph nodes and affected tissues.

Other investigations for detecting the primary disease can also be useful and sometimes are already taken. They include a chest X-ray, sputum culture and microscopy and Mantoux test.

DIFFERENTIAL DIAGNOSIS

Because of the weight loss, intestinal obstruction and the right iliac fossa mass, tuberculous enteritis is often confused with colonic cancer or crohn's disease. presentation in an endemic area and colonoscopy with biopsy may aid in the diagnosis.

COMPLICATIONS

Intestinal obstruction may result from hypertrophy of the lymphoid follicles or strictures from massive ulceration seen in the ulcerative type. Bowel perforation usually follows the ulcerative type but can be seen in the hypertrophic type if there is longstanding complete obstruction. This usually results in tuberculous peritonitis.

If the inflammation involves the wail of the intestines up to the serosa, inflammation and ulceration can result in the formation of enteric fistulas.

TREATMENT

Treatment with antituberculotic antibiotics (Isoniazid, rifampicin, pyrazinamide, ethambutol) is of utmost importance to prevent progression of the disease and it complication. Surgical intervention is needed when there is obstruction, perforation, peritonitis and fistula formation.

MESENTERIC BOWEL ISCHEMIA

Mesenteric bowel ischemia or ischemic bowel disease is a disease caused by insufficient blood supply to the intestines resulting in infarction and death of the intestinal loops with concomitant life-threatening complications.

ETIOLOGY

Occlusion of the mesenteric arteries may be either thrombotic or embolic Thrombotic lesions usually start at the origin of a major artery (celiac and superior mesenteric artery), resulting in complete or partial obstruction that cannot be overcome by arterial anastomosis of arcades. Thrombotic mesenteric occlusion usually occurs following atheromatous narrowing but can be caused by vasculitis or arterial dissection. Thrombosis may also affect the arterial branches.

Embolic occlusion usually occurs at the arterial branches allowing arterial arcades to partially compensate for the low perfusion. Thus, ischemia is usually limited. Emboli may block major branches. Embolic occlusion is mostly of cardiac origin usually following a heart disease (atrial fibrillation, endocarditis, valve disease).

Intestinal ischemia may also follow systemic hypoperfusion (i.e. shock) or secondary to ingestion of vasoconstrictive drugs (e.g. digitalis). Mesenteric venous thrombosis is a less frequent cause of vascular compromise. Predisposing factors for developing mesenteric ischemia.

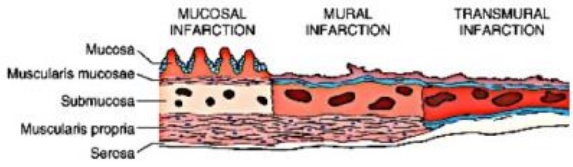


Figure 150 mesenteric ischemia according to severity

Mesenteric ischemia risk factors

- **age greater than 50 years**
- **Congestive heart failure, Digitalis therapy**
- **Recent transmural myocardial infarction**
- **Cardiac arrythmias especially atrial fibrillation**
- **Hypercoagulable state**
- **Hypovolaemia with hypoperfusion**

PATHOLOGY

As illustrated in Figure 11, the severity of injury ranges from transmural infarction of the gut, involving all visceral layers, to mural infarction of the mucosa and submucosa, sparing the muscular wall, to mucosal infarction,) if the lesion extends no deeper than the muscularis mucosae. Almost always, transmural infarction is caused by acute occlusion of a major mesenteric artery.

The ischemic injury usually begins in the mucosa and extends outward; an early event is sloughing of the epithelium from the tip of the villi and then downwards. This is followed by the formation of a membrane formed of necrotic debris, fibrin, leukocytes and bacteria. This is followed by marked submucosal edema and interstitial hemorrhage. If the ischemia is transmural, within 18 to 24 hours a thin, fibrinous exudate over the serosa will appear.

Within 24 hours intestinal bacteria produce outright gangrene and sometimes perforation of the bowel. The gangrene and necrosis extends to outside (mucosal, then mural, then transmural) according to the severity of occlusion.

CLINICAL FEATURES

Medical history may be significant for presence of, stroke, myocardial infarction, or peripheral artery disease. In cases of chronic mesenteric ischemia; Patients may present with a long history of weight loss, post- prandial pain (mesenteric angina), and phagophobia (fear of eating because of pain), the patient develops severe acute unrelenting central abdominal pain 15- 30 minutes after eating, which may be associated with diarrhea and vomiting. The patient may also notice passing frank blood in the stool.

Acute mesenteric ischemia most likely follows an embolic occlusion of the superior mesenteric artery and the patient usually presents with sudden onset of excruciating abdominal pain, collapse, bloody diarrhea and peritonitis.

Physical findings are usually minimal; thus, ischemic pain is suspected when there is Abdominal pain out of proportion to physical findings. When signs of peritonitis are present, then bowel perforation is the most likely cause.

INVESTIGATIONS AND DIAGNOSIS

- Blood tests; Complete blood count (CBC) may re- veal haemoconcentration and leucocytosis. Metabolic acidosis develops as a result of anaerobic metabolism. Elevated serum amylase and lactate levels are nonspecific findings.
- Abdominal radiographs; Plain abdominal radiographs may provide helpful information to exclude other abdominal pathologies. in a third of patients it may show portal venous gas, pneumatosis intestinalis and pneumoperitoneum.
- Duplex scanning; this investigation evaluates the perfusion inside the mesenteric artery. It is further explained in the 'vascular' surgery lectures.
- CT scan; this has a relatively high sensitivity and specificity in diagnosing mesenteric ischemia. Findings on CT scan are shown in the table below.

CT scan findings in mesenteric bowel ischemia

- Site of mesenteric artery occlusion
- Bowel wall thickening from edema or hemorrhage
- Lack of enhancement indicates infarction
- The presence of intestinal pneumatosis (submucosal gas)
- The presence of portal venous gas (caused by invasion with gas producing bacteria).

- Angiography; The definitive diagnosis of mesenteric thrombosis is made by mesenteric arteriography. It typically shows occlusion or near occlusion of the celiac and superior mesenteric trunks at or near their origins from the aorta.

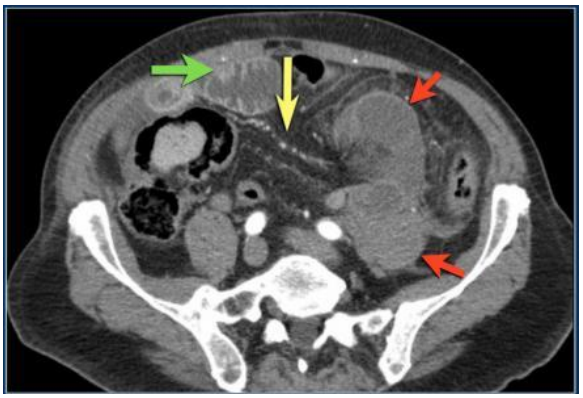


Figure 151 Increased venous pressure in strangulation also leads to engorgement of veins (yellow arrow).This patient also has a closed loop obstruction with gray enhancement pattern of the strangulated bowel loops (red arrows).Notice the normal enhancement of small bowel proximal to the obstruction (green arrow).

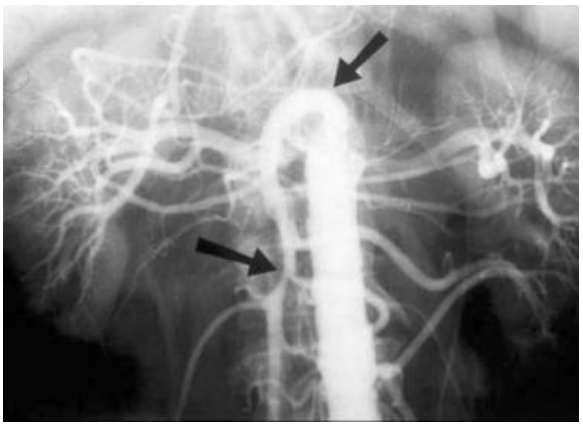


Figure 152 Lateral aortogram demonstrating normal origins of celiac and superior mesenteric arteries.

MANAGEMENT

PREOPERATIVE MANAGEMENT

Acute mesenteric artery thrombosis cannot be cured medically. If vasospasm is observed on arteriogram, intraarterial **papaverine** (opium alkaloid antispasmodic) may be started to improve flow via the the arteriogram catheter. anticoagulation therapy should be commenced immediately upon diagnosis. intravenous fluids to correct dehydration, acid-base abnormalities are very important.

Preoperatively, All patients should receive broad-spectrum antibiotic therapy. 4 units of packed red blood cells should be typed and crossmatched before patient enters the theater.

OPERATIVE MANAGEMENT

Surgical treatment involves exploratory laparotomy, followed by identification of the involved artery and bowel. To assess the viability of the bowel during direct observation, look for peristalsis, observe the color of the bowel (i.e. pink and healthy vs dusky red and edematous), the presence or absence of palpable pulses, and the use of Intraoperative Doppler ultrasound of the bowel. The dead bowel should be resected and anastomosis attempted, if extent of bowel viability is uncertain, a second look laparotomy 12-24 hours later Is mandatory.

Once the affected artery is identified, it is opened and endarterectomy (for thrombotic causes) or embolectomy, (for embolic occlusion) is performed. Bypass techniques may also be performed using prosthetic grafts.

POST-OPERATIVE MANAGEMENT

This should include close monitoring of blood pressure and hemoglobin level to evaluate for sepsis or hemorrhage. Patients should continue to have postoperative heparin anticoagulation therapy in order to reduce thrombotic events.

SMALL INTESTINAL NEOPLASMS

Despite comprising 75% of the length and 90% of the surface area of the gastrointestinal tract, the small bowel harbors relatively few primary neoplasms accounting for 5% of all gastrointestinal neoplasms and fewer than 2% of gastrointestinal malignancies.

Factors suggested to explain the scarcity of small bowel lesions and the infrequency of their malignant transformation are shown in the table below.

Causes for low frequency of neoplasms in the small intestines

- **Rapid Transit of contents fluidity**
- **High turnover rate of epithelial cells.**
- **Alkalinity of small intestinal Media**
- **High level of IgA**
- **High level of benzyl peroxidase(detoxify potential carcinogen)**

BENIGN TUMORS

Benign tumors of the small intestines are shown in the table below. Benign tumors are slowly growing tumors and thus are rarely symptomatic and so the true incidence is unknown. Symptoms may arise as a result of intussusception or bleeding, particularly in the case of leiomyoma.

Benign tumors of the small intestines

Relatively common	Relatively rare
• **adenoma**	• fibromas
• **leiomyoma**	• fibromyomas
• **lipoma**	• neurofibromas
	• ganglioneuromas
	• hemangiomas

MALIGNANT TUMORS

Malignant tumors of the small intestines accounts for about 2% of all gastrointestinal cancers and 64% of all small bowel tumors are malignant. 40% of the malignant tumors of small intestine are adenocarcinoma.

ADENOCARCINOMAS

similar to adenocarcinomas in the colon, these tumors usually arise from a premalignant adenoma. These tumors occurs both sporadically and , the context of familial adenomatous polyposis.

PATHOLOGY

These tumors occur frequently in the proximal small intestines and approximately 50% arise in the duodenum, 30% in the jejunum and 20% in the ileum.

Two pathological variants have been described, either the ulcerating type which is likely to result in bleeding or the annular type which is likely to present with obstruction. In 30% there is a synchronous malignant tumor.

CLINICAL FEATURES

Patients usually present with symptoms and signs of intestinal obstruction (abdominal pain, projectile vomiting, distended abdomen). Ulceration of the tumor may result in bleeding which might lead to anemia later on. Weight loss is usually evident on most patients.

On examination, patients can have signs of intestinal obstruction, peritonitis secondary to perforation, anemia or an abdominal mass.

INVESTIGATIONS

CBC may show mild anemia related to chronic blood loss. Liver function tests may reveal hyperbilirubinemia, which may be related to biliary obstruction from a periampullary tumor (in the duodenum around the ampulla of vater). Elevated trans-aminase levels also may be found in the presence of liver metastases. Carcinoembryonic antigen (CEA) levels may be elevated also.

Plain abdominal X-ray films may reveal partial or complete small-bowel obstruction. Upper gastrointestinal series with small-bowel follow through show abnormalities in 53-83% of patients with small-bowel cancer. Abdominal CT scan may elucidate the site and extent of local disease and the presence of liver metastases.

MANAGEMENT

surgical resection, with removal of lymph nodes (Margins of 5 cm are considered acceptable) is the treatment of choice. Chemotherapy usually incorporates 5-fluorouracil and nitrosoureas. In patients with involvement of the proximal duodenum, pancreatoduodenectomy might be necessary.

CARCINOID TUMORS

Carcinoid tumors (carcinoma like tumors) are a well- differentiated neuroendocrine tumor that arise from Argentaffin cells' (enteroendocrine cells) which are found normally at the base of intestinal crypt and was named as such because of its ability to reduce silver compounds. These cells are distinguished by their ability to secrete humoral agents i.e. serotonin.

The incidence of carcinoid is similar between males and females and peaks at approximately age 62 years

PATHOLOGY

These tumors are the most common distal small bowel malignancy (in contrast to adenocarcinoma which arises from the proximal intestines). Studies depending on surgical findings have demonstrated that about 3% of carcinoid tumors were in the duodenum, 5% in the jejunum, 32% in the proximal ileum and 60% in the distal ileum. The small bowel is the second most common site for carcinoid tumor after the appendix.

The tumor is a well circumscribed mass that grow inside the lumen of the intestines. microscopically it appears as a group of masses that are palisaded.

CLINICAL FEATURES

Due to their slow rate of growth and indolent course, carcinoids tend to remain asymptomatic for long periods. Symptoms of abdominal pain and intestinal obstruction usually present. On examination, if symptomatic, patients usually have a palpable abdominal mass. Patients may also present with bleeding if the mass ulcerates.

LYMPHOMA

In 10% of patients, the presentation is with neurohormonal symptoms caused by the release of 5-hydroxytryptamine (5-HT) or serotonin resulting in variety of manifestations referred to as the carcinoid syndrome. It manifests as bronchoconstriction, diarrhea, flushing and cardiac manifestations (arrhythmia).

The incidence of metastasis is quite low. Common sites of metastatic spread include the regional mesenteric and para-aortic lymph nodes and the liver. Usually carcinoid syndrome is not evident unless there is liver metastasis.

DIAGNOSIS AND TREATMENT

The clinical features, the site of the mass and the use of radiological studies is usually sufficient to make a diagnosis, although definitive diagnosis can only be made after biopsy. The measurement of the 5-HT metabolites in urine may aid in the diagnosis.

The primary tumor should be resected where possible. In many cases, the lesions are multifocal and may require multiple resections.

Intestinal lymphoma is tumor of the lymphoid tissue that arises either as primary lesion or secondary to the presence of a systemic lymphoma. These tumors are most commonly found in the ileum and ileocecal region (may due to abundance of lymphoid tissue at that region). Small bowel lymphoma is particularly common in the Middle East and This Mediterranean lymphoma is found in children and young adults. Celiac disease is a predisposing factor in primary small bowel lymphoma.

Patients usually present with symptoms and signs of intestinal obstruction, bleeding which is usually occult and weight loss plus features of lymphoma in other sites in cases of secondary intestinal lymphoma. Perforation may occur in some patients, causing an acute abdomen.

Surgery might be required for palliation of complications. Chemotherapy is the treatment of choice for curative intent.

GASTROINTESTINAL STROMAL TUMORS (GISTS)

Gastrointestinal stromal tumors (GISTs) is the current nomenclature for a diverse group of benign or malignant gastrointestinal neoplasms derived from embryonic mesoderm that may have smooth muscle or neural differentiation, or may appear as undifferentiated spindle cell lesions.

Most present either as an abdominal mass causing bowel obstruction evidenced as nausea and vomiting and abdominal pain. Patients may also present with gastrointestinal bleeding.

THE LARGE INTESTINES

ANATOMY

The large intestine, also known as the large bowel, has an average length of about 1.5 meters and a width of 7.5 cm. it can be divide into three parts: (1) the pouchlike cecum, the first portion of the large intestine; (2) the colon , the largest portion (1.3 meters); and (3) the rectum , the last 15 cm of the large intestine and the end of the digestive tract.

The cecum is the widest portion of the large intestines (7.5 to 8.5 cm). The ileum attaches to the medial surface of the cecum and opens into the cecum at the ileocecal valve. The appendix is attached to the posteromedial surface of the cecum.

The colon has a larger diameter and a thinner wall than the small intestine. The wall of the colon forms a series of pouches, or haustra. Haustra permit the expansion and elongation of the colon, rather like the bellows that allow an accordion to lengthen. Three separate longitudinal bands of smooth muscle-called the taeniae coli are on the outer surfaces of the colon just deep to the serosa. The serosa of the colon contains numerous teardrop-shaped sacs of fat called fatty appendices, the colon is subdivided into four regions ((figure 1); the ascending colon (15 cm), transverse colon (45 cm), descending colon (30 cm), and sigmoid colon (45 cm), the ascending colon end at the left colonic flexure, while the descending colon begins at the splenic or left colonic flexure. The transverse and sigmoid colons have mobility by virtue of possessing a mesentery, whereas ascending and descending colon are only partially peritonealised.

The rectum, is an expandable organ for the temporary storage of fecal material and begins where the taenia coli of the sigmoid colon join to form a cominuous outer longitudinal muscle layer at the level of the sacral promontory.

- ascending and descending colon >> intra and retro peritonium

Don't give abdominal symptoms until the inflammatory process go on,,, not mobile

- transverse colon >> intra peritonium >> there is a mesentery So its mobile and more prone to volvulus

- presence of some fatty tissue "can be very long in obese people >> on of the rare causes of acute abdominal pain (nonspecific) which is the most common cause of abdominal pain in the ER

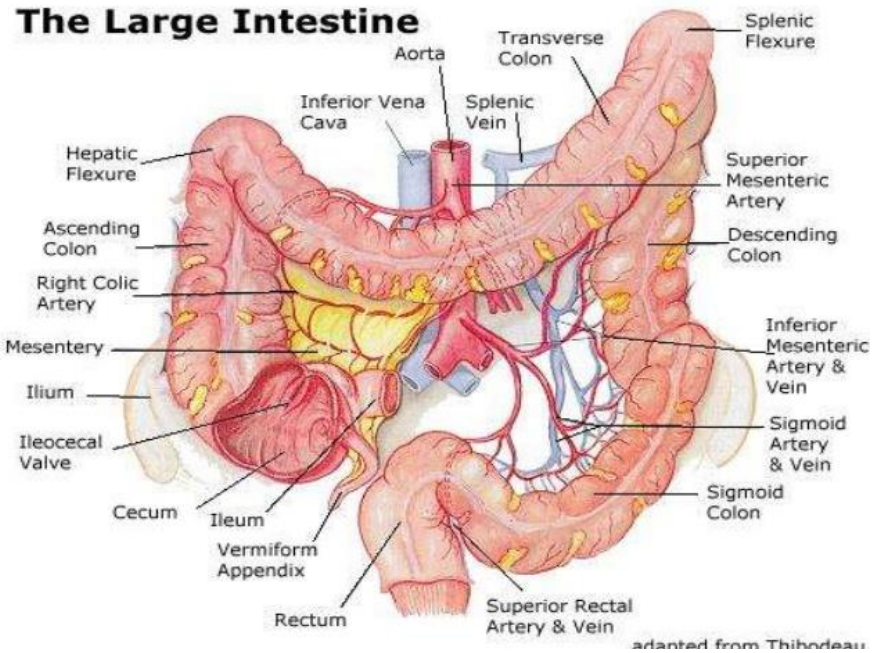


Figure 153 anatomy of the large intestine

- the caliber of the right side is wider than that of the left side >> so the tumors in the left side is presented earlier than in the right side

BLOOD SUPPLY AND VENOUS DRAINAGE

The arterial supply to the colon is shown below, the superior mesenteric artery branches into the ileocolic artery, which supplies blood flow to the terminal

ileum and proximal ascending colon, the right colic artery, which supplies the ascending colon, and the middle colic artery, which supplies the transverse colon. The inferior mesenteric artery branches into the left colic artery, which supplies the descending colon, several sigmoidal branches, which supply the sigmoid colon, and the superior rectal artery, which supplies the proximal rectum. The terminal branches of each artery form anastomoses with the terminal branches of the adjacent artery and communicate via the marginal artery of Drummond.

Except for the inferior mesenteric vein, the veins of the colon parallel their corresponding arteries and bear the same terminology. The inferior mesenteric vein ascends posterior to the pancreas to join the splenic vein. The superior mesenteric vein will join the splenic vein to form the portal vein.

LYMPHATIC DRAINAGE

The lymphatic drainage of the colon originates in a network of lymphatics in the muscularis mucosa. Lymphatic vessels and lymph nodes follow. The regional arteries. Lymph nodes are found on the bowel wall (epicolic), along the inner margin of the bowel adjacent to the arterial arcades (paracolic), around the named mesenteric vessels (intermediate), and at the origin of the superior and inferior mesenteric arteries (main).

INNERVATION

The nerve supply is derived from the sympathetic plexus surrounding the superior and inferior mesenteric arteries.

HISTOLOGY

Although the diameter of the colon is roughly three times that of the small intestine, its wall is much thinner. The major characteristics of the colon are the lack of villi, the abundance of goblet cells, and the presence of distinctive intestinal glands.

Like the small intestines, the wall of the large intestines is formed of a mucosa, a muscularis mucosae, a submucosa, a musclaris externa and a serosa. The glands in the large intestine are deeper than those of the small intestine and are dominated by goblet cells. The mucosa of the large intestine does not produce enzymes; any digestion that occurs results from enzymes introduced in the small intestine or from bacterial action. Large lymphoid nodules are scattered throughout the lamina propria and submucosa.

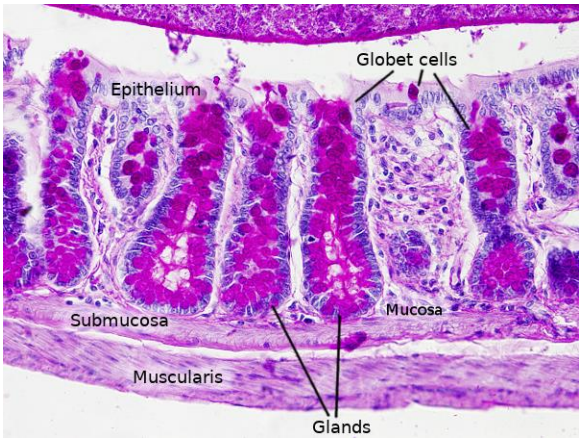


Figure 154 histology of the small intestine

PHYSIOLOGY

Less than 10 percent of the nutrient absorption under way in the digestive tract occurs in the large intestine. The reabsorption of water is an important function of the large intestine. Although roughly 1500 ml of material enters the colon each day, only about 200 ml of feces is ejected.

the large intestine absorbs a number of other substances that remain in the feces or that were secreted into the digestive tract along its length. Examples include useful compounds, such as bile salts and vitamins, organic waste products, such as urobilinogen, and various toxins generated by bacterial action.

The colon is also responsible for the synthesis of vitamins through its bacteria; these are vitamin K, vitamin B7 and vitamin B5.

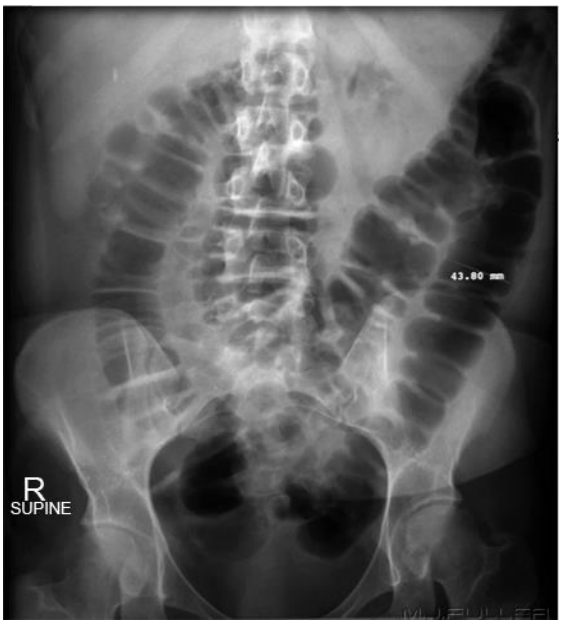


Figure 155 X-ray of normal colonic Haustration

DIVERTICULAR BOWEL DISEASE

Typically acquired disease, rarely may be congenital.

EPIDEMIOLOGY AND ETIOLOGY

The incidence of this disease increases with age with being highest after 70 years. It has an equal distribution among males and females. The disease incidence is highest in western countries and lowest in third world countries. This is attributed to the consumption of a refined, low-fiber diet in Western societies, resulting in reduced stool bulk (see the table below) with increased difficulty in passage of intestinal contents (constipation). Exaggerated spastic contractions of the colon isolate segments of the colon (segmentation) in which the intraluminal pressure becomes markedly elevated, with consequent herniation of the bowel wall through the anatomic points of weakness.

Although the whole colon can be affected, the segment most commonly involved is the sigmoid colon (in 95% of the cases), probably related to the high intraluminal pressure generated at this site when a low-residue diet is consumed.

the effect of fiber diet on stool parameters		
	Primitive life	Western life
Stool content	High fibres	Fibbers deficient
stool weight	> 400 gms	100 gms
stool transit time	12-24 hours	+ 72 Hours

1. Precise etiology of this disease is unknown.

- High intraluminal pressure and a weak colonic wall

-The condition also may be caused by abnormal colonic motility

2. .Genetic & environmental factors may play a role

- defective muscular structure, defects in collagen consistency.

3. Predisposing factors:

a. obesity

b. decreased physical activity

c. alcohol, coffee, cigarette smoking, low fiber diet

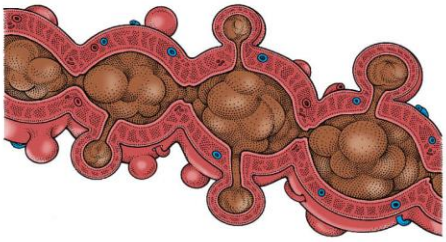


Figure 156 Segmentation

PATHOLOGY

1. It is a pseudo diverticulum

2. Usually found between mesenteric &a mesenteric taenia

3. occurs at the week sites in the circular m., mesen.vesseles penetration.

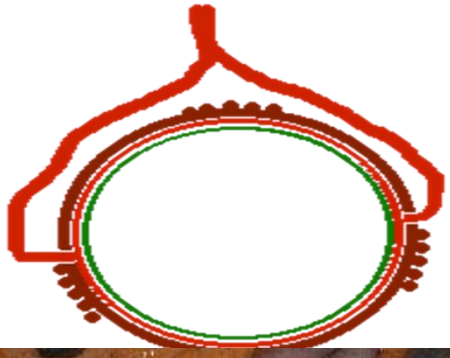
4. Elevated I.L. pressure by tonic &rhythmic contractions result in segmentation " non propulsive contractions produce isolated segments"

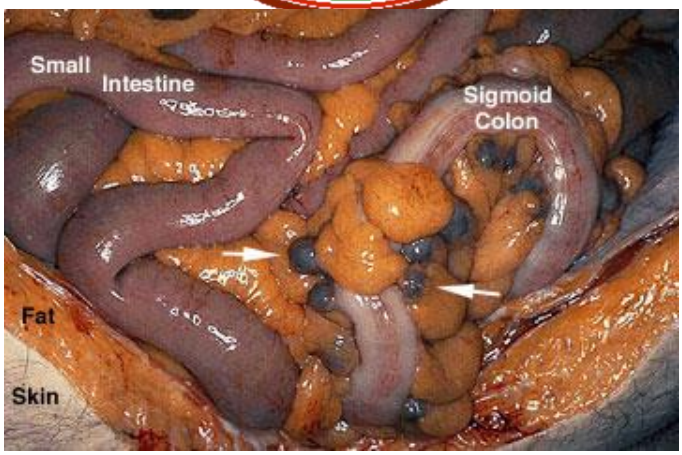
5. Thickening of long & circular muscles can lead to narrowing of colonic lumen

So, it's considered as one of the causes of intestinal obstruction due to hypertrophic of the muscle

Diverticulosis never occur in the rectum , because there is no tenia only circular and longitudinal muscles .

Weak points are the sites of entrance of the mesenteric arteries between the anti- mesenteric tenia and the mesenteric tenia. so, cause severe bleeding





DIAGNOSIS

1. Clinical features
2. Radiology, Barium enema
3. sigmoidoscopy, colonoscopy
4. CT
5. U/S

CLINICAL FEATURES

In most persons, diverticulosis is asymptomatic and is discovered only at autopsy or by chance during a laparoscopy or barium enema for some other problem. In only about a fifth of the cases does colicky abdominal pain appear, with flatulence and a sensation of never being able to completely empty the rectum.

Migratory or shifting pain >> hypogastric to the left iliac fossa

erosion of the wall by increased intraluminal pressure or inspissated food can result in inflammation that is quickly walled off with pericolic fat. This can result in Diverticulitis which presents with chills, nausea and vomiting, Pain and tenderness in the left lower quadrant and there is often a history of altered bowel habit, irritation of the bladder may result in urinary symptoms such as frequency, urgency and dysuria.

Examination may reveal fever, guarding and tenderness of the abdomen, especially the left iliac fossa. Rectal examination may, but does not usually, reveal a tender mass.

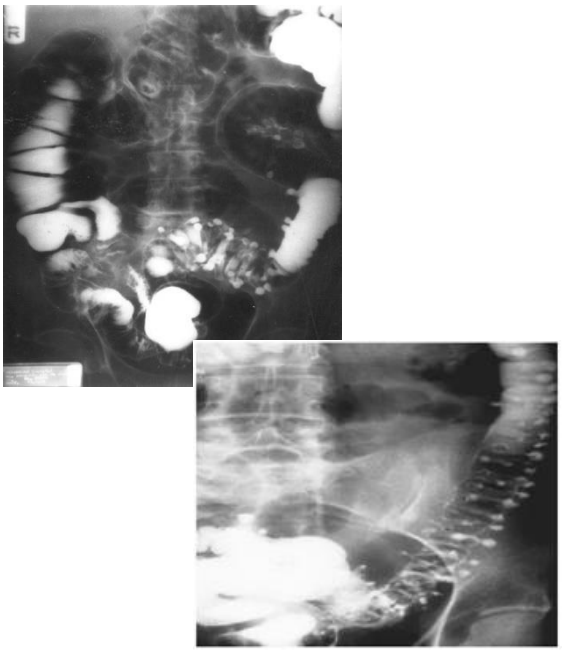
INVESTIGATIONS

The diagnosis of acute diverticulitis is made on clinical grounds, it can be confirmed by;

- Plan abdominal radiograph; will show local ileus (sentinel loop) or signs of large bowel obstruction.
- computerized tomography (CT); has high sensitivity and specificity for diverticulitis. It shows Bowel wall thickening and is also useful for diagnosing complications such as an abscess, Fluid or free air in peritoneal cavity, fistulas.

- Barium enema is a useful test to make the diagnosis of diverticular disease but is contraindicated during an acute attack because of the risk of chemical peritonitis. It shows the diverticula and a saw tooth pattern (figure 5).
- Colonoscopy/sigmoidoscopy; The diagnosis may be secured by gentie colonoscopy or flexible sigmoidoscopy and biopsy of inflamed segments. However, diverticulitis may not be apparent from within the bowel lumen itself.

it is safer to wait at least 6 weeks to do barium enema or Colonoscopy/sigmoidoscopy after the inflammation has subsided. Colonoscopy/sigmoidoscopy can be used to rule out associated malignancy.



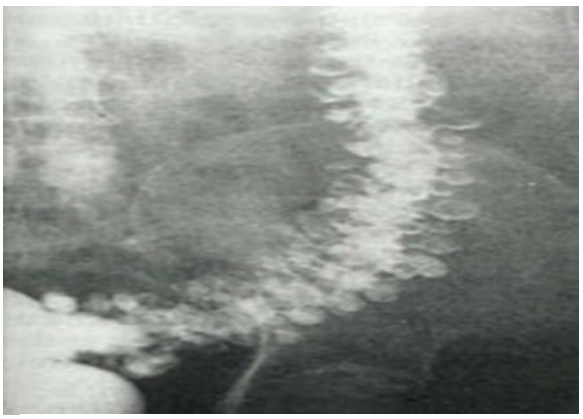


Figure 157 B+C Barium double contrast enema showing sigmoid Diverticular disease 'saw-teeth' and diverticula

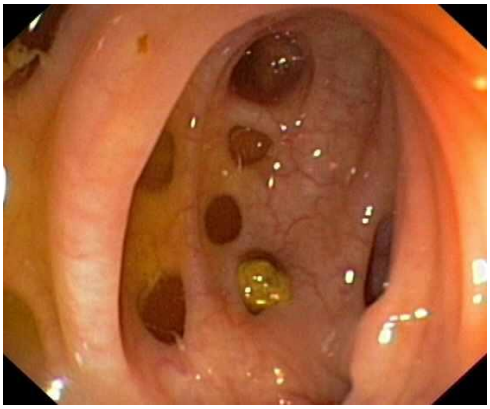


Figure 158 Colonoscopy showing fecal material impacted in the opening of the diverticulum causing pressure necrosis and sever bleeding

COMPLICATIONS

- Abscess; An abscess represents a further extension of the pathological process just described. The Hinchey staging system is often used to describe the severity of complicated diverticulitis: Stage I includes colonic inflammation with an associated pericolic abscess; stage II includes colonic inflammation with a retroperitoneal or pelvic abscess; stage Ill is associated with purulent peritonitis; and stage IV is associated with fecal peritonitis. The patient is often pyrexic and on physical exam one may find palpable abdominal mass.

- Bleeding; Diverticular disease may present with an episode of acute rectal bleeding, which, unlike the other complications of diverticula, is usually spontaneous and not the result of inflammation.

- Fistula; Fistula formation occurs when an inflamed diverticulum lies in close proximity to another hollow viscus. An inflammatory adhesion develops between them and the diverticulum then ruptures into the other viscus. A fistula between the large bowel and a loop of small bowel (coIoenteric) causes diarrhea, A colovesical fistula causes pneumaturia, passage of feces in urine and severe urinary tract infection and a fistula into the vagina (colovaginal) causes a purulent vaginal discharge.

- Intestinal obstruction; Diverticular disease occasionally presents with complete large bowel obstruction due to a combination of acute inflammatory thickening, muscle hypertrophy, spasm and stricture formation.

MANAGEMENT

DIVERTICULOSIS

Patients should be advised to take a high-fiber diet, supplemented if necessary by bran or a bulk laxative. This lowers intraluminal pressure, reliefs symptoms and prevents complications. Antispasmodics may be useful if there is smooth muscle spasm and colicky pain. Prophylactic antibiotics can also be used.

And Encourage physical activity, i.e. walking

Surgery is indicated for treatment of complications only. The treatment of diverticular bleeding is discussed in the lower gastrointestinal bleeding seminar.

DIVERTICULITIS

-It depends upon the severity of symptoms and clinical findings

Acute diverticulitis is treated by bed rest, the patient kept NPO, given fluids, a nasogastric tube is inserted for suction (in case the patient is vomiting and intestinal obstruction is suspected), and intravenous antibiotics (usually broad spectrum) are given.

The indications for surgery in diverticulitis are shown in the table below.

Indications for surgical intervention in diverticulitis

- **Failure of conservative management**
- **Recurrence for 2 episodes**
- **1 episode of diverticulitis in patient who is young, diabetic or immunocompromised patient.**
- **Treatment of complications**
  - **Perforation**
  - **Obstruction**
  - **Abscess not amenable to drainage (Hinchey 2-4)**
  - **Fistula**

Two procedures are performed. Hartmann's procedure is reserved for emergency settings when immediate anastomosis is not possible (risk of anastomotic leak) such as in the case of perforation, obstruction, edematous colon and adhesion. It involves resection of the portion containing the lesion, closing The rectum at the peritoneal reflection, with the left colon brought out as a left iliac fossa colostomy, colostomy reversal is done in 3 months.

For an elective setting. The ideal operation carried out (with careful preparation of the gut) is a one-stage resection. This involves removal of the affected segment and restoration of continuity by end-to-end anastomosis.

ULCERATIVE COLITIS

Ulcerative colitis is an idiopathic chronic episodic (relapses and remissions) inflammatory disorder limited to the rectum and colon and is characterized by bloody diarrhea, the disease is the result of abnormal activation of the immune system in the intestines.

It is found worldwide, but is most common in the United States, England, and northern Europe, recently increased frequency has been observed in developing nations. The sex distribution is nearly equal with *a* slight female preponderance. The onset of ulcerative colitis is most common between 15 and 40 years of age, with a second peak in incidence between 50 and 80 years.

PATHOLOGY

The precise etiology of ulcerative colitis is not well understood , abnormal activation of the immune system in the intestines is suggested.

Ulcerative colitis involves the rectum and sigmoid and may involve the entire colon. Colonic involvement is continuous starting from the rectum in 90% of the cases, so that skip lesions are not encountered.

The characteristic feature of ulcerative colitis is involvement restricted to the mucosa and submucosa of the large bowel. Diffuse inflammation of the mucosa with increase vascularity and congestion is seen. Because of the mucosal inflammation, water absorption is decreased and thus diarrhea results. Abscesses form at the base of the colonic crypts. The abscesses have a surrounding inflammatory infiltrate and coalesce to form crypt abscesses, which undermine the mucosa and cause ulceration of the overlying mucosa. The intervening mucosa becomes

edematous and may form inflammatory pseudopolyps. Because of the engorged vessels found at the ulcers, bleeding tendency is increased and thus the classic 'bloody diarrhea' results. When remission is induced, the ulcerated areas are soon covered by granulation tissue and later end with scarring and shortening. According to the extent the disease can be;

• Proctitis; Involvement limited to the rectum

• Proctosigmoiditis; Involvement of the

rectosigmoid colon

• Left-sided colitis; Involvement of the descending colon, up to the splenic flexure and the beginning of the transverse colon.

• Pancolitis; Involvement of the entire colon, extending from the rectum to the cecum.

ULCERATIVE COLITIS AND SMOKING

- current smokers with ulcerative colitis tend to have fewer and less severe disease flare-ups.

- Cigarette smokers have a 40 percent lower risk of developing ulcerative colitis than do nonsmokers

- Researchers recently reported that smoking appears to alter the makeup of the various types of bacteria living in the intestinal tract

- Ulcerative colitis is an immune disease; it occurs when a person's immune system mistakenly attacks and destroys the tissues of the colon

*The relationships among intestinal microbes and the immune system are of particular relevance to inflammatory bowel disease

CLINICAL FEATURES

The first symptom is watery or bloody diarrhea. a lower or even diffuse abdominal pain is present in majority of patients but is not the first symptom and is relieved after defecation. Other than the increased fecal frequency, patients often have fecal urgency and tenesmus which can be very distressing.

Patients usually have low grade fever, tachycardia, are pallor as a result of anemia and usually when they present, they are dehydrated and malnourished. abdominal tenderness is seen during a relapse. Careful rectal examination should detect associated conditions as fissure, fistula and hemorrhoids and show the presence of blood during a relapse. The rectal mucosa often feels thick and boggy.

Although ulcerative colitis is primarily a disease of the large bowel, systemic manifestations (iritis, polyarthritis, ankylosing spondylitis, hepatitis, erythema nodosum, pyoderma gangrenosum) can occur. Primary sclerosing cholangitis (PSC). affects 2-5% of cases of ulcerative colitis.

ASSESSMENT OF SEVERITY

Assessment of severity has important therapeutic considerations, because patients with more severe disease (based on these criteria) respond less well to therapy, the criteria for assessing severity of ulcerative colitis are shown in the table below.

Ulcerative colitis according to severity

- **Mild; <4 motions /day, no systemic signs**
- **Moderate; >4 motions /day, no systemic signs**
- **Severe; >4 motions /day with systemic signs (fever, tachycardia, weight loss, Hypoalbunemia)**

ULCERATIVE COLITIS VS CROHN'S DISEASE

ulcerative colitis and crohn's disease both fall under the category of inflammatory bowel disease (IBD), and they usually overlap in many clinical features, pathologically speaking however, they are very different diseases, the table below presents the similarities and differences between the two.

Crohn's disease vs ulcerative colitis

Similarities;

- **Both are chronic inflammatory diseases.**
- **Both are of unknown etiology**
- **Both have no cure following medical treatment**
- **Both have exrtraintestinal manifestations**
- **Presence of diarrhea in both cases.**

Non similarities;

- **Anatomical site in gastrointestinal tract**
- **Anatomical site in bowel wall.**
- **Presence of skipped lesion.**
- **Mucosal appearance**
- **Surgical cure**

INVESTIGATIONS

- complete blood count; anemia is seen in the majority of cases with uicerative colitis, it is a microcytic anemia due to blood loss and chronic inflammation. it might be hemolytic in nature.
- ESR and CRP are both elevated and their elevation correlates with disease activity (i.e, relapse). thus are useful in monitoring disease, but are not specific for diagnostic purposes.
- others; ulcerative colitis is usually manifested by Hypoalbuminemia(<3.5 mg/dl) and its level has been correlated with severe severity, hypokalemia is also noted in most patients.
- barium enema; barium enema may also help in the assessment of the extent of disease but is contraindicated in patients with fulminant colitis and those with toxic dilatation because of the risk of precipitating perforation. Typical changes include loss of haustrations, fluffy granularity of the mucosa, and pseudopolyps. Undermining ulcers may create a double contour to the edge of the colon. Ultimately, over a period of years of chronic colitis, the bowel may become short and featureless, resembling a smooth tube (burnt-out colitis).
- colonoscopy; Colonoscopy has an important place in the diagnosis and surveillance of colitis by detecting evidence of dysplasia.

COMPLICATIONS

Toxic colonic dilatation (Fulminating colitis) is the most common cause of death in ulcerative colitis and is characterized by a thin-walled, large, dilated colon that can eventually become perforated. Symptoms and signs Include abdominal pain and distension, fever and weakness, patient become disoriented Plain radiograph may show a colonic diameter> 6 cm with mucosal islands.

Perforation, sever hemorrhage are also seen in complicated ulcerative colitis. Benign strictures may rarely cause intestinal obstruction. Colonic adenocarcinoma develops in 3-5% of patients with ulcerative colitis. The risk increases with the duration of disease. The risk of colonic malignancy is higher in pancolitis and in cases in which disease occurs before the age of 15 years.

MANAGEMENT

The management of ulcerative colitis depends on location and severity of the disease, the presence of complications and patient response to treatment.

MEDICAL TREATMENT

medications used for symptomatic relieve are important part of the management and include Antidiarrheals, Pain reliever and Iron supplements for blood loss, other medications are used to induce remission and include;

• Anti-inflammatory drugs; 5-aminosalicylic acid (5-ASA) and daughter compounds is effective in inducing remission in mild to moderate ulcerative colitis. Maintaining remission, are often the first step in the treatment of inflammatory bowel dis-ease. They include Asacol, Pentasa, Dipentum, Colazal, and Rowasa.

• Steroids; In moderate to severe disease and in patients who fail to respond to 5- ASA compounds, systemic (oral) corticosteroids can be used, prednisolone can be used in 20-40 mg/day for 3 weeks to induce remission.

• Immunomodulators; these are used for treating severe ulcerative colitis and include azathioprine/6-mercaptopurine, methotrexate, and cyclosporine.

Admission to hospital is required for severe cases. Fluid and electrolytes balance needs to be maintained since most patients with severe attacks are dehydrated and malnutrition. Anemia should be corrected, with adequate nutrition -TPN. patients should be put on I.V. hydrocortisone (100 -200 mg 4TD). Rectal predsol infusion along hydrocortisone may decrease the intensity of the inflammation and may sometimes induce temporary or even permanent remission.

SURGERY

indications for surgical treatment of patients with ulcerative colitis are shown in the table below. Surgery can often eliminate ulcerative colitis, in emergency settings Total colectomy with ileostomy is the procedure of choice. Proctocolectomy plus ileo-anal. anastomosis with ileal pouch is the procedure of choice for elective settings.

Indications for surgery in ulcerative colitis
• **Severe cases failing to respond to medical therapy.**
• **Chronic disease with frequent motions anemia, urgency and tenesmus**
• **Severe dysplasia, risk of neoplastic changes**
• **Extra-intestinal manifestations**
• **Massive bleeding, perforation, Toxic megacolon**

PROGNOSIS

bad prognostic features for patients with ulcerative colitis include age above 60 years, when the whole colon is involved and sever initial attack.

COLORECTAL NEOPLASMS

COLORECTAL POLYPS

A colorectal polyp (plural polyps) is a small mucosal outgrowth into the lumen of the colon or rectum, traction on the mass may create a stalked, or pedunculated polyp. Alternatively, the polyp may be sessile, without a definable stalk (figure 1).

Polyps may be formed as a result of abnormal mucosal maturation (metaplastic or hyperplastic), inflammation (inflammatory), or architecture (hamartomata's). These polyps are non-neoplastic and do not have malignant potential. Those polyps that arise as the result of cellular proliferation and dysplasia are termed neoplastic polyps. They can either be benign of malignant.

INFLAMMATORY POLYPS

Inflammatory polyps occur secondary to an inflammatory process. And most of the underlying causes are infections. These include;

- Bilharzial polyp; prevalent in Egypt and Sudan and occurs in the course of bilharziasis (schistosoma mansoni).

- Ameaboma; occurs secondary to infection with amoeba species and is caused by chronic inflammation (granuloma).

- Tuberculoma; these are also granulomas that result from infection with mycobacterium tuberculosis.

- Psuedopolyps; these are seen in patients with inflammatory bowel disease, especially ulcerative colitis, and occur secondary to submucosal inflammation and exudation. They are called psuedopolyps because they lack a fibrovascular core.

HAMARTOMATOUS POLYPS

Hamartomas encompass a family of masses in which the tissue constituents are the same as those of the surrounding tissues, but the arrangement is different. Hamatomatous polyps occur in a variety of syndromes as discussed below.

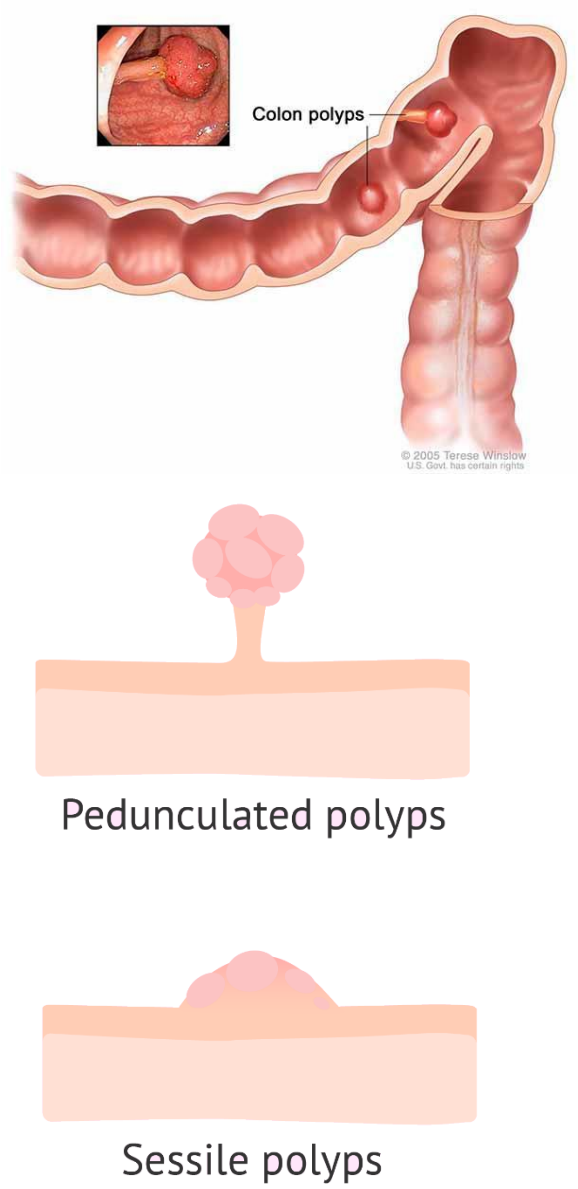


Figure 159 differentiation between pedunculated and sessile polyp

PEUTZ-JEGHERS SYNDROME

Peutz-Jeghers syndrome is an autosomal dominant inherited disorder. The manifestations include gastrointestinal hamartomatous polyps (figure 2) most commonly in the jejunum and melanin pigmentation at mucocutaneous junctions, and occasionally on the dorsum of the hands and feet. This condition appears in children and may present as obstruction (due to intussusceptions) or as rectal bleeding.

Treatment is conservative wherever possible. However, bleeding and intussusceptions are indications for surgical excision (polypectomy)'.

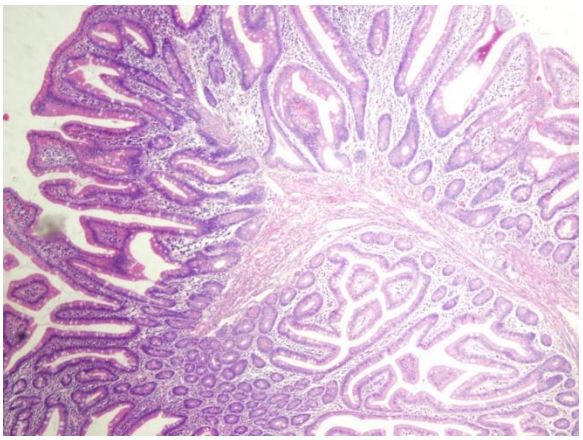


Figure 160 structure of peutz-jeghers polyp

JUVENILE POLYPOSIS SYNDROME (JPS)

This rare disease occurs in children of ages less than 10 years of age. The formed polyps are once thought to be the result of cyst formation secondary to obstruction of intestinal crypts, however, it's now agreed that these are hamartomatous in nature. Polyps ulceration may lead to rectal bleeding, they are associated with increased risk of malignancy. Surgical excision in symptomatic polyps might eventually be needed.

COWDEN'S DISEASE

This is a rare disseminated form of gastrointestinal hamartomatous polyposis with an autosomal dominant pattern of inheritance, but most cases are due to mutations. There is a greater risk for benign and malignant disease of skin, breast and thyroid. All patients have warty tricholemmomas around the eyes and the nose, and these lesions are diagnostic when present in association with oral fibromas and keratoses of the hands and feet.

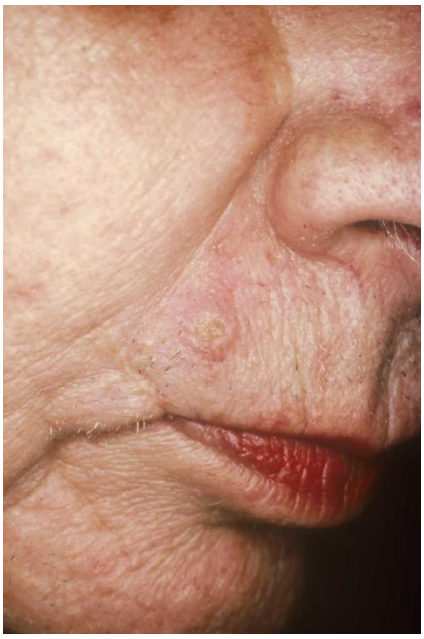


Figure 161 warty tricholemmomas around the nose

CRONKHITE-CANADA SYNDROME

This rare syndrome comprises intestinal polyposis with alopecia, atrophy of the nails and brown macular hyperpigmentation. It may present with diarrhea, weight loss, and malnutrition. Histological examination shows cystic dilatation of the crypts, similar to that seen in juvenile polyposis. The condition does not seem to be inherited (not familial) and does not have a malignant potential.

GARDNER SYNDROME

Gardner syndrome is an autosomal dominant form of polyposis characterized by the presence of multiple polyps in the colon, desmoid tumors, osteomas, epidermoid cysts, lipomas, dental abnormalities (exotosis) and periampullary carcinomas.

ENDOMETRIOMAS

Endometrioma is a rare mass that is associated with endometriosis and represents an ectopic endometrial tissue in the rectum. It usually presents in teenage girls who started having menses and complain of rectal bleeding during menstruation along with secondary dysmenorrhea. On sigmoidoscopy, endometriosis involving the rectosigmoid junction usually presents as a stricture, with the mucous membrane intact. Hormonal manipulation is the first line of therapy, but sometimes total abdominal

hysterectomy and bilateral salpingoophorectomy is required.

HYPERPLASTIC POLYPS

hyperplastic polyps (sometimes referred to as hyperplastic), are small (<5 mm in diameter), smooth protrusions of the mucosa. They may occur singly but are more often multiple (figure 6). Although they may be anywhere in the colon, well over half are found in the rectosigmoid region. Histologically, they are sessile and contain abundant crypts lined by well- differentiated goblet or absorptive epithelial cells, separated by a scant lamina propria.

These polyps have no malignant potential, however because of the difficulty to distinguish them from benign adenomas which do have a malignant potential, they are often removed. Plus, it is now being recognized that some polyps, the so-called sessile serrated adenomas, located on the right side of the colon, may be precursors of colorectal carcinomas.

NEOPLASTIC POLYPS

COLORECTAL ADENOMA

Colorectal adenomas represent a true neoplastic epithelial polyps.

PATHOLOGY

They are classified as tubular, tubulovillous and villous adenomas, depending on their histological architecture, and such classification has clinical relevance with respect to cancer risk. Tubular adenomas are usually sessile that soon becomes pedunculated. Tubular adenomas account for 70% of all adenomas, villous adenomas account for 10% of adenomas, However, villous adenomas account for 60% of lesions larger than 2 cm. Villous tumors are sessile and are most common in the rectum and some may carpet the rectum giving a velvety feeling on rectal exam. The tubulovillous types an account for 20% of the cases.

After FAP, villous adenoma is the riskiest condition that can predispose adenocarcinoma, followed by tubulovillous, followed by tubular adenoma.,

CLINICAL FEATURES

The majority of polyps are asymptomatic ¸They may induce rectal bleeding or **abdominal pain,** especially when a large polyp has caused intussusception. Occasionally, a rectal polyp may prolapse through the anus.

Patients with rectal villous adenomas can present with severe watery diarrhea, and water and electrolyte depletion (hypokalaemia, hyponatremia) due to excessive mucus loss. Rectal adenomas may be palpable as nodular masses, but villous tumors are soft and may be missed (velvety feeling).

DIAGNOSIS

Distal polyps are detected readily by sigmoidoscopy; but there is a need for full colonoscopy in view of the risk of synchronous lesions. The colonoscopic appearance of a rectal adenoma.

MANAGEMENT

Colonoscopic removal -of asymptomatic polyps has been shown to reduce future risk of malignant conversion. polypectomy using **an electrocautery snare** (figure 4) is required for pedunculated polyps but might be dangerous for sessile ones. It may be necessary to perform surgical excision of larger polyps (villous adenomas) per-anally or by bowel resection.

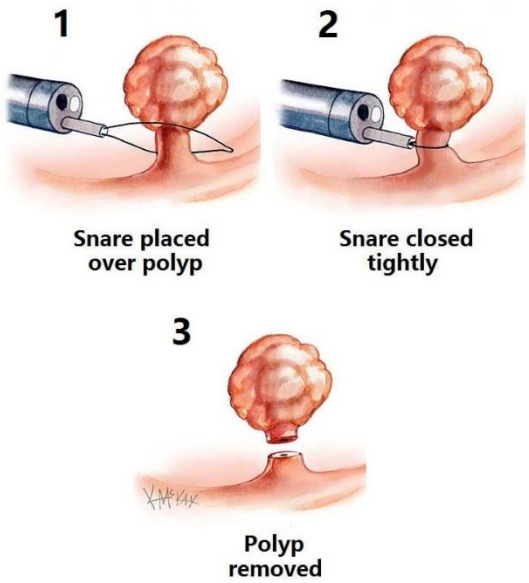


Figure 162 snaring with electrocautery

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)

Familial adenomatous polyposis (FAP) is an autosomal dominantly inherited condition that is characterized by the development of multiple rectal and colonic polyps. individuals with this syndrome typically develop 500 to 2500 colonic villous adenomas that carpet the mucosal surface (figure 10); a minimum number of 100 is required for the diagnosis.

ETIOLOGY

The genetic defect underlying FAP has been localized to the APC (adenomatous polyposis coli, a tumor suppressor gene) gene on chromosome 5q21. The gene seems to run in families in an autosomal dominant fashion. Around 25% of affected individuals have no family history of FAP, the disease arising in these sporadic cases as the result of a new mutation of this gene. The molecular pathogenesis regarding how this gene results in this syndrome is still under study.

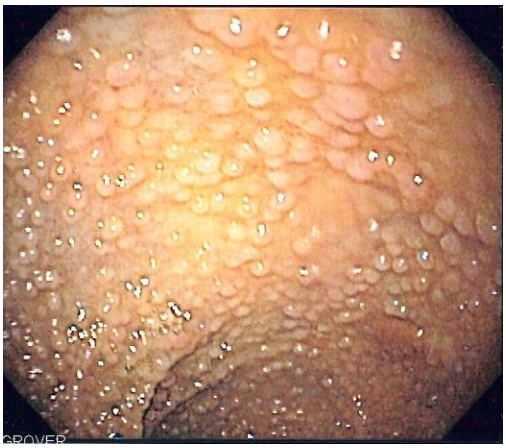


Figure 163 pathology of colon with FAP

CLINICAL FEATURES

Polyps usually develop during the teenage years and early adulthood (usually 20 years), the risk of colorectal cancer is virtually 100% by midlife (by age 30 years), unless a prophylactic panproctocolectomy is performed. FAP can be asymptomatic, but can present as diarrhea, weight loss, or if the polyps are ulcerated, presents with rectal bleeding. The blood; is usually mixed with mucus. Pigmented lesions of the retina, known as congenital hypertrophy of the retinal pigment epithelium (CHRPE), are well described in association with FAP.

FAP when associated with Osteomas, epidermoid Cysts, exostosis, multiple fibromas, multiple lipomas, and desmoids tumors give rise to Gardner's syndrome. The occurrence of neuronal tumors along with

FAP should raise the suspicion Turcot syndrome.

DIAGNOSIS AND MANAGEMENT

The diagnosis can be established by sigmoidoscopy and biopsy. Screening of affected individuals by direct APC gene mutation analysis is used for the following;

• To confirm the diagnosis of FAP and the diagnosis of attenuated FAP (more than 20 polyps)

• To provide pre-symptomatic testing of individuals at high risk (first degree relatives of a patient

Pre-symptomatic detection of FAP allows prophylactic surgery before malignancy supervenes. There is no general consensus on the preferred surgical strategy, as both restorative panproctocolectomy with ileoanal pouch formation and total colectomy with ileorectal anastomosis have particular advantages. The upper gastrointestinal tract should be screened for duodenal adenoma or carcinoma.

OTHER NEOPLASTIC POLYPS

Colorectal adenomas and FAP fall under the classification of adenomatous polyps, however other neoplastic polyps might also affect the large intestines. They include lipomas, hemangiomas and leiomyomas.

COLORECTAL CARCINOMA

Colorectal carcinoma is a primary tumor of the large intestines.

EPIDEMIOLOGY

Colorectal carcinoma is the most common gastrointestinal malignancy and is second only to lung cancer as a cause of cancer death in developed countries. The incidence of this cancer increases with

age, reaching about 39 in 100,000 after the age of 50 to about 450 in 100,000 after the age of 80 years.

The male: female ratio for colon cancer is close to unity with a slight preponderance of right colon cancers to affect the females, whereas that for rectal cancer is 1.7:1 in high-incidence populations. The rectum and sigmoid are particularly common sites for tumors (figure 6).

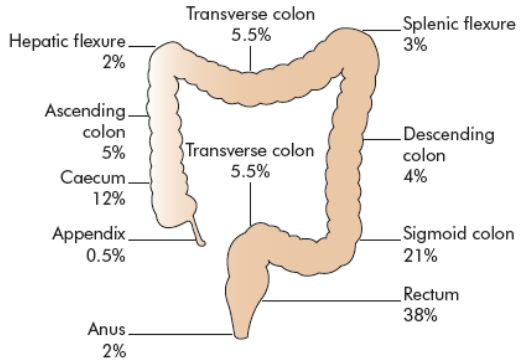


Figure 164 distribution of colorectal cancer by site

ETIOLOGY

Both genetic and environmental influences contribute to the development of colorectal cancers. The following discusses the risk factors leading to colorectal carcinoma;

• Genetics; individuals with hereditary nonpolyposis colorectal cancer syndrome (HNPCC, also known as Lynch syndrome), caused by germline mutations of DMA mismatch repair genes, are at a high risk of developing colorectal cancers.

• Precancerous lesions; the most established precancerous lesion for colorectal carcinoma is a colorectal adenoma wither isolated or in association with FAP. This has led to the recognition of what is known as the adenoma-adenocarcinoma sequence. In this sequence, the earliest event is loss of the tumor suppressor gene; the APC gene on the long arm of chromosome 5 resulting in an adenoma. This followed by mutation of k-RAS gene (when mutated it prevents apoptosis) found on the long arm of chromosome 12. This will usually result in dysplasia. Loss of p53 (tumor suppressor gene) is noted in 70% to 80% of colon cancers, yet similar losses are infrequent in adenomas, suggesting that mutations in p53 occur late in colorectal carcinogenesis.

Malignant transformation is also seen in peutz-jeghers syndrome and juvenile polyposis syndrome.

- Diet; The dietary factors receiving the most attention are (1) a low content of fiber, (2) a corresponding high content of refined carbohydrates (toxic oxidative byproducts of carbohydrate), (3) a high fat content (as from meat), and (4) decreased intake of protective micronutrients such as vitamins A, C, and E. It is theorized that reduced fiber content leads to decreased stool bulk, increased fecal retention in the bowel with increased contact time to fecal toxic material.

- Inflammation; The risk of colorectal cancer in ulcerative colitis and Crohn's disease is increased. Some reports indicate that bilharziasis increases the risk of colorectal cancer.

- Others; radiation, alcohol and smoking.

PATHOLOGY

The rectum and sigmoid are particularly common sites for tumors (figure 6). grossly, the tumor may take one of four forms (Fig. 7). The annular variety (type 1) tends to give rise to obstructive symptoms (and usually the earliest to present). the others; tubular (type 2), ulcerating (type 3) and cauliflower (type 4) will present more commonly with bleeding. Type 4 is the least malignant form. Type 3 typically, has raised everted edges, a slough-covered floor and indurated base (figure 8).

Regardless of their gross appearance, all colon carcinomas are microscopically similar. Almost all are adenocarcinomas that range from well-differentiated (11%) to moderately differentiated (in 64% of cases), to frankly anaplastic masses (25%). Around 10-20% of tumors have mucinous histology and this tumor type has a poor prognosis.

CLINICAL FEATURES

Colorectal cancers remain asymptomatic for years. Symptoms depend on the site of the tumor, the pathological variety and the stage of the tumor.

Because the dominant pathological variety is the cauliflower type, Cecal and right colonic cancers most often are called to clinical attention by the appearance of fatigue, weakness, and iron deficiency anemia secondary to bleeding.

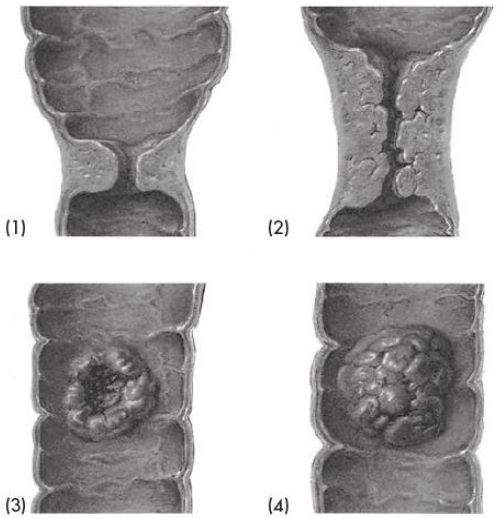


Figure 165 pathological types of colorectal cancer

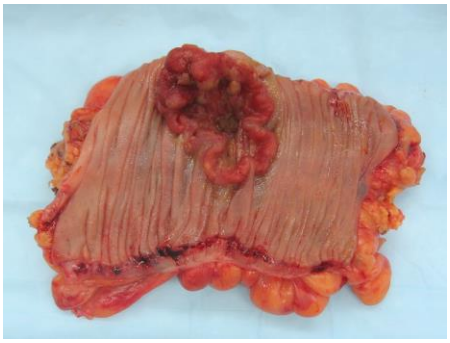


Figure 166 ulcerative type if colorectal cancer

They may also present with an abdominal mass, obstruction (rarely in cecal masses (because it is wide)), intussusception or appendicitis secondary to obstruction of the lumen of the appendix. sometimes the tumor may seed in the appendix and cause an appendicular tumor.

Left-sided lesions are predominated by the annular variety and thus may present with changes in bowel habit (caused by a cycle of obstruction and constipation, bacterial overgrowth and diarrhea), or left lower quadrant pain. The patient may have occult or apparent bleeding mixed with the stool (hematochezia). On examination an abdominal mass might be felt in the left half of the abdomen (usually it reflects an impacted feces) and there might be signs of large bowel obstruction. Although anaemia in females may arise from gynecologic causes, it is a clinical maxim that *iron deficiency anaemia in an older man means colorectal cancer until proved otherwise.*

Patients with tumors in the transverse colon usually have an epigastric mass and may be mistaken for a case gastric cancer. They may present with large bowel obstruction. Rectosigmoidal tumors may also present with large bowel obstruction but can also present with tenesmus and due to bladder initiation, may present with lower urinary symptoms. A digital rectal examination can allow the mass to be directly felt in the case of these tumors. The ulcerating variety is predominant in these tumors and thus rectal bleeding is seen.

INVESTIGATIONS

• Blood tests; a complete blood count usually shows microcytic, hypochromic anemia. elevated blood levels of carcinoembryonic antigen (CEA) is not specific for this cancer (elevated in stomach pancrease, breast and thyroid also in ulcerative colitis, pancreatitis and cirrhosis) and is significant only when there is metastasis. Thus it is only used for monitoring recurrence after treatment.

• Proctosigmoidoscopy or Colonoscopy is the investigation of choice and is effective in identifying tumors throughout the colon and rectum. colonoscopy has the advantage of allowing diagnostic biopsy and snaring of any adenomas proximal to the cancer.

• Radiological studies; air contrast Barium enema has similar diagnostic accuracy to colonoscopy. It shows a filling defect sometimes with an applecore lesion (in annular lesions, figure 15). Computed tomography and other radiographic studies (e.g. ultrasonography for liver metastasis) are usually used to assess metastatic spread.

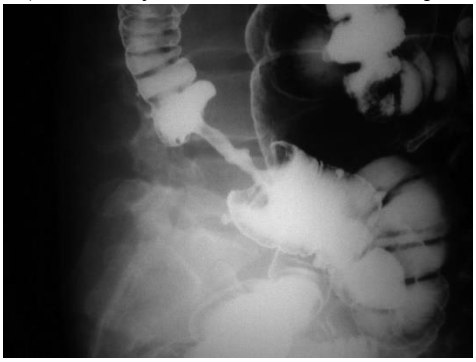


Figure 167 barium enema showing a filling defect with shouldering effect (apple core lesion).

STAGING AND SPREAD

All colorectal tumors spread by direct extension into adjacent structures and by metastasis through the lymphatics and blood vessels.

Staging can be done using proctosgmoidoscopy with biopsy, colonoscopy, CT scan and ultrasonography (endoanai or liver ultrasound) along with other techniques for detecting distant metastasis (X-rays or PET scan). The challenge is to discover these neoplasms when curative resection is possible.

two staging systems has been used, the oldest being the dukes' staging system. Dukes was a pathologist and staged tumors based on the specimens he received. His classification was first described for rectal carcinomas and later for colonic carcinomas (shown in the table below).

Dukes' staging of colorectal cancer	
stage	Description
A	Spread into, but not beyond, muscularis externa
B	Spread through full thickness of bowel wall
C	Spread to involve lymph nodes C_1; spread to the epicolic lymph nodes C_2; spread to the paracolic lymph nodes
D	Distant metastases

this system has largely been replaced by the more detailed TNM staging system (see the table below) and is no longer recommended for use in clinical practice.

TNM staging of colorectal cancer		
TO	none evident	
Tis	in situ (limited to mucosa)	
T1	invasion of lamina propria or submucosa	
T2	invasion of muscularis externa	
T3	invasion through muscularis externa into subserosa or nonperitonealize perimuscular tissue	
T4	invasion of other organs or structures	
NO	none evident	
N1	1 to 3 positive regional nodes	
N2	4 or more positive regional nodes	
N3	any positive node along a named blood vessel	
MO	none evident	
Ml	any distant metastasis	
Stage	TNM	Corresponding Dukes'
1	$T_1N_0M_0$ or $T_2N_0M_0$	A
II		
IIa	$T_3N_0M_0$	B
lIb	$T_4N_0M_0$	B
III		
Illa	$T_{1-2}N_1M_0$	C
lllb	$T_{3-4}N_1M_0$	C
lllc	Any T N $_2$M $_0$	C
IV	Any T Any N M1	D

All colorectal tumors spread by direct extension into adjacent structures and by metastasis through the lymphatics and blood vessels.

MANAGEMENT

The mainstay of treatment comprises resection of the primary tumor and excision of regional nodes, a proximal mesenteric ligation will eliminate the blood supply to a greater length of colon and require a more extensive "colectomy." This might be needed when the tumor is curable, because of the need to remove the maximum amount of regional lymph nodes that could be involved.

Carcinoma of the cecum or ascending colon is treated when respectable by right hemicolectomy (figure 10-A) an end-to-end anastomosis is fashioned between the ileum and the transverse colon. An extended right colectomy (figure 16-B) may be used for curative intent resection of tumors located at the hepatic flexure or proximal transverse colon, tumors in the mid and distal transverse colon may be resected by ligating the middle colic vessels and resecting the transverse colon (transverse colectomy, figure 10-C), followed by a coIocolonic anastomosis. For tumors confined to the distal transverse colon, splenic flexure, or descending colon, a left colectomy is performed (figure 10-D,E).

tumors in the sigmoid colon require ligation and division of the sigmoid branches of the inferior mesenteric artery. In general, the entire sigmoid colon should be resected to the level of the peritoneal reflection (Sigmoid Colectomy, figure 10-F) and an anastomosis created between the descending colon and upper rectum.

The management of rectal tumors is special. A low anterior resection is used to remove tumors in the upper and mid rectum. It involves ligation of the inferior mesenteric artery distal to the origin of the left colic artery followed by removal of the rectum (provided the lower edge of the tumor is 1-2 cm above the anal sphincters, the sphincter can be preserved in most patients). Ideally this is to a distance of 5 cm below the primary tumor, although a margin of 1 cm is acceptable when the tumor is very low. This should be followed by a colo-anal anastomosis, and this can be combined with a small colonic J-pouch to improve defecatory function.

For low rectal cancer involving the sphincter muscle, it may be necessary to remove the anal sphincter as part of an abdominoperineal resection and fashion a permanent end-colostomy.

Palliative surgery along with radiotherapy and chemo-therapy is used for incurable tumors.

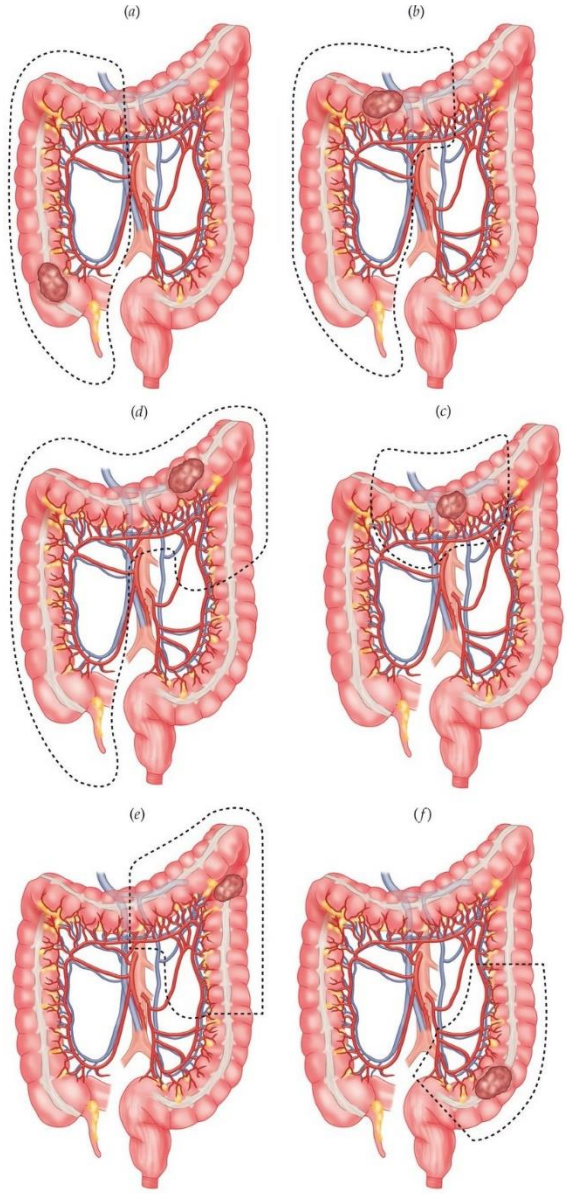


Figure 168 Extent of resection for carcinoma of the colon. *A.* Cecal cancer. *B.* Hepatic flexure cancer. *C.* Transverse colon cancer. *D.* Splenic flexure cancer. E. Descending colon cancer. *F.* Sigmoid colon cancer.

STOMAS

COLOSTOMY OR ILEOSTOMY

Stoma: is an artificial opening made in the colon (or small intestine) to divert faeces and flatus outside the abdomen where they can be collected in an external appliance (bag)

Other classification:

- Permanent colostomy.
- Temporary colostomy: to give the bowel a chance to rest and heal , after healing colostomy can be reversed and normal bowel function restored.

Indications of stoma :

- Inflammatory bowel disease
- Ulcers
- Polyps
- Cancers
  - Colon carcinoma
  - Rectal carcinoma
- Disorders of bowel function – Hurschprung's disease
- Accidental injury
- Congenital deformities of anus and rectum

TYPES:

1-LOOP COLOSTOMY

It is now less commonly employed, as it is difficult to manage and potentially disrupts the marginal arterial supply to the anastomosis

*A loop ileostomy is now more commonly used

2-END COLOSTOMY

INDICATIONS:

1- abdominoperineal excision of the rectum
2- as part of a Hartmann's procedure

bringing the divided colon through a left iliac fossa trephine in rectus abdominis and skin .

The best site is usually through the lateral edge of the rectus sheath, above and medial to the bony prominence

3-DOUBLE BARREL STOMA

- Bowel is surgically severed and 2 ends are brought out into the abdomen as 2 separate stomas
- Proximal end – functional stoma
- Distal end – non functioning (mucus fistula)
- Used in temporary diversion – cases where resection is required due to perforation or necrosis

4-LOOP ILEOSTOMY

is often used for defunctioning a low rectal anastomosis or an ileal pouch.

A knuckle of ileum is pulled out through a skin trephine in the right iliac fossa.

5-END ILEOSTOMY

INDICATIONS:

1- After a subtotal colectomy without anastomosis when it may later be reversed

2-permanent after a panproctocolectomy.

The ileum is normally brought through the rectus abdominis muscle.

There may be an **'ileostomy flux'** while the ileum adapts to the loss of the colon.

While ileostomy output can amount to 4 or 5 litres per day, losses of 1–2 litres are more common.

A consistent ileostomy output in excess of 1.5 litres is usually associated with dehydration and sodium depletion in the absence of intravenous therapy.

The stools thicken in a few weeks and are semisolid in a few months.

Complications of an ileostomy include : prolapse, retraction, stenosis, bleeding, fistula and parastomal hernia.

6- CAECOSTOMY

In unstable patients with advanced obstruction, a caecostomy may be useful.

In late cases of obstruction, the caecum may become so distended and ischaemic that rupture of the caecal wall may be anticipated.

STOMA BAGS AND APPLIANCES

Stoma output is collected in disposable adhesive bags.

Ileostomy appliances tend to be drainable bags, which are left in place for 48 hours, while colostomy appliances are simply changed two or three times each day.

A wide range of such bags is currently available. Many now incorporate an adhesive backing, which can be left in place for several days.

STOMA COMPLICATIONS

Early: 1. Ischemia 2. Bleeding 3. Retraction 4. Skin irritation

Late: 1. Prolapse 2. Parastromal hernia 3. Recurrent disease 4. Bowel obstruction

EXAMINATION

INSPECTION

SITE

LIF: Colostomy

RIF: Ileostomy or urostomy

NUMBER OF LUMENS

1 and in RIF: End ileostomy or urostomy

1 and in LIF: End colostomy

2 joined and in RIF: Loop ileostomy

2 joined and in LIF: Loop colostomy

SPOUT

Spout present:

- **Ileostomy** *(contents toxic to skin)*
- **Urostomy**

No spout: Colostomy

EFFLUENT (WHAT'S COMING OUT)

Hard stool – Colostomy

Soft stool – Ileostomy

Urine – Urostomy

Remember to feel the bag!

SURROUNDING SKIN QUALITY

Any inflammation or excoriations? – *infection / poor stoma maintenance*

ANY EVIDENCE OF COMPLICATIONS?

Haemorrhage – Peristomal skin inflammation

Parastomal hernia – Risk of bowel strangulation and necrosis

Prolapse – High output

Retraction – Obstruction

AUSCULTATION

Auscultate for bowel sounds:

- Absent bowel sounds – ileus
- High pitched tinkling indicates obstruction

MANAGEMENT OF ENTEROCUTANEOUS FISTULA

Management of ECF can be divided into the following phases:

1. Stabilisation within 24 to 48 hours.
2. Investigations within 7 to 10 days.
3. Decision: 7-10 days or after 4-6 weeks.
4. Definitive therapy when spontaneous closure is unlikely or after 4-6 weeks.
5. Healing: 5 to 10 days after closure.

FIRST: STABILISATION

RESUSCITATION:

Most patients with GI fistulas experience significant fluid and electrolyte imbalances. Resuscitation aims to restore intravascular fluid volume and to ensure a urine output of 30 mL/h or greater. The circulation volume deficits result from extracellular fluid losses, and replacement is best achieved with isotonic crystalloid solutions, such as normal saline or Ringer lactate. Simultaneous electrolyte repletion is necessary. Correction of acid base imbalance as patients with high output and proximal fistulae develop significant metabolic acidosis. Plasma oncotic pressure should be restored by exogenous albumin administration. Mortality in 42% in patients with a serum albumin <2.5 g/dL.

NASOGASTRIC TUBE:

As little evidence support its role, it should be removed if There is no obstruction, fistula is low in intestinal tract.

MANAGEMENT OF SKIN CARE:

The skin surrounding the fistula opening is exposed to intestinal contents, leading to excoriation and breakdown. Problems that may occur in skin around the fistula: wetness, burning pain, discomfort from skin oedema The goals of skin care are: containing the effluent, patient independence and mobility, and to prevent maceration, breakdown, cellulitis.

TECHNIQUES OF SKIN CARE:

1) Wound pouch dressing: One/two-piece design or clip closure. May be attached to a bed side bag or suction catheter.

2) Skin barriers: solid wafers (pectin based), powders (Pectin / Karaya based), paste, spray and wipes, ointments and creams (zinc/petroleum based)

3) Sump drainage: For fistulae draining with open abdominal wound. They are large bore drains or sumps with high pressure suction (better results).

Sump drain: a drainage device consisting of two tubes, one to allow fluid to be drained from a cavity and the other to allow air to enter the cavity to replace the fluid. It may be attached to a suction apparatus.

NUTRITIONAL MANAGEMENT:

Plays central role in management. Adequate circulation and tissue oxygenation is a must Is either enteral or parenteral.

TPN should be started in virtually all patients with an enterocutaneous fistula. In low output fistula (less than 200 mL/24 hr), enteral feedings may be considered. Enteral feeding may be initiated orally or via a catheter placed distal to the fistula.

There is an increased trend towards enteral support when possible due the following:

- Decreases nosocomial infection rate, particularly fungi
- Supports immunologic, hormonal, and barrier functions of gut
- Fistula closure rates slightly less than TPN alone
- Fistuloclysis (feeding via the intestinal fistula)

Malnutrition is present in 55-90% of patients with ECF and is responsible for much of the morbidity and mortality in these patients. Malnutrition should be considered if there is a body weight loss of at least 10% and if there is hypoproteinemia.

- Chapman and colleagues demonstrated that patients receiving optimal nutritional support (3000 calories per day) had a mortality rate of 12% as compared to 55% mortality among patients receiving a sub optimal nutritional regimen.
- Robauk and Nichdoff reported closure of 73% enteric fistulae in patients with adequate caloric supplementation but only 19% healed when nutritional support was inadequate.

CONTROL OF SEPSIS:

- Drainage if Intra-abdominal collections (percutaneous) mandatory.
- Laparotomy may be required for

— Extensive cellulitis/necrotising fascitis

— Incomplete percutaneous drainage of collections

— Disruption of anastomosis

Antibiotics (broad spectrum to control sepsis) to be withheld unless the patient is septic. Uncontrollable sepsis associated with 85% mortality (usually underlying cancer or radiation treatment).

CONTROL OF FISTULA DRAINAGE

The goal of controlling enterocutaneous fistula output is to prevent the intraperitoneal accumulation of intestinal contents and to protect the skin from the effects of the intestinal contents.

H2 antagonists/ Proton pump inhibitors: H2 blockers and octreotide may decrease the volume of flow through the fistula and accelerate healing.

Somatostatin / octreotide : Reduces fistula output, easing management of fluid and electrolyte losses. No improvement in fistula closure rates. Ineffective after 14 days. Somatostatin: inhibits gastric, pancreatic, biliary, and enteric secretions. Half-life in circulation is 1-3 minutes, must be given as continuous IV infusion with TPN. Rebound hypersecretion of growth hormone, insulin, and glucagons when infusion stopped.

Octreotide (somatostatin s synthetic analogue) Half life 2 hours, lack of rebound hypersecretion of hormones, is allowed for the subcutaneous or intramuscular administration.

Infliximab (monoclonal antibody) (in Crohn's disese): chimeric monoclonal antibody to tumor necrosis factor-alpha that has been demonstrated to heal as many as 50% of chronic intestinal fistulae in patients with Crohn disease. Adverse effects, including headaches, abscess, upper respiratory tract infection, and fatigue, occur in more than 60% of patients.

Oral tacrolimus (in Crohn's disese): Tacrolimus is a calcineurin inhibitor that suppresses pro- inflammatory cytokine production and T-cell activation. These immunosuppressant effects have been used to treat inflammatory bowel disease, especially fistulising Crohn's disease and refractory ulcerative colitis.

EMOTIONAL SUPPORT:

External drainage of enteric contents can be demoralising. Psychiatric evaluation and use of antidepressant drugs.

SECOND: DECISION

• 60-75% of ECF will close spontaneously.

• After 4-6 weeks of sepsis-free adequate nutritional support -> surgical management should be considered.

- Abscess drainage
- Patient general condition is very poor -> only abscess drainage should be done.
- Malignancies -> early operation

Conservative therapy: 90% of small intestinal fistulae that closed did so within 1 month. < 10% of the fistulae closed after 2 months, and none closed spontaneously after 3 months.

THIRD: DEFINITIVE THERAPY

usually occurs if the fistula fails to respond to medical treatment after 4 to 6 weeks. A low output-controlled fistula is a relative indication for surgery.

Patients with Crohn's, s/p radiation, or tumour have a high complication rate (40%) and a high recurrence rate (15%).

Before surgery:

- Patient should be amply resuscitated
- Drainage cultured
- Intraluminal and intravenous antibiotic
- Discontinuation enteral nutrition 1-2 day prior while continuing parenteral nutrition
- Operative approach preferably through a new incision.
- Best results are with definitive resection and end-to-end anastomosis.
- Simple closure of fistula tract associated with 41% failure rate, so secure abdominal wall closure over fistula is essential.

Post operatively:

- Post-op nasogastric decompression
- Feeding jejunostomy (for proximal fistulae)
- Post op continuation of nutrition with gradual shift from parenteral to enteral form.

Other operative interventions:

- Over-sewing of the fistula
- Exteriorisation of the proximal and distal ends of the intestine: This is performed in the presence of extensive intra-abdominal sepsis, for which primary anastomosis is not appropriate.
- Serosal patch with either jejunum or a defunctionalised Roux. The goal is to defunctionalise the intestinal segment containing the fistula.

New-non-surgical techniques: No good clinical trials, mostly case reports, may be tried in selected patients with an established controlled fistula, good nutritional status and free of infection) Fibrin glue, Histoacryl, Vacuum assisted closure, Flaps.

1- VAC

"Removes chronic oedema, leading to increased localized blood flow, and the applied forces result in the enhanced formation of granulation tissue"

2- Fistuloscopy with fibrin glue' injection: 13/14 patients successfully treated. High output fistulae required multiple sessions. Closure within 2-30 days.

FOURTH: HEALING

- In the postoperative period, it is necessary to ensure that the patient continues to receive full nutritional support
- Adequate protein and calories must be provided to maximize healing and minimize complications.
- Although enteral nutrition may be attempted early in the post-operative course, it is nearly impossible to meet the patient's entire nutritional demand by this route.
- Postoperative care will most likely include parenteral and enteral supplementation in an overlapping manner.
- After fistula closure, whether by spontaneous or surgical means, the patient will need to resume oral intake.
- This may be especially difficult in an individual who has had little or no oral intake for 4 to 6 weeks or more, and enlisting the assistance of a dietician and the patient's family is often helpful.

PROGNOSIS

- Patients with a wedge repair or oversewing of an ECF recurrence rate of 32.7%, compared with 18.4% if the ECF is resected or the anastomosis revised.
- The overall mortality in patients with all types of enterocutaneous fistulas is 6.5% to 21%.
- The major morbidity and mortality from ECFs are related to sepsis, fluid losses,

electrolyte imbalances and malnutrition.

- Recognising and treating these complications are the goals in the treatment of patients with ECF.

PREVENTION

- Appropriate hydration to prevent Hypotension and compromised circulation

- Anastomosis in healthy bowel with adequate blood supply; without tension
- Meticulous and precise haemostasis
- Selection of proper needle size, suture.
- Omental covering if possible
- Dead space obliterated with live tissue and properly drained
- Drains kept away from anastomosis site.

INTESTINAL OBSTRUCTION

The term 'intestinal obstruction' refers to any form of impedance to the normal passage of intestinal content through the small or large intestine. The normal flow of intestinal contents can be blocked by a mechanical (dynamic) obstruction or by a functional (adynamic) obstruction that occurs because of impaired intestinal motility. Each of the two entities will be discussed separately.

MECHANICAL OBSTRUCTION

in which peristalsis is working against a mechanical obstruction.

Mechanical obstruction may be complete (total blockage of the lumen) or incomplete (partial blockage). Because in the early stages of mechanical obstruction, peristalsis is heard, it is referred to as dynamic obstruction.

The site of obstruction determines wither it's either small-bowel or large-bowel obstruction. It may present *acutely* with dramatic symptoms when the obstruction is situated at any point between the second part of the duodenum and the cecum (acute intestinal obstruction). Alternatively, the presentation may be more insidious, over several weeks, when the obstruction is located in the colon (chronic intestinal obstruction). If the blood supply to the obstructed segment is not jeopardized, the obstruction is referred to as simple in order to distinguish it from those obstructions in which the blood supply is compromised at an early stage (strangulating intestinal obstruction).

Adhesions remain the most common cause of small intestinal obstruction 40%), followed by hernias 12%) and malignancy (both primary and secondary - 15%). In the large intestines, malignancy is the most common cause (65%), followed by complicated diverticular disease (10%) and volvulus (5%).

SIMPLE MECHANICAL OBSTRUCTION

ETIOLOGY

The various causes of simple mechanical intestinal obstructions can be grouped as extramural, intramural and intraluminal (see the table below);

Causes of simple mechanical obstruction

Extramural

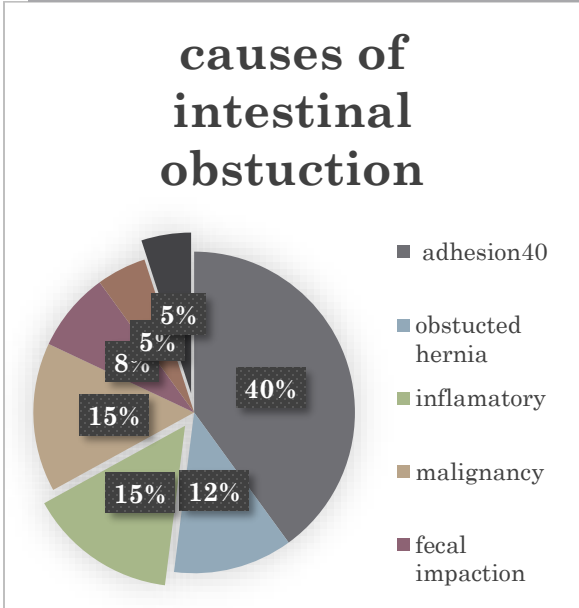
- **Adhesions, bands**
- **Hernias: external and internal**
- **Compression tumors (nodal tumor deposits)**

Intramural

- **Inflammatory disease; Crohn's disease, diverticular disease.**
- **Tumors; carcinomas, lymphomas, etc.**
- **Strictures**
- **Volvulus**
- **malignancy**

Intraluminal

- **Fecal impaction**
- **Swallowed foreign bodies**
- **Bezoars**
- **Gallstone**



• *Extramural obstructions;* These are due to extrinsic compression of the walls of the gut by bands, adhesions or tutors (particularly secondary deposits in lymph nodes). Adhesive small-bowel obstruction secondary to previous peritonitis or surgical intervention is the most common cause of intestinal obstruction. Adhesions are thought to result from a

reduction in peritoneal plasminogen **activating activity (PAA),** which in turn leads to a failure to break down the post-operative fibrinous adhesions that follow all intra-abdominal operations. The cause of reduction is unknown, but may be due to drying.

• *Intramural obstructions;* These are caused by lesions (neoplastic, inflammatory or cicatricial) arising from the wall of the intestine. The most common cause of this type of obstruction is carcinoma, usually of the colon.

• *Intraluminal obstructions;* Although intraluminal small-bowel obstructions are rare, chronic colonic intraluminal obstruction by impacted feces in constipated elderly patients is quite common. The small intestinal lumen maybe blocked by a swallowed object (children, mentally subnormal individuals) or a bolus composed of indigestible material (orange pith or hair bezoar, particularly in gastrectomized patients) or by a gallstone. The latter arises when a large gallstone in a chronically inflamed gallbladder that has become adherent to the duodenum erodes through the two organs by a process of pressure necrosis, thus entering the duodenum and becoming impacted lower down the small intestine, usually in the terminal ileum. The condition is known as gallstone ileus. By virtue of its origin it is always accompanied by a cholecystoduodenal fistula, which allows reflux of enteric contents and air into the biliary tract.

PATHOPHYSIOLOGY

Following the onset of obstruction, the distal bowel empties to a collapsed state whereas the proximal bowel becomes hyperactive, with vigorous peristaltic contractions, in an effort to overcome the obstruction. These cluster contractions are the cause of the *severe colicky abdominal pain* experienced by these patients. Additionally, the bowel proximal to the obstruction dilates due to the accumulation of swallowed air and increased intestinal secretions. The interface between air and fluid in the dilated loops accounts for the *air-fluid levels* seen in the erect abdominal film of these patients. The wall of the obstructed gut becomes edematous. This is the result of increased transudation across the capillary membrane as the venous drainage of the affected segments is impaired by the distension. The fluid and electrolytes that accumulate in the lumen of the obstructed bowel and. within its wall are effectively lost *(sequestrated third-space losses)* and contribute (together with vomiting, decreased oral intake and defective absorption) to the fluid and electrolyte deficit in these patients. Bacterial overgrowth occurs within the obstructed loops of intestine. Unless the distension is relieved, there is progressive occlusion (by stretching) of the intestinal intramural vessels such that, untreated, a mechanical intestinal obstruction leads to ischemia and eventually necrosis with *perforation* of the bowel.

STRANGULATING MECHANICAL INTESTINAL OBSTRUCTION

causes	
Direct pressure on the bowel wall	Hernial orifices Adhesions/bands
Interrupted mesenteric blood flow	Volvulus Intussusception
Increased intraluminal pressure	Closed-loop obstruction

Here, in addition to the luminal obstruction, the viability of the gut is compromised because of impairment of its blood supply at an *early stage.* Common examples include strangulation caused by bands, adhesions and tight hernial sacs **(strangulated hernias).** There are special forms that merit separate attention, including intussusception, volvulus, closed-loop obstruction and mesenteric infarction.

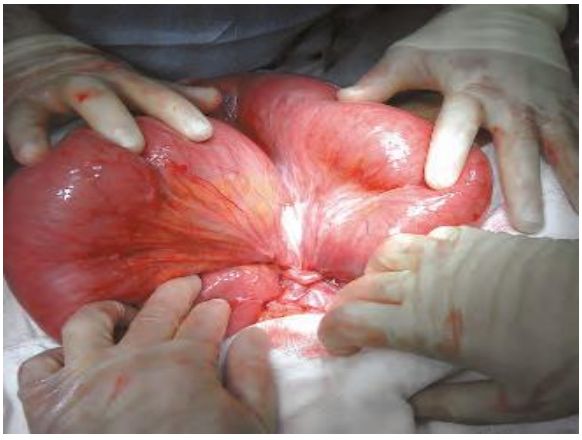


Figure 169 Band adhesion causing closed-loop obstruction

INTUSSUSCEPTION

This consists of telescoping of a loop of bowel inside itself (ileoileal, ileum inside ileum; ileocolic, ileum inside cecum or ascending colon). **Intussusception** occurs most commonly in infants and children usually 3- 18 months of age, but may be encountered in adults.

Further discussion on intussusception is made in the "abdominal emergencies" lecture in pediatric surgery.

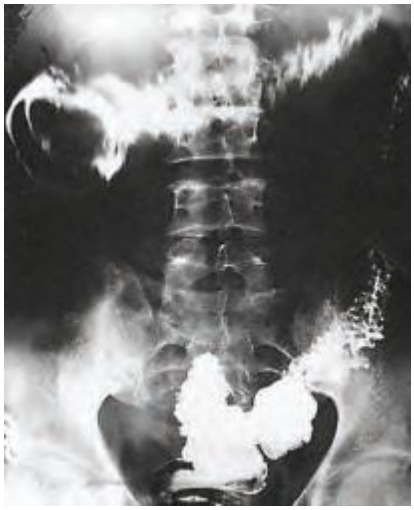


Figure 170 Claw' sign of iliac intussusception. The barium in the intussusception is seen as a claw around a negative shadow of the intussusception

VOLVULUS

Volvulus is a 360° twist of a loop or loops of intestine around it's mesentery The rotation causes early obstruction of the vascular pedicle supplying the affected portion. Risk factors for small intestinal volvulus include adhesions or bands between the anti- mesenteric aspect of the bowel and the anterior abdominal wall and congenital malrotation of the gut (Volvulus neonatorum, see pediatric surgery). Cecal volvulus may also occur as part of volvulus neonatorum or *de novo*. Sigmoid volvulus is encountered in the elderly, patients with chronic constipation and those with a redundant pelvic mesocolon. Unless recognized early, volvulus leads to intestinal infarction, which often involves large segments of the gut.

CLOSED-LOOP OBSTRUCTION

Although volvulus is an example of this type of obstruction i.e. segment of the affected bowel closed at proximal and distal ends, the term 'closed- loop obstruction' is usually reserved for a complete obstruction of the left colon (usually by an annular carcinoma of the descending or sigmoid colon) in the presence of a competent ileocecal valve. This prevents the proximal distended colon from decompressing into the small intestine. Meanwhile, smallbowel contents may continue to pass into the cecum through the one-way ileocecal valve. The result is a rapid build-up of pressure in the colon, with the brunt being taken by the cecum, which becomes markedly distended to the point of ischemia when it perforates, usually through a clear-cut hole (pistol-shot perforation).

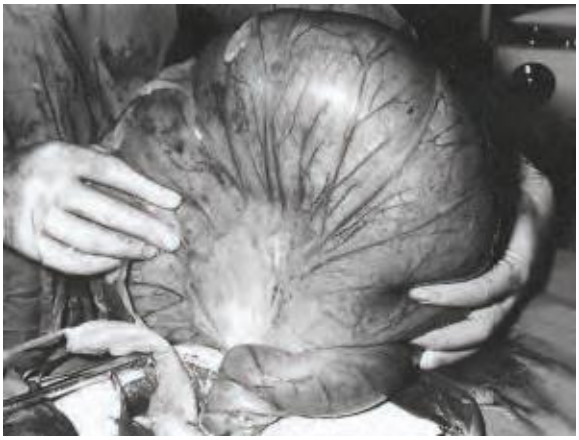


Figure 171 Volvulus of the sigmoid colon

MESENTERIC INFARCTION

In this serious condition, there is primary occlusion of the blood supply to the intestine as a result of thrombotic or embolic disease of the mesenteric vessels (see the small intestines lecture). Ischemia and edema will prevent the normal motility of the intestines. Although this a mechanical obstruction, it is a dynamic.

CLINICAL FEATURES MECHANICAL OBSTRUCTION

SYMPTOMS

Intestinal obstruction is described by the symptoms of *pain, vomiting, Abdominal distension* and *absolute constipation* (in ability to pass feces and flatus).

• *Pain* is usually the first symptom. The pain in mechanical small-bowel obstruction *is colicky in* nature, situated in the centre of the abdomen around the umbilicus and accompanied by hyper- peristaltic rushes that can easily be heard by the stethoscope. In colonic obstruction the pain is more of a discomfort and is situated in the suprapubic region. With extreme distention, the the nature of pain turns from colicky to *constant. Constant severe* pain is ominous and indicates either infarction of the bowel or the onset of peritonitis.

•*Vomiting* is a marked feature of **high** small-bowel obstruction but is rarely encountered in colonic obstruction. Initially, the vomit consists of food followed by bile-stained fluid, which later becomes feculent. This is caused by bacterial overgrowth in the obstructed small intestine.

•*Abdominal distension* becomes progressively more marked the lower the obstruction is situated and may reach extreme degrees in low colonic obstruction. It is caused by accumulation of gas and fluid within the obstructed bowel.

• *absolute constipation* indicates no passage of either flatus or feces, this should occur following emptying of the distal segment. This is not seen in partial obstruction in which the relative constipa**tion** is the case

SMALL VERSUS LARGE-BOWEL OBSTRUCTION

Colicky pain and vomiting are early features of small- bowel obstruction, with constipation appearing late and distension only really occurring if the obstruction is fairly distal. Compared to small-bowel obstruction, abdominal distension and constipation are common early features of large bowel obstruction, with colicky, pain being less marked and vomiting only appearing very late.

SIGNS

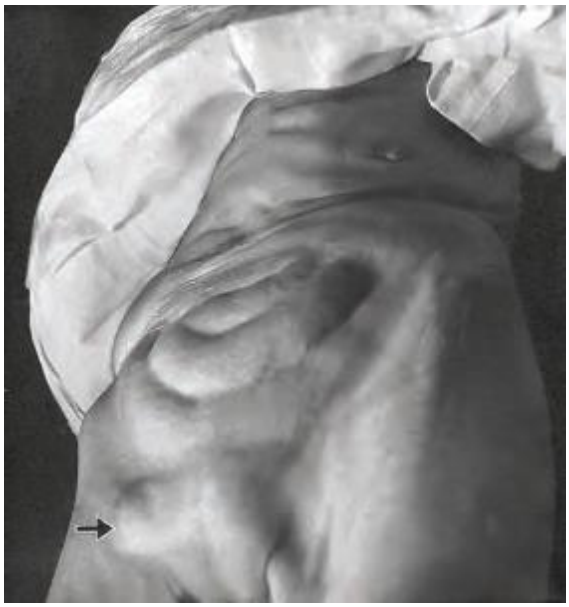


Figure 172 Visible peristalsis. Intestinal obstruction due to a strangulated

Vomiting, together with the sequestration of fluid in the dilated loops, the reduced oral intake and the defective absorption rapidly leads to dehydration with significant water and electrolyte deficits. Thus patients have signs of dehydration along with signs of electrolytes abnormality, including hyponatremia and hypokalemia. Continuing fluid loss will result in tachycardia and hypotension. Pyrexia is mild.

The distended abdomen is resonant to percussion on the anterior aspect but is dull towards the flanks. Auscultation confirms the presence of excessive peristaltic activity (normally they occur once every 8-10 seconds) that coincide with attacks of colic. Rectal examination in small bowel obstruction usually confirms an empty rectum. In colonic obstruction the findings may be fecal impaction or the presence of a rectal tumor, diverticular masses or malignant deposits in the pouch of Douglas. It is essential that the groins are examined for hernias.

FEATURES FOR STRANGULATION

The major concern for the attending clinician is to exclude the possibility of strangulation. Suspiciousness should be raised when there is sever constant pain, tenderness and rigidity, rising temperature,

The development of shock, pain refractory to conservative management.

When strangulation occurs in an external hernia, the lump is tense, tender and irreducible, there is no expansile cough impulse and it has recently increased in size. Patients with volvulus may show a palpable tympanic sausage shaped mass.

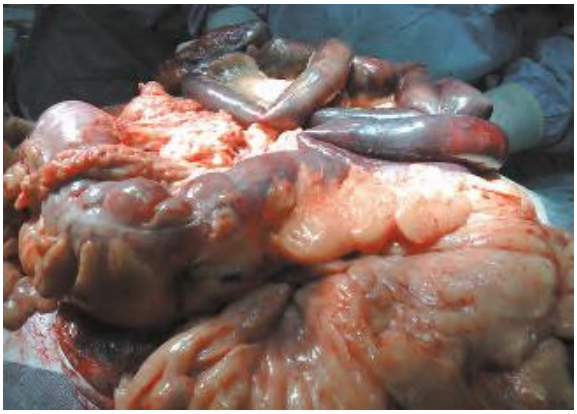


Figure 173 Ischaemic small and large bowel in a strangulated

INVESTIGATIONS

The essential investigations in patients with intestinal obstruction are complete blood count, urea and electrolytes, chest X-ray, plain erect and supine abdominal films.

BLOOD TESTS

Both the hemoglobin and PCV are elevated because of hemoconcentration. The white cell count is usually normal or slightly elevated unless there is bowel infarction and/or peritonitis.

The raised blood urea is the result of an element of prerenal failure due to the hypovolemia, serum sodium and chloride are usually low. Hypokalemia might be seen but Hyperkalemia may be observed in patients with infarcted intestine.

X-RAY STUDIES

An erect abdominal X-ray is take first to to outline airfluid levels. They represent the air-fluid interfaces when the air is above fluid, they are multiple and centrally placed in a ladder fashion in small-bowel obstruction and should be more numerous when the obstruction is distal. In large-bowel obstruction they are less numerous and located in the flanks and suprapubic regions. In adults, two inconstant fluid levels (one at the duodenal cap and the other in the terminal ileum) may be regarded as normal. The *routine* use of erect abdominal films is shrinking.

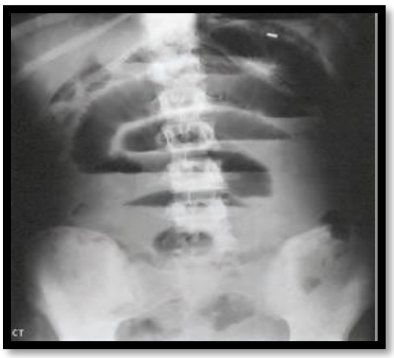


Figure 174 Fluid levels with gas above; 'stepladder pattern'. Ileal obstruction by adhesions; patient erect

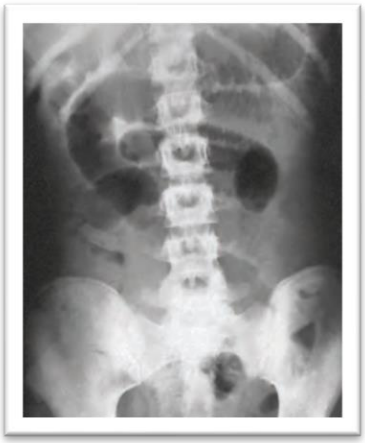


Figure 175 Gas-filled small bowel loop; patient supine

the supine film is used to assess distension of the intestine and helps to differentiate small from large intestine. The key is to look for bowel distention. Small bowel distension is usually more than 2.5 cm in diameter, but rarley exceeds 5 cm, while The colon can distend to 10 cm and more in severe cases. Its generally agreed upon that small intestinal loops are located centrally, while the colon is located peripherally. Dilated jejunum often exhibits parallel soft-tissue shadows that extend the *whole width* of the involved segment (due to the folds of the smallbowel mucosa, so called valvulae conniventes or plica circularis), whereas in the obstructed colon the haustra cause crescentic soft-tissue shadows that do not traverse the entire width of the

bowel. The obstructed ileum is relatively featureless. Other features include gas may be seen in the biliary tree or the presence of stones in gallstone ileus.

Features suggesting strangulation include loss of mucosal pattern, pneumatosis intestinalis and air in the portal vein.

In cecal volvulus, radiography may reveal a gas-filled ileum and occasionally a distended cecum. A barium enema may be used to confirm the diagnosis, with an absence of barium in the cecum and a bird beak deformity. In sigmoid volvulus, a plain radiograph shows massive colonic distension. The classic appearance is of a dilated loop of bowel running diagonally across the abdomen from right to-left, with two fluid levels seen, one within each loop of bowel.

Other investigations that may be necessary are contras examinations (water-soluble contrast swallow and meal, gentle barium enema) and sigmoidoscopy. A barium follow-through is contraindicated in the presence of acute obstruction and may be life-threatening.

MANAGEMENT

The management of intestinal obstruction is based on four principles (1) decompression of the obstructed gut;(2) replacement of fluid and electrolyte losses and (3) definitive management.

DECOMPRESSION

decompression is nowadays achieved by the insertion of a nasogastric sump suction tube (Salem). This is aspirated at least every hour and left draining into a bag in the intervening periods. The daily aspirate is measured and the amount used in calculating the daily fluid and electrolyte requirements. As well as facilitating decompression proximal to the obstruction, this also reduces the risk of subsequent aspiration during induction of anesthesia and post-extubation.

If the intestinal obstruction responds to conservative management, the daily amount of aspirate gradually

reduces and its nature changes to clear, often bile-stained fluid.

FLUID AND ELECTROLYTE THERAPY

Fluids and electrolytes are giver, through a peripheral venous line. As the major losses are water, sodium and chloride, the usual crystalloid solution consists of isotonic saline and 5% dextrose solution. Initially, large amounts are administered (1 L every 3-4 h) to replace the losses. However, this is not always that simple, and should largely be determined by the amount and composition of the nasogastric aspirate. An example of this is presented in the IV fluid therapy seminar. Please refer to it for more information on that topic.

OTHERS

Antibiotics are not mandatory but many clinicians initiate broad-spectrum antibiotics early in therapy because of bacterial overgrowth. Attention should be made to the nutrition of the patient.

DEFINITIVE TREATMENT

Definitive treatment is attempted after identification of the specific diagnosis. in some cases definitive treatment is mandatory, while in others, conservative management would be enough Monitoring of conservative management is by vital signs, change in the colour of the aspirate and reduction in its amount and the relief of symptoms. Surgery might be required in cases that require surgery as the only treatment (e.g. hernias), failure of conservative management (usually after 72 hours) and in cases of strangulation.

The following presents the management relevant to each condition;

- adhesions; conservative management is sometimes enough. Surgery for relieving adhesions is called enterolysis and involves division of the obstructing adhesions. Limiting recurrence of adhesion which is increased by opening the abdomen is by restricting division only to the obstructing adhesion and by covering the surface of the affected segment with omental patches.
- Gallstone ileus; At laparotomy, it may be possible to crush the stone within the bowel lumen, after milking it proximally. if not, the intestine is opened and the gallstone removed. If the gall stone is faceted, a careful check for other enteric stones should be made.
- Bolus obstruction; The management is similar to that for gallstone, with intraluminal crushing usually being successful.
- Bezoars; the lesion may be kneaded into the cecum, otherwise open removal is required.

- **Fecal impaction; manual removal of feces and/or oil retention enema.**
- **Inflammatory strictures; Standard surgical management consists of resection and anastomosis. In Crohn's disease, strictureplasty may be considered in the presence of short multiple strictures without active sepsis.**
- **Hernias and tumors are discussed elsewhere. In the large intestines, palliation of unresectable tumors can be achieved by shaping a proximal stoma, depending on the site of the tumor.**

When strangulation is diagnosed or even suspected, operation must be performed urgently (after rapid fluid resuscitation) to try to prevent infarction and perforation. *The patient is otherwise managed as for uncomplicated obstruction.* There are no specific investigations to help diagnose bowel strangulation, which is a clinical diagnosis best confirmed at laparotomy;

Differentiation between viable and non-viable tissue		
	Viable	Non-viable
Circulation	Dark colou becomes lighter Visible pulsation in mesenteric arteries	Dark colour remains No detectable pulsation
General appearance	Shiny	Dull and lustreless
Intestinal musculatur	Firm	Flabby, thin and friable
	Peristalsis may be observed	

- Vovlulus; cecal volvulus is treated according to the viability of the cecum. Viable cecum is reduced at the operation after being decompressed with a needle. A cecopexy (fixation of the cecum to the right iliac fossa) or cecostomy is then performed. Right hemicolectomy is performed for non-viable cecum. Sigmoid volvulus is treated temporarily with untwisting by Flexible sigmoidoscopy or rigid sigmoidoscopy. Failure will require laparotomy. After untwisting. Viable sigmoid colon is fixed to the posterior abdominal wall. For Non-viable sigmoid colon, sigmoid colectomy is the operation of choice.
- Intussusception, strangulated hernias and mesentric ischemia are discussed elsewhere

FUNCTIONAL OBSTRUCTION

This form of obstruction results from atony of the intestine with loss of normal peristalsis, in the absence of a mechanical cause. In the small bowel it is usually- referred to as paralytic ileus, whereas in the large bowel the term pseudo-obstruction is used.

PARALYTIC ILEUS

This term is used to describe a syndrome in which intestinal obstruction is due to absence of the normal peristaltic contractions, usually in the small intestines.

ETIOLOGY

Causes of paralytic ileus are shown in the table below.

Causes of paralytic ileus
• **Post operative ileus**

• **Metabolic**
  • **Hypokalemia**
  • **Uremia**
  • **Hyponatremia**
  • **Dehydration**
  • **Diabetic ketoacidosis**
  • **Hypothermia**
  • **Hypoxia**

• **Infective**
  • **Generalized peritonitis**
  • **Pancreatitis**

• **Drugs**
  • **Tricyclic antidepressants**
  • **General anesthesia**

• **Reflex ileus**
  • **Retroperitoneal hematomas**
  • **Retorperitoneal malignancies**
  • **Pelvic and spinal fractures**

Paralytic ileus is most commonly encountered after intra-abdominal surgery, when it is short-lived (few

days) and often referred to as physiological ileus. The temporary cessation of intestinal motor activity is due to handling and exposure of the intestinal loops.

Paralytic ileus may also be caused by spinal injuries and by the accumulation of retroperitoneal blood or irritant exudates that disturb the functional activity of the coeliac plexus and splanchnic nerves (retroperitoneal hematoma from renal injuries, ruptured abdominal aneurysms, acute pancreatitis, etc.), this is referred to as reflex ileus.

Infective paralytic ileus is the most serious and is secondary to peritonitis from any cause. Ileus may occur secondary to metabolic causes, most commonly due to hypokalemia and hyponatremia.

CLINICAL FEATURES

In contrast to, mechanical obstruction, in paralytic ileus, pain is not a feature and usually there is no bowel sounds (thus the term adynamic). Distension is seen and is usually tympanic to percussion. Radiologically, the abdomen shows gas filled loops of intestine with multiple fluid levels. The rectosigmoid gas bubble is not absent as opposed to what occurs later in complete mechanical obstruction.

In surgical practice, paralytic ileus is most commonly encountered in the post-operative period. In this setting the differentiation between mechanical due to postoperative adhesions and paralytic obstruction is difficult and often the clinical picture is mixed.

MANAGEMENT

The management of these patients is aimed at the cause. Post-operative ileus is treated conservatively with nasogastric decompression, intravenous fluids and parenteral nutrition (when prolonged). The passage of flatus and the presence of bowel sounds are ‘indicators of improvement (gastric and small bowel motility returns by 24-48 hours, colonic motility by 3-5 days).

PSEUDO-OBSTRUCTSBN

Pseudo-obstruction, sometimes referred to as Ogilvie's syndrome, is a syndrome that describes the presence of colonic obstruction for which no mechanical cause can be found.

ETIOLOGY AND PATHOGENESIS

It is postulated to be caused by an imbalance between sympathetic and parasympathetic activity, with unopposed parasympathetic activity. Some investigators suggest that it the result of impairment of the reflex circuits within the enteric nervous system that ensure normal peristaltic progression.

This rare functional obstruction is usually encountered in elderly patients with severe extra-abdominal illness or injury (heart failure, sepsis, trauma, etc.). Other documented associations include chronic administration of hypnotics and sedatives, lead toxicity, hypothyroidism and various neurological disorders.

CLINICAL FEATURES AND MANAGEMENT

The syndrome is characterized by massive dilatation of the colon, suggesting distal organic colonic obstruction. *The diagnosis is made by exclusion of organic disease.* Air-fluid levels are often absent in this condition. Untreated the dilatation is progressive and when it exceeds 10 cm in the cecum, rupture with peritonitis may ensue.

Treatment involves correction of the underlying cause, supportive management and decompression of the colon by passage of a rectal tube, sigmoidoscope or colonoscope. Operative decompression is rarely required.

THE APPENDIX

The vermiform (worm like) appendix is blunt ended tube-shaped structure that arises from the posteromedial wall of the cecum 2 cm below the ileocecal valve. The relationship of the base of the appendix to the cecum remains constant, whereas the tip can be found in a retrocecal, pelvic, subcecal, preileal, post- ileal, or right paracecal position (figure 1). The three taenia coli converge at the junction of the cecum with the appendix and can be a useful landmark to identify the appendix. The appendix can vary in length from less than 2 cm to greater than 25 cm; most appendices are 6 to 9 cm in length.

BLOOD SUPPLY

The appendix has its own mesentery, the mesoappendix, and its blood supply comes from the appendicular artery, a branch of the ileocolic artery.

HISTOLOGY

The lumen is irregular, being encroached upon by multiple longitudinal folds of mucous membrane lined by columnar cell intestinal mucosa of colonic type. In children, there are abundant lymphoid follicles in the submucosa, but these will atrophy with age. Crypts are present but are not numerous. In the base of the crypts lie argentaffin cells, which may give rise to carcinoid tumors.

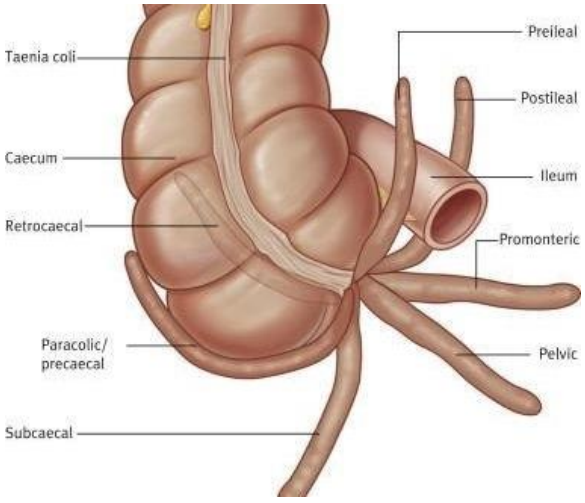


Figure 176 normal appendix variation

APPENDICITIS

Appendicitis is the most common cause of acute abdominal pain requiring surgery in Europe, Australia and the US and up to 16% of the population undergoes appendectomy. The lifetime rate of appendectomy is 12% for men and 25% for women, with approximately 7% of all people undergoing appendectomy for acute appendicitis. Appendicitis is most frequently seen in patients in their second through fourth decades of life, with a mean age of 31.3 years and a median age of 22 years. There is a slight male to female predominance (M: F 1.2 to 1.3:1).

ETIOLOGY

The etiology of acute appendicitis remains unclear. Obstruction of the lumen is the dominant causal factor in acute appendicitis. Fecaliths (inspissated faecal material, calcium phosphates, bacteria and epithelial debris) are the usual cause of appendiceal obstruction. Less-common causes are hypertrophy of lymphoid tissue (secondary to a viral infection), inspissated barium from previous x-ray studies, tumors, vegetable and fruit seeds, and intestinal parasites.

Diet may be involved (reduced fiber content) as the disease is very common in the West but rare in Africa, with a consequent slow transit time and alteration in bacterial flora.

PATHOGENESIS

Obstruction of the appendiceal lumen seems to be essential for the development of appendiceal gangrene and perforation. There is a predictable sequence of events leading to eventual appendiceal rupture. The proximal obstruction of the appendiceal lumen produces a closed-loop obstruction, and continuing normal secretion by the appendiceal mucosa rapidly produces distention; This raises the intraluminal pressure. Distention continues from continued mucosal secretion and from rapid multiplication of the resident bacteria of the appendix. As pressure in the organ increases, venous pressure is exceeded. Capillaries and venules are occluded, but arteriolar inflow continues, resulting in engorgement and vascular congestion. The inflammatory process soon involves the serosa of the appendix and in turn parietal peritoneum in the region, producing the characteristic shift in pain to the right lower quadrant.

As progressive distention encroaches upon first the venous return and subsequently the arteriolar inflow, the area with the poorest blood supply suffers most: ellipsoidal infarcts develop in the antimesenteric border. As distention, bacterial invasion, compromise of vascular supply, and infarction progress, perforation occurs, usually through one of the infarcted areas on the antimeseteric border.

Typically, two clinical syndromes of acute appendicitis can be discerned, acute catarrhal (non obstructive) appendicitis and acute obstructive appendicitis. The latter is characterized by a much more acute course.

CLINICAL FEATURES

HISTORY

Abdominal pain is the prime symptom of acute appendicitis. In the majority of patients, pain starts in the umbilical region and consists of a dull ache or colic (presumably from obstruction of the appendiceal lumen) with superimposed cramping (from peristalsis). After a variable period (1 to 12 hours), the pain shifts to the right lower quadrant of the abdomen as the inflamed appendix irritates the parietal peritoneum.

Anorexia nearly always accompanies appendicitis. It is so constant that the diagnosis should be questioned, if the patient is not anorectic. Although vomiting occurs in nearly 75% of patients, it is neither prominent nor prolonged and most patients vomit only once or twice, Nausea is reported by most patients. About 20% of patients have diarrhea and this may lead to a mistaken diagnosis of gastroenteritis.

In more than 95% of patients with acute appendicitis, anorexia is the first symptom, followed by abdominal pain, which is followed, in turn, by vomiting (if vomiting occurs). If vomiting precedes the onset of pain, the diagnosis of appendicitis should be questioned.

Many patients who are found at operations for appendectomy give a history of previous similar, but less severe, attacks of right lower quadrant pain. Pathologic examination of the appendices removed from these patients often reveals thickening and scarring, suggesting old, healed, acute inflammation.

PHYSICAL EXAMINATION

The signs of appendicitis depend on the whether the state is complicated or not.

- Non complicated appendicitis is minimal alterations of vital signs, usually with low grade pyrexia and mild but sustained tachycardia. Abdominal examination will reveal many of the following in most cases (most of these occur when the inflamed appendix lies in the anterior position);
- Inspection of the abdomen may show limitation of respiratory movement in the lower abdomen. Patients usually prefer to lie supine and they move cautiously.
- The patient is asked to point to where the pain began and where it moved (the pointing sign).
- Superficial palpation reveals tenderness and guarding is often maximal at or near McBurney's point (the located at the line between the umbilicus and anterior superior iliac spine on the junction of the lateral one third and medial two thirds; it localizes the base of the appendix, figure 2). Cutaneous hyperesthesia in the area supplied by the spinal nerves on the right at T10, T11, and T12 frequently accompanies acute appendicitis.
- Deep palpation may reveal the following;
    1. Rovsing's sign; Palpation in the left iliac fossa may reproduce the pain in the right iliac fossa.
    2. Blumberg sign: Direct rebound tenderness is elicited by deep palpation at the McBurney's point with sudden withdrawal of the hand" with quick release"

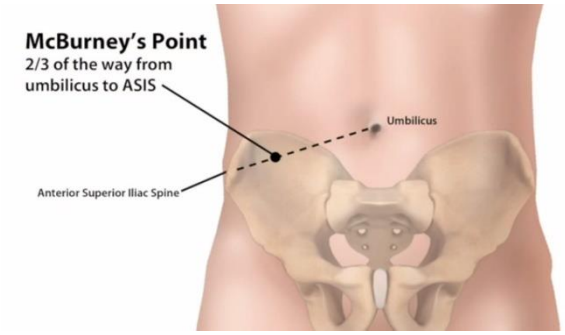


Figure 177 McButney's Point

3. Dunphy's sign ; A sharp pain in the right lower quadrant after a voluntary cough.

Other signs include;

- Psoas stretch sign; the patient may find it painful to extend the right hip owing to irritation of the psoas muscle.
- Obturator sign; is an indicator of irritation to the obturator internus muscle. Internal rotation of the hip will cause spasm of the muscle with pain in the lower abdominal region.

ANATOMICAL VARIATIONS

The table below presents the clinical features associated with different anatomic locations of the appendix?

Clinical features of anatomical variations	
Retro-cecal	• Possible absence of rigidity • tenderness on deep palpation • positive psoas sign
pelvic	• Diarrhea and frequency of micutrition. • Absence of abdominal findings, lower abdominal tenderness on deep palpation • tenderness on rectal examination. • Positive psoas and obturator signs.
postileal	• Diarrhea • Retching • Tenderness immediately right to the umbilicus • Hard to identify

INVESTIGATIONS

The diagnosis of acute appendicitis is made largely on clinical grounds but there are some investigations that maybe of value:

- WBC; The majority of patients have a polymorphonuclear leukocytosis (> 12 x 109/L).
- Plain abdominal X-ray; this is of little diagnostic value in a patient with obvious symptoms and signs, it is indicated if there is some clinical suspicion of intestinal obstruction or ureteric colic or pain referred from a right lower lobe pneumonic process. The presence of a fecalith is rarely noted on plain films.
- Urinalysis; this can be useful to rule out the urinary tract as the source of infection. It may show white blood cells (pyuria) and hematuria if the ureters and bladder are involved.
- Ultrasound examination of the pelvis is particularly useful in female patients (exlusion of gynecological pathology), in distinguishing between an appendix mass and an abscess, and in making a specific diagnosis of appendicitis, although this is not as yet routine practice. Graded compression sonography has been suggested as an accurate way to establish the diagnosis of appendicitis. Sonographically, the appendix is identified as a blindending, nonperistaltic bowel loop originating from the cecum. With maximal compression, the diameter of the appendix is measured in the anteroposterior dimension. A scan is considered positive if a noncompressible appendix 6 mm or greater in the anteroposterior direction is demonstrated.
- Laproscopy; Differentiating acute gynecologic pathology from acute appendicitis can be effectively accomplished by using the laparoscope
- CT; selective use for right iliac fossa mass, but several features (some yet to be identified) are indicative of appendicitis (will not be discussed here). It is also used to rule out some differential diagnoses.

DIAGNOSIS

A number of clinical and laboratory-based scoring systems have been devised to assist diagnosis. The likelihood of appendicitis can be ascertained using the Alvarado scale. This scoring system was designed to improve the diagnosis of appendicitis and was devised by giving relative weight to specific clinical manifestation. The below table lists the eight specific indicators identified. Patients with scores of 9 to 10 are almost certain to have appendicitis; there is little advantage in further workup, and they should go to the operating room. Patients with scores of 7 to 8 have a high likelihood of ap-

pendicitis, while scores of 5 to 6 are compatible with, but not diagnostic of appendicitis.

The Alvrado score		
	Manifestations	Value
Symptoms	Migration of pain	1
	Anorexia	1
	Nausea/vomiting	1
Signs	RLQ tenderness	2
	Rebound	1
	Elevated temperature	1
Laboratory values	Leukocytosis	2
	Left shift	1
Total Points 10		

DIFFERENTIAL DIAGNOSIS

The differential diagnoses of acute appendicitis and their differentiating features are shown in the table on the side. The differential diagnoses in children is discussed in the pediatric surgery lecture on that topic (in children they include mesenteric adenitis, meckel's diverticulitis, primary peritonitis, omental torsion, deep iliac lymphadenitis).

MANAGEMENT

Because of the risk of rupture of the acutely inflamed appendix in the first 12 hours, emergency surgery by appendectomy is mandatory. IV fluids are given to establish a good urine output after urinary catheterization is done and prophylactic antibiotics are given in all patients to prevent postoperative infections along with the control of local sepsis.

Differential diagnoses of acute appendicitis	
Old age	
Diverticulitis	CT scan to differentiate
Intestinal obstruction	In elders, conservative management with lapratomy
Colonic carcinoma	Palpable mass, unexplained anemia, altered bowel habit. Require CT scan
Mesenteric infarction	Post prandial pain, requires angiography
Adults	
Ureteric colic	Radiates to the groin, severe pain, diagnosed by urinanalysis
Perforated peptic ulcer	History of upper abdominal pain that suddenly increased in severity, gas under diaphragm.
Testicular torsion	Examination of the scrotum rules out.
Regional enteritis	Weight loss, diarrhea, serum titers for Yersinia
Pancreatitis	Pain is diffuse, relieved by bending forward, amylase levels are very high.
Rectal sheath hematoma	History of harsh exercise or anticoagulation, mass and pain with no gastrointestinal upset.
Female	
Mittelschmerz	No systemic upset, midcyclic pain
Pelvic inflammatory disease	Bilateral pain, vaginal discharge, dysmenorrhea, dysparunia, dysuria.
Pyelonephritis	Chills, flank pain and abnormal urinanalysis.
Ectopic pregnancy	Amenorrhea, positive pregnancy test, tenderness on bimanual examination hemoperitoneum
Torsion or rupture of an ovarian cyst	Difficult, requires gynecological counseling.

The process of conventional appendectomy involves opening a skin incision perpendicular to the line containing McBurney's point (gridiron incision), sometimes using other incisions like the lanz

incision (2 cm below the umbilicus on the mid-inguinal line). When the diagnosis is in doubt, particularly in the presence of intestinal obstruction, a lower midline abdominal incision is to be preferred. The deeper layers are divided until reaching the cecum where the base of the appendix is identified by the point of convergence of the teniae coli. The mesoappendix is identified, clamped, divided and then ligated, the appendix is then removed after crushing and ligation at its junction with the cecum.

Laproscopic appendectomy is useful for women of child bearing age when the diagnosis of appendicitis cannot be differentiated from other gynecological differential diagnoses. Thus, it is needed as both a therapeutic and diagnostic tool.

COMPLICATIONS OF ACUTE APPENDICITIS

APPENDICEAL RUPTURE

It has been suggested that delays in presentation are responsible for the majority of perforated appendices. Appendiceal rupture occurs most frequently distal to the point of luminal obstruction along the antimesenteric border of the appendix. Rupture should be suspected in the presence of fever greater than 39 °C and a white blood cell count greater than 18 x 109/L. In the majority of cases, rupture is contained and patients display localized rebound tenderness. Generalized peritonitis will be present if the walling-off process is ineffective in containing the rupture. Acute consequences of perforation include generalized peritonitis, and abscess formation. Treatment is appendectomy, peritoneal irrigation, and broad-spectrum intravenous antibiotics for several days.

APPENDICULAR MASS AND ABSCESS

In 2 to 6% of cases, an ill-defined mass will be detected on physical examination, This could represent a phlegmon, which consists of matted loops of bowel adherent to the adjacent inflamed appendix, or a periappendiceal abscess. Patients who present with a mass have a longer duration of symptoms, usually at least 5 to 7 days. An abscess is usually located within the mass and should be suspected when the mass fail to resolute or when there is a swinging pyrexia.

CT scan may be beneficial in guiding therapy. Phlegmons and small abscesses can be treated conservatively with intravenous antibiotics; well-localized abscesses can be managed with percutaneous drainage; complex abscesses should be considered for surgical drainage. Interval appendectomy performed at least 6 weeks following the acute event has classically been recommended for all patients treated either nonoperatively or with simple drainage of an abscess.

OTHER COMPLICATIONS

other complications include intraperitoneal and pelvic abscesses, fecal fistula usually following drainage of an abscess, recurrent intestinal obstruction due to adhesions, portal pyemia and post-operative wound infection.

APPENDICITIS AND PREGNANCY

Appendicitis is the most frequently encountered extrauterine disease requiring surgical treatment during pregnancy, it is more frequent during the first two trimesters. As fetal gestation progresses, the diagnosis of appendicitis becomes more difficult as the appendix is displaced laterally and superiorly. Suspicion is made with the presence of new onset vomiting, abdominal pain, leukocytosis and confirmed by ultrasonography. Intervention with laproscopy is mandatory, because the fetal and maternal mortality associated with appendiceal rupture outweighs that associated with the risk of preterm birth resulting from the operation.

APPENDICULAR NEOPLASMS

The appendix is the most common site for carcinoid tumor formation and carcinoid tumor of the appendix is the most common appendiceal neoplasm, argentaffine cells are the cells of origine.

The vast majority are found incidentally at the time of appendectomy, usually at the tip of the appendix. Rarely they arise at the base of the appendix and may then obstruct the lumen causing acute appendicitis. The vast majority of appendiceal carcinoids are less than 1 cm in diameter and these do not metastasize. Carcinoid tumors greater than 1.5 cm in diameter can and do metastasize. Carcinoid syndrome is rarely associated with appendiceal carcinoid unless widespread metastases. Thus while appendectomy is curative for a carcinoid less than 1

cm in diameter, tumors greater than 1.5 cm require a right hemicolectomy with radical removal of the ileocecal lymph nodes.

Adenocarcinoma is a rare tumor and accounts for approximately 15% of all malignant tumours of the appendix. It may arise from the base of the appendix. Treatment is right hemicolectomy irrespective of exact location.

Simple mucocele of the appendix is rare. It arises as a sequel to obstruction of the appendix without the onset of infection. The appendix becomes distended by mucoid secretion and the normal mucosa becomes replaced by a single layer of mucus-secreting cells. Eventually the lesion may calcify. Malignant mucocele on the other hand is a papilliferous cystadenoma or cystadenocarcinoma consisting of mucus-secreting cells. It often leads to pseudomyxoma peritonei (the formation of mucus within the peritoneal cavity) following rupture and spillage of the mucus-secreting cells.

ANORECTAL CONDITIONS

SURGICAL ANATOMY

The anal canal commences from the rectum at the level of the anorectal ring or bundle, and ends at the anal verge (figure 1). It is 3-4 cm in males and is shorter in females.

ANAL CANAL ANATOMY

-ANORECTAL RING

-it is formed by the joining of the puborectalis muscle, the deep external sphincter, conjoined longitudinal muscle and the highest part of the internal sphincter, can be felt with the finger as thickened ridge, especially on its posterior and lateral aspects

-THE PUBORECTALIS MUSCLE

originates on the posterior aspect of the pubis, forms a sling around the rectum (figure 2) and returns to the posterior aspect of the pubis, this pulls the anorectal junction anteriorly forming an acute angle which is important in maintaining continence (figure 2). The levator ani muscles are also

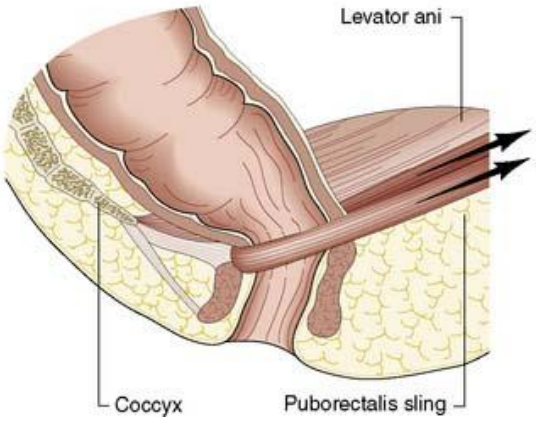


Figure 179 the anorectal ring formd by the joining of the puborectails muscle

important in maintaining the relationship of the anus and rectum during defecation, as well as providing support for the rectum.

-THE EXTERNAL ANAL SPHINCTER

(which guards the anus) consists of a ring of skeletal muscle fibres that encircles the distal portion of the anal canal. This sphincter is under voluntary

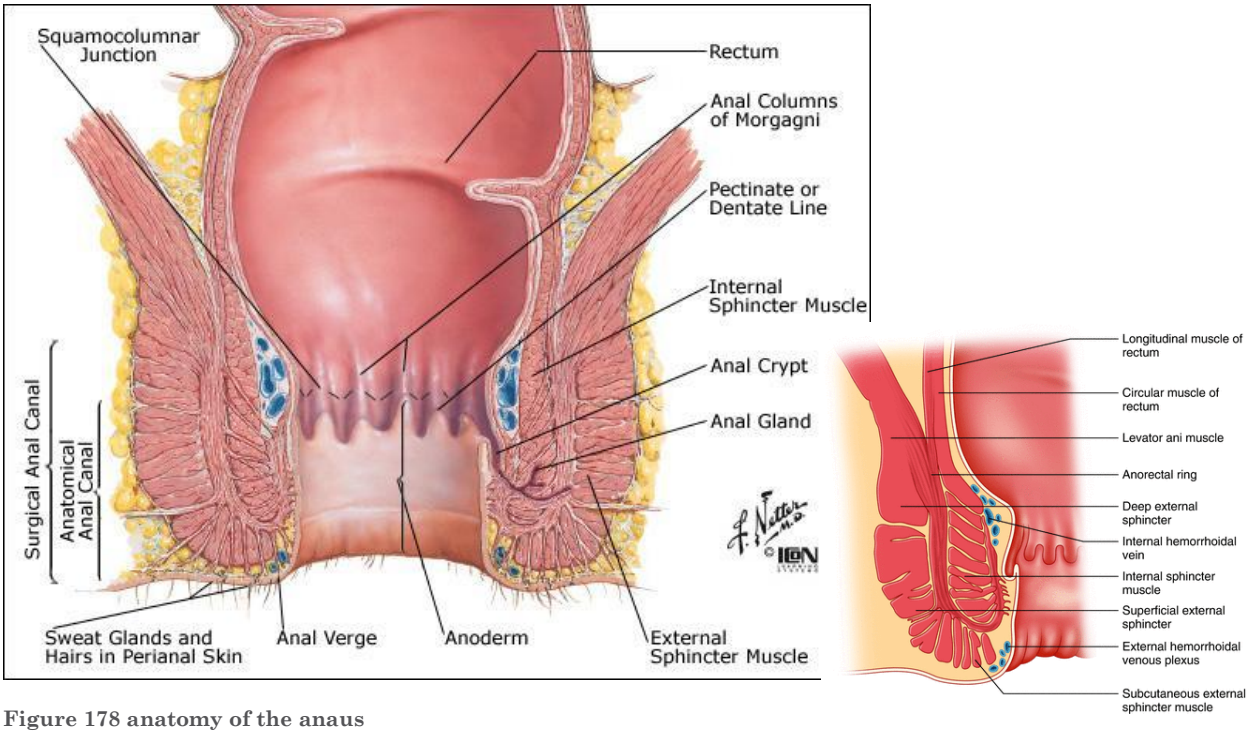


Figure 178 anatomy of the anaus

control. The external sphincter has 3 parts; deep, superficial and sub cutaneous.

The deep part blends with the fibres of the puborectalis ring.

- INTERNAL ANAL SPHINCTER

The circular muscle layer of the muscularis externa in this region forms it, the smooth muscle cells which are under involuntary control and are continuous with the circular muscle of the whole gastrointestinal tract.

-THE INTERSPINCTERIC SPACE

Between the external sphincter muscle laterally and the longitudinal muscle medially exists as a potential space, the intersphincteric space. This plane contains the intersphincteric anal glands (these glands have ducts that open directly on to the dentate line) and is also a route for the spread of pus, which occurs along the extensions of the longitudinal muscle layer. The plane can be opened surgically to provide access for operations on the sphincter muscles.

the perianal fascia continues from the adventitia of the anal canal and separates the perianal region into an ischioanal (perianal) fossa below and ischiorectal fossa above. The pelvirectal space lies above the levator ani and surrounds the rectum in the pelvic region

LINING OF THE ANAL CANAL

Above the dentate or pectinate line, lies the transition point between rectal mucosa (columnar epithelium) and squamous anoderm (stratified squamous epithelium). The 1 to 2 cm of mucosa just proximal to the dentate line shares histologic characteristics of columnar, cuboidal, and squamous epithelium and is referred to as the anal transition zone (ATZ). The lower margin of this zone is hilton's white line (rather than the dentate line). Outside the anal canal at the anal verge, there is normal skin composed of stratified squamous epithelium with skin appendages (sweat glands, hair follicles and sebaceous glands)

The dentate line is surrounded by longitudinal mucosal folds, known as the columns of morgagni, into which the anal glands empty (figure 1). Each column containing a terminal branch of the superior rectal artery and vein. The folds are most prominent in the left lateral, right posterior and right anterior sectors where the vessels form prominent anal cushions (figure 3). the anal cushions contain a submucosal venous plexus that connects with portal system. This communicates with another submucosal plexus that is located beneath the anoderm, below the dentate line (inferior hemorrhoidal plexus, figure 3).

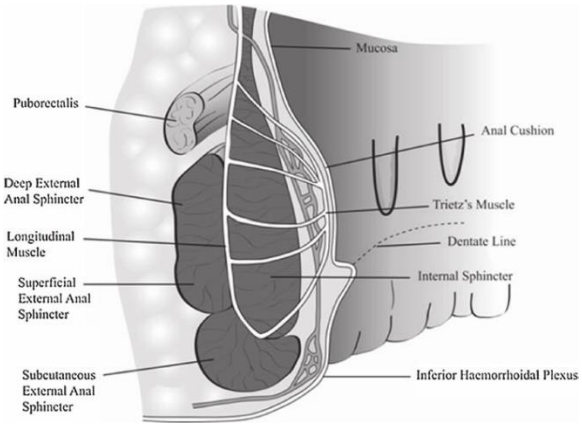


Figure 180 the cushions and the inferior hemorrhoidal plexus

BLOOD SUPPLY

The arterial supply of the anorectum is via the superior, middle, and inferior rectal arteries. The inferior rectal arteries branches of the internal pudendal arteries provide blood to the anal sphincters and epithelium.

The venous drainage of the anorectum is via the superior, middle, and inferior rectal veins draining into the portal and systemic systems. The superior rectal veins drain the upper and middle thirds of the rectum. They empty into the portal system via the inferior mesenteric vein. The middle rectal veins drain the lower rectum and the upper anal canal into the systemic system via the internal iliac veins. The inferior rectal veins drain the lower anal canal, communicating with the pudendal veins and draining into the internal iliac veins.

LYMPHATIC DRAINAGE

lymph from the upper half of the anal canal flows upwards to drain into the postrectal lymph nodes and from there goes to the para-aortic nodes via the inferior mesenteric chain . Lymph from the lower

half of the anal canal drains on each side , first into the superfecial and then the deep inguinal group of lymph nodes .

INNERVATION

The external sphincter is under voluntary control, and is supplied by the pudendal nerve. The internal sphincter is an involuntary smooth muscle and is innervated by the autonomic nervous system.

The internal sphincter is in a state of tonic contraction that maintains resting intra-anaT pressure. Sensation can be felt below the dentate line as the anoderm represents part of the normal skin. Above the dentate line the rectal mucosa is senseless as it does not contain somatic sensory nerve endings.

CONTROL OF CONTINENCE

The control of continence is achieved through several mechanisms and these include;

The maintenance of pressure inside the anal canal by tonic contraction of the internal sphincter.

Closure of the anus by the external sphincter voluntarily.

• The formation of an acute angle by the action of anterior pulling produced by the puborectalis muscle.

• The closure of the canal by the anal cushions.

HEMORRHOIDS

Hemorrhoids (piles) are vascular cushions located in the anal canal. Whereas, Hemorrhoidal disease is a Dilation of anal venous structures causing protrusion and /or bleeding Internal hemorrhoids are located proximal to the dentate line and are covered by insensate rectal mucosa Painless bleeding and 4 grades according to level of prolapse. they represent collapse of the anal cushions. External hemorrhoids are located distal to the dentate line and are covered with Richly innervated anoderm , Painful thrombosis and No grading, either present or absent.

They represent collapse or congestion of the inferior hemorrhoidal plexus (figure 3). Mixed hemorrhoids (interoexternal piles) result from progression of the latter to involve both haemorrhoid plexuses , and are best thought of as being external extensions of internal haemorrhoid .

hemorrhoids are extremely common, affecting nearly half of the population at some time in their lives.

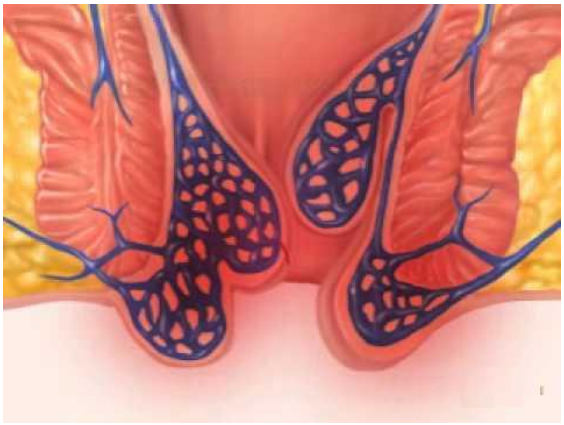


Figure 181 mixed hemorrhoids

ETIOLOGY

The direct mechanism underlying the formation of hemorrhoids is still under investigation. Several risk factors have been associated with their development and are shown in the table below.

Risk factors for developing hemorrhoids

- locale's. e.g. anorectal deformity
- abdominal e.g. ascites
- pelvic, e.g. pregnancy (couid be hormonal) , uterine neoplasm (fibroid , carcinoma of the cervix or uterus) , bladder carcinoma .
- neurological,e.g. multiple sclerosis
- Diarrhea
- Increased pelvic pressure (ascites, tumors)

Hemorrhoidal disease is no more common in patients with portal hypertension than in the normal population. however, Rectal varices may occur and may cause hemorrhage in these patients.

It might be that the initial event is engorgement of the submucosal veins (due to increased intrabdominal pressure or increased portal pressure with resultant degeneration of the surrounding

connective tissue support, eventually resulting in collapse of the whole tissue. Some investigators suggest that a hemorrhoidal tissue represent a physiological change with aging that involves enlargement of the cushion and is only abnormal when symptomatic.

Histologically, can result from changes in the erectile tissue that forms part of the continence mechanism, such as hyperplasia of the 'corpus cavernosum recti.

CLINICAL FEATURES

Internal hemorrhoids may prolapse or bleed, but rarely become painful as they are covered with a rectal mucosa. bleeding is separate from the stool and appears on toilet paper. If bleeding is chronic and prolonged, signs and symptoms of anemia may be present.it is painless, unless there is thrombosis, ulceration, or gangrene. Due to incomplete closure of the anus, the patients are usually mildly incontinent to flatus and mucus. Because of the mucous discharge, the patients usually have anal itching (pruritis ani).

Hemorrhoids are classified according to the extent to which they prolapse through the anal canal into the following;

- First degree : Do not prolapse Painless bleeding , The anoscope must be used to visualize them and Cannot be diagnosed with PR examination
- second degree (grade II); prolapse during defecation and then return spontaneously into the anal canal;
- third degree (grade III); remain outside the anal margin unless replaced digitally.
- Fourth Degree: Not reducible and Strangulated, predominantly prolapsed

On examination, the patient often has an engorged dilated sac at the 3,7 and 11 o'clock positions of the anal canal when the patient is in the lithotomy position. Although less frequent, but hemorrhoids may occur secondary to the presence of colorectal, cancer (pressure on the draining veins), thus (Digital examination is essential to exclude carcinoma and provides a useful measure of anal tone.)

External hemorrhoids are associated with mild symptoms including pain after defecation. They are apparent on examination. When multiple give 'figs' like appearance.

DIFFERENTIAL DIAGNOSIS:

- Rectal mucosal prolapse
- Hypertrophied anal papillae
- Rectal prolapse
- Carcinoma
- Melanoma
- Intersphincteric abscess

INVESTIGATIONS

A CBC is important in patients to exclude anemia. Proctoscopy is needed to demonstrate internal piles, which are seen bulging into the lumen as the proctoscope is withdrawn. Sigmoidoscopy is important in patients over 40 years,if there is a history of bleeding or any symptoms that leads to suspiciousness in malignancy.

MANAGEMENT

Hemorrhoids are managed when symptomatic. When there are mild symptoms or when the hemorrhoid is a grade I or is external, treatment includes a high fibre diet, prescribing laxatives for constipation and applying local creams, many of which contain local anesthetic agents or steroids.

Grade I and II hemorrhoids are treated by minor procedures which are performed on an outpatient basis. They include application of small rubber bands to strangulate the pile (using a special Barron's bander, figure 5); injection sclerotherapy; and the application of heat by infrared photocoagulation.

Grade III and IV hemorrhoids are approached surgically. The operation most commonly performed is the one described by Milligan and Morgan in which the hemorrhoidal masses are excised together with over- lying mucosa and some skin (open hemorrhoidectomy). This leaves skin and mucosal defects which heal by secondary intention and wound contraction. A skin bridge must be preserved between each wound to prevent the serious late complication of anal stenosis. When the wound is closed with absorbable suture, it is called Closed hemorrhoidectomy.

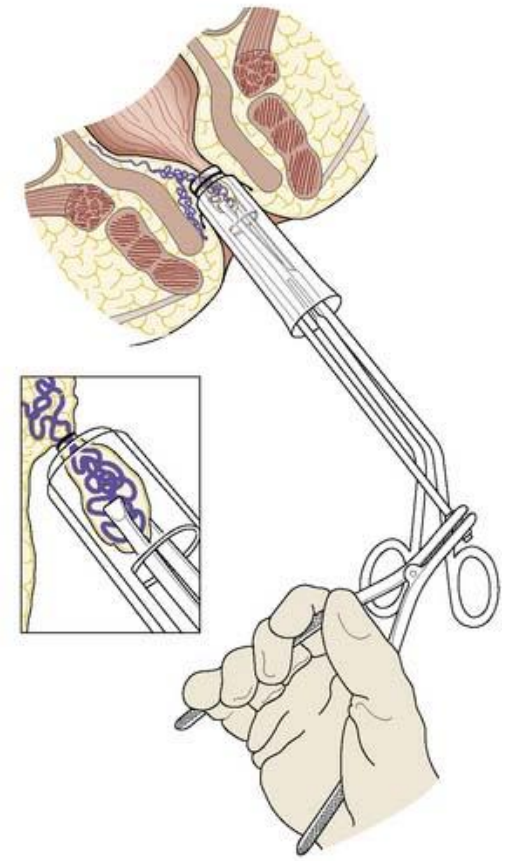


Figure 182 Barron's bander

SURGERY

INDICATIONS

The indications for haemorrhoidectomy include:

1 - third with a large external component

2 - fourth-degree haemorrhoids;

3 - second-degree haemorrhoids that have not been cured by non-operative treatments;

4 - fibrosed haemorrhoids;

5 - Acute hemorrhoidal attack (with gangrene, severe ulceration= emergency)

COMPLICATIONS OF HEMORRHOIDS

Complications of haemorrhoidectomy
Early
• Pain
• Acute retention of urine
• Reactionary haemorrhage
Late
• Secondary haemorrhage
• Anal stricture
• Anal fissure
• Incontinence

The bleeding mainly occurs externally but it may continue internally after the bleeding haemorrhoid has been retracted or has been returned.

Thrombosis usually occur in external hemorrhoids as a result of veins rupture and leakage of blood under the skin. A patient with a thrombosed external hemorrhoid may present with complaints of an acutely painful mass. The diagnosis of thrombosed hemorrhoids is usually obvious on inspection as an edematous, congested purplish mass seen at the anal margin.

Strangulated hemorrhoids refers to internal hemorrhoids that have prolapsed to the outside and then been strangulated by the sphincter muscles. Strangulated hemorrhoids are even more painful, and the strangulated mass may become necrotic or even ulcerated. Strangulated internal hemorrhoids usually become thrombosed.

thrombosed external hemorrhoids are treated by excision outside the mucocutaneous junction, which can be done in the office or emergency room with the wound left open. If the thrombosis is more than 48 hours old, the patient is treated with non-surgical management.

Major hemorrhage, resulting in significant hypovolemia and anemia, is unusual.

ANAL FISSURE

Anal fissure or fissure in ano is a common condition characterized by a linear ulcer, longitudinal ulcer affecting the anal canal below the dentate line from the anal transition zone to the anal verge. Fissures may be acute and settle spontaneously, but chronic anal fissure is defined as an ulcer that has been present for at least 6 weeks.

SURGICAL ANATOMY

About 90% are located on the posterior midline, with 10% being on the anterior midline usually in women after child birth. fissures affecting areas other than the midline should raise the suspicion of Crohn's disease or immunodeficiency. A fissure is a linear ulcer, longitudinal ulcer affecting the anal canal below the dentate line from the anal transition zone to the anal verge. There is often little in the way of granulation tissue in the ulcer base. In chronic fissures, owing to failed attempts at healing, there may be a tag of skin at the lowermost extent of the fissure, known as a 'sentinel pile'. At the proximal extent of the fissure there may be a hypertrophied anal papilla (figure 6).

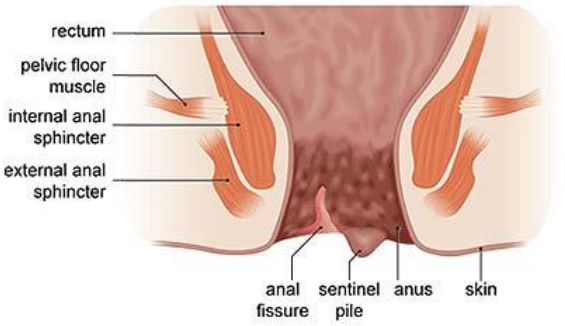


Figure 183 characteristic of anal fissure

ETIOLOGY AND PATHOPHYSIOLOGY

Most fissures are idiopathic, but it is clear that the pathophysiology involves ischemia in the base of the ulcer, associated with marked anal spasm and a significantly raised resting anal pressure. Successive bowel motions provoke further trauma, pain and anal spasm, resulting in a vicious circle of anal pain and sphincter spasm that causes further trauma to the anal mucosa during defecation.

Fissures located outside the midline should raise the suspicion of crohn's disease and immunodeficiency.

CLINICAL FEATURES

The most common symptoms are pain on defecation in a young patient. There is often associated rectal bleeding of the outlet type; with blood on the paper or dripping into the pan after passing the motion. In the history patients may report an episode of constipation.

The diagnosis is confirmed by gently parting the superficial part of the anal sphincter with the gloved fingers to reveal the characteristic linear ulcer. In chronic fissures, There may be an associated 'sentinel pile', which consists of heaped-up skin at the lowermost extent of the linear ulcer (figure 7).

It is often too painful to perform a digital rectal examination or a proctoscopy, and so this is best left until after treatment is instigated.

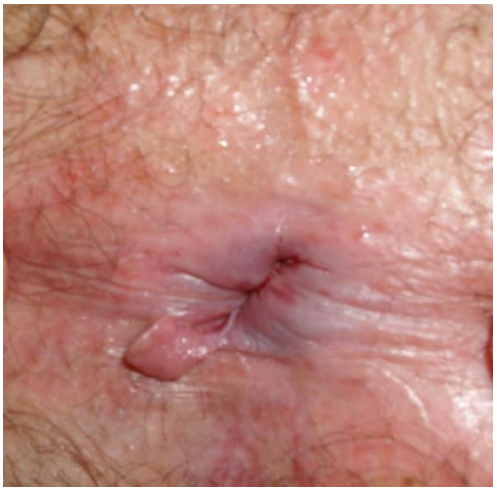


Figure 184 chronic anal fissure

The condition most commonly affects people in their twenties and thirties, with a slight male preponderance. Anal fissures in children are discussed in the pediatric surgery lecture (gastrointestinal bleeding).

MANAGEMENT

Many acute fissures resolve spontaneously. Until then treatment with sitz paths or stool softeners is sufficient.

treatment should be reserved for chronic symptoms of 6 weeks' or more duration, and should commence after exclusion of inflammatory bowel disease and immunodeficiency. treatment is aimed at alleviating pain and anal spasm in order to break out of the vicious circle and prevent ischemia (caused by pressure of the spastic sphincters on the supplying arterioles) from delaying healing. Stool softeners have no curative value. medical sphincterotomy (treating spasm with medications) is the first-line treatment of choice, using topical nitrates (glyceryl trinitrate 0.2-0,5% or 0.5% diltiazem) as a cream applied 12-hourly to the anal canal. other means of reduction in sphincter tone include direct injection of the sphincter with botulinum toxin, which temporarily paralyses the sphincter.

Surgery has a major role in the management of patients who have fissures resistant to medical treatment. Lateral sphincterotomy is the most common operation for anal fissure and involves controlled division of the lower half of the internal sphincter at the lateral position.

ANORECTAL ABSCESS

Anorectal abscess is a generic term encompassing the collection of pus and the formation of an abscess in the spaces surrounding the anal canal and rectum.

SURGICAL ANATOMY

Abscesses are named and classified according to the region they invade;

- Intersphincteric abscess results from infection in the anal glands between the internal and external sphincters. Fecal coliforms are typically the offending organisms. As the abscess enlarges within the intersphincteric plane, it can spread in any and several directions.
- Perianal abscess results when pus spreads downwards between the two sphincters. It manifests as a tender swelling of the anal verge.
- Ischiorectal abscess is formed if a growing intersphincteric abscess penetrates the external sphincter below the puborectalis. Infection can spread into the fat of the ischiorectal fossa and the abscess can become quite large.
- Supralevator (high intermuscular or pelvirectal) abscess develops when an intersphincteric abscess expands upwards between the internal and external sphincters.

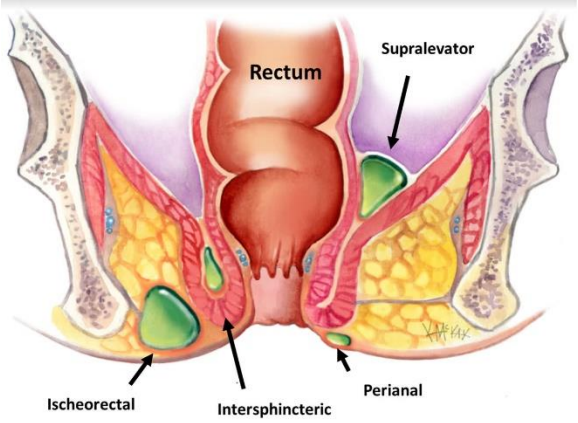


Figure 185 sites of perianal abscess

ETIOLOGY

Most patients who present with anorectal abscess have no predisposing factors and most abscesses are cryptoglandular, initiated by blockage of the anal gland ducts. blockage may follow impaction of vegetable matter or edema from trauma (firm stool or foreign body) or as a result of an adjacent inflammatory process. The obstructed anal gland becomes secondarily infected with large bowel organisms such as Bacteroides, Streptococcus faecalis and coliforms. The fact that the anal glands are situated in the intersphincteric space explains the routes that the infection may take as pus tracks along the line of least resistance through the tissue spaces.

CLINICAL FEATURES

Intersphincteric abscess is common, affecting men three times more frequently than women. In cases where the abscess remains localized within the intersphincteric space, the patient presents with acute anal pain and tenderness. There is usually no evidence of suppuration on inspection of the perianal region. Pain often prevents digital examination, and so an anesthetic is required (examination under anesthesia (EUA)). The main differential diagnosis is acute anal fissure. The diagnosis is confirmed by demonstration of a localized pea-sized lump in the intersphincteric space.

Perianal abscess is the most common type, in which pus tracks inferiorly to appear at the perianal margin between the internal and external sphincters.

Symptoms are usually of 2-3 days duration and the abscess may have discharged spontaneously. Systemic upset is minimal and throbbing anal pain is the predominant presenting complaint. There is tender erythematous swelling appearing on the, anal verge. Main differential diagnoses include pilonidal abscess, hydradenitis suppurativa, folliculitis, periprostatic abscess, Bartholin gland abscess and perianal hematocrit

infection tracking form the anal gland through the external sphincter results in an ischiorectal abscess. Ischiorectal abscess is a relatively uncommon but serious problem; it may be associated with uncontrolled diabetes and so diabetes should be excluded in all cases. As the ischiorectal space is horseshoe-shaped and there are no fascial barriers within it, infection can track posteriorly around the anus to affect the contralateral space (Horseshoe abscesses). In such cases, the patient is toxic and pyrexic with a large, painful, fluctuant, brawny swelling affecting both buttocks, due to large volumes of pus. There is a history of perianal pain for several days, associated with difficulty in sitting.

Infection tracking upwards from the infected anal gland through the upper part of the intersphincteric space may result in a high intersphincteric (high intermuscular) abscess or a pelvirectal abscess. As these spaces encircle the anorectum above the levator muscles, abscesses can be bilateral and often present with a major systemic upset. These are complex problems meriting specialist management.

MANAGEMENT

For most patients, particularly those with perianal abscess, incision and drainage alone are adequate. The primary opening of the anal crypt is rarely identified. Drainage with antibiotics is curative in 50% of cases, however, about 50% treated with this modality develop fistulas (see below). Intersphincteric abscess is treated by using the proctoscope to observe the bulging area and then performing an internal sphincterotomy over the abscess. Supralevator abscesses are drained through the appropriate ischiorectal space or through the rectum. Horseshoe abscesses are drained through the rectum, with corner drains placed in each ischiorectal abscess.

FISTULA IN ANO

A fistula is an abnormal communication between two epithelial/endothelial-lined surfaces. A Fistula in ano has its external opening in the perianal skin and its internal opening in the anal canal.

ETIOLOGY

A fistula in ano forms during the chronic stage of an inflammatory process that begins in the intersphincteric anal glands. As previously discussed, extension of the acute inflammation can result in a Supralevator, ischiorectal or perianal abscess. With chronic inflammation, the abscess communicates with the external surface. Approximately 50% of patients develop fistula in ano after the treatment of anorectal abscess. Figure 9 is a simplified diagram showing the classification of fistula in ano. Generally they are classified as being high or low according to their position in relation to the anorectal ring.

Anal fistulae may be found in association with specific conditions, such as Crohn's disease, tuberculosis, lymphogranuloma venereum, actinomycosis, rectal duplication, foreign body and malignancy.

CLINICAL FEATURES

Pain is rare with fistula in ano, patients more commonly complaining of itching, irritation and discharge. When there is pain, it's usually increases gradually and disappears with discharge. The passage of flatus or feaces through the external opening is suggestive of a rectal rather than an anal internal opening.

Further Investigation requires examination under anesthesia and the fistula tract should be probed by an experienced surgeon. When the fistula opens on the perianal skin of the anterior anus, the tract passes radially, directly to the anal canal. However, when the opening is posterior to a line drawn between the 3 o'clock and 9 o'clock positions, then the tract usually passes circumferentially backwards to the midline and

enters the anal canal at the 6 o'clock position. This is known as and can help define the extent of the course of a fistula (figure 10). An exception is an external opening that is anterior to the imaginary line and more than 3 cm from the anus, in which

case the tract may be curve posteriorly and end in the posterior midline.

INVESTIGATIONS

It is essential to avoid inducing further fistulae by ill- advised probing of the region. It is important to determine. whether the fistula is low or high, as the prognosis and treatment are different for each. Investigation of most fistulae requires formal examination under anaesthesia (EUA), but complex cases merit detailed investigation to define the extent of the tracts using magnetic resonance imaging (MRI). Endo-anal ultrasound also may be useful. A barium follows- through and colonoscopy may be indicated when inflammatory bowel disease is suspected.

MANAGEMENT

Treatment is determined by the course of the fistula tract. Usually, low fistulae can simply be laid open and allowed to heal. Opening achieved through a guiding probe that is inserted into the fistula, once inserted its pulled externally and the resulting protrusion is cut. This is called anal fistulotomy (figure 11).

However, when a significant proportion of the internal and/or external sphincter is involved then laying open, the tract will result in fecal incontinence. In such complex cases, the fistula tract can be probed and a seton passed along its length to allow the fistula to drain. Once it is drained, a tighter seton can be applied that will gradually cut out through the sphincters, allowing them to heal behind the seton. This approach keeps the ends of the sphincters together and so minimizes the chances of inducing incontinence.

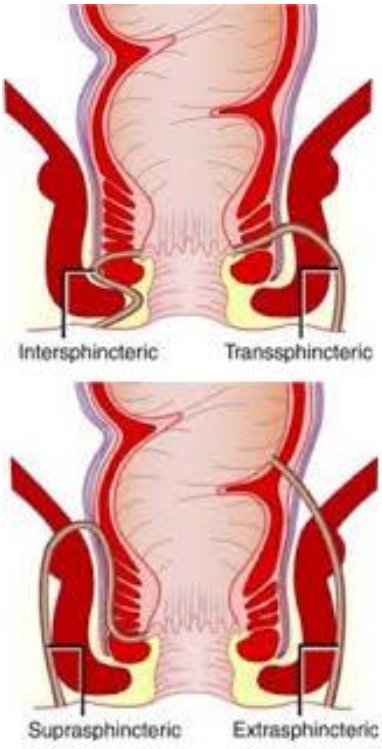


Figure 186 A- Low intersphincteric and transsphinteric B- Ischiorectal and suprasphincteric

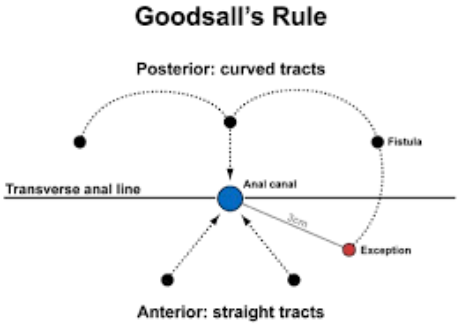


Figure 187 Goodsall's rule

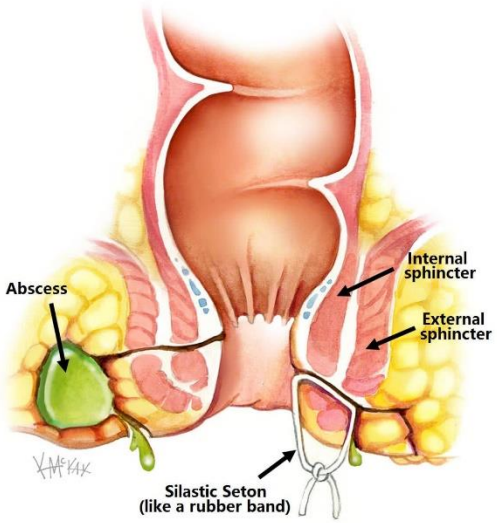


Figure 188 seton

PROCTALGIA FUGAX

This problem is characterised by attacks of severe pain arising in the rectum, recurring at irregular intervals and apparently unrelated to organic disease. The pain is described as camplike, often occurs when the patient is in bed at night, usually lasts only for a few minutes and disappears spontaneously. It may follow straining at stool, sudden explosive bowel action or ejaculation. It seems to occur more commonly in patients suffering from anxiety or undue stress, and it is also said to afflict young doctors. The pain may be unbearable – it is possibly caused by segmental cramp in the pubococcygeus muscle. It is unpleasant and incurable but is fortunately harmless and gradually subsides. If patients have frequent attacks, they may benefit from amitriptyline. Salbutamol inhalers have been suggested as treatment for acute attacks. A more chronic form of the disease has been termed the 'levator syndrome' and can be associated with severe evacuatory dysfunction. Biofeedback techniques have been used to help such patients; in the past, some surgeons tried severing the puborectalis muscle, but this can cause incontinence and should never be carried out. If this is being consider an acceptable alternative is Botox into the puborectalis muscle

MISCELLANEOUS CONDITIONS OF THE ANORECTUM

CONDYLOMATA ACUMINATA (ANAL WARTS)

Condylomata-acuminata is the name given for anal warts. It is caused by infection with human papillomavirus (HPV) which is encountered in after sexual intercourse (especially anal intercourse). It is seen in immunocompromised patients.

CLINICAL FEATURES

Associated warts on the penis and along the female genital tract are common Many are asymptomatic but pruritus, discharge, bleeding and pain are usual presenting complaints. Examination Will reveal single or multiple cauliflower lesions on the anoderm within the distal anal canal.

The diagnosis is aided by aceto-whitening upon application of acetic acid but confirmed by biopsy, which will also indicate the presence or absence of dysplasia.

MANAGEMENT

Lesions on the skin are treated by application of 25% podophyllinal Surgical excision is needed for lesions near the inside of the canal.

ANAL INTRAEPITHELIAL NEOPLASIA

Anal intraepithelial neoplasia (AIN) is virally induced dysplasia of the perianal or intra-anal epidermis.it is associated with anoreceptive intercourse and HIV infection. It is classified according to the degree of dysplasia on biopsy into AIN I, AIN II and AIN III, according to the degree of cytological atypia and the depth of that atypia in the epidermis (figure 1). AIN I is the earliest manifestation of dysplasia, while AIN III is as-sociated with severe dysplasia (Bowen's disease).

CLINICAL FEATURES AND DIAGNOSIS

Patients are often asymptomatic. Thus, diagnosis is made usually incidentally and screening programs are being developed. With high index of suspicion (e.g. the presence of condylomata accuminata), targeted histological mapping with staining can help identify the lesion histologically. Suspicious areas

are raised, scaly, white, erythematous, pigmented or fissured.

MANAGEMENT

AIN II and III should be regularly monitored clinically and, if necessary, by repeat biopsy to exclude invasive disease. Focal disease may be excised. More widespread disease can be dealt with surgically by wide local excision and closure of the resultant defect by flap or skin graft. Anorectal reconstruction and the use of colostomy might be needed. Various topical agents (e.g. retinoids) are being used.

STRICTURES

Strictures of the anal canal are either benign or malignant. They may underlie a wide variety of diseases, but the importance coincides in excluding the malignant ones. Spasmodic strictures may result from an anal fissure which spasm of the internal sphincter. Rarely, a spasmodic stricture accompanies secondary megacolon, possibly as a result of the chronic use of laxatives. Causes of anal strictures are shown in the table below.

Causes of anal stricture

- **Postoperative strictures (e.g. hemorrhoidectomy)**
- **Irradiation strictures**
- **Senile anal stenosis**
- **Inflammatory bowel disease**
- **Endometriosis (of the rectovaginal pouch)**
- **Lymphogranuloma inguinal**
- **Neoplastic (should always be excluded)**

ANAL TUMORS

SQUAMOUS CELL CARCINOMA

Squamos cell carcinoma occurring below the dentate line is the most common cancer of the anal canal. It is predisposed by infection with HPV and AIN III and II are considered a precursor lesion. Patients may present with anal pain, bleeding, mass, ulceration and incontinence if the sphincter is involved. Small tumors are managed by excision. The primary treatment for extensive tumors is chemotherapy and/or radiotherapy. 5-year survival for those diagnosed early is 80%.

MALIGNANT MELANOMA

Malignant melanoma of the anus is very rare and usually presents as a bluish-black soft mass that may mimic a thrombosed external pile, although it may be amelanotic (figure 2). Because the disease is aggressive and distant metastases is common at time of diagnosis, the prognosis, irrespective of treatment, is extremely poor.

BASAL CELL CARCINOMA

Bleeding, itching, and pain are the presenting symptoms of basal cell carcinoma. The superficial, mobile lesions have raised, irregular edges and central ulceration. They are more frequent in men. As with squamous cell carcinoma of the margin, treatment is by wide local excision when possible. Deeply invasive lesions may require abdominoperineal resection. Metastasis is rare.

EPIDERMOID (SQUAMOUS, BASALOID, MUCOEPIDERMOID) CARCINOMA

The name epidermoid tumors is called for tumors arising from the anal canal, above the dentate line, patients may report a history of minor perianal complaints such as bleeding, itching, or discomfort. An indurated anal mass may be present. Disease may be extensive at presentation. Inguinal nodal metastases are found in 20% at diagnosis and another 15% over time.

Abdominal CT and chest radiographs may reveal liver or lung metastases. Endorectal ultrasound can determine the depth of invasion of the primary lesion and may identify pararectal nodes.

Early lesions that are small, mobile, confined to the submucosa, and well differentiated may be treated with local excision. Chemotherapy with radiation has now replaced surgery as first-line therapy for all but the earliest lesions, and surgery is advised only as a salvage procedure for persistent or recurrent disease.

Other tumors that may affect the anal region include lymphoma, basaloid, mucoepidermoid tumors and Kaposi sarcoma.

INGUINOSCRO-TAL CONDITION

INCOMPLETELY DESCENDED TESTIS

Incomplete descent: of the testis occurs when the testis is arrested in some part of its normal path to the scrotum.

An ectopic testis: is a testis that is abnormally placed outside this path

INCIDENCE

- About 4% of boys at born
- The incidence of testicular maldescent at the age of 1 year is around 1%
- About two thirds of these reach the scrotum during the first 3 months of life,
- In 10 % of unilateral cases, there is a family history.
- The condition is more common on **the right** and is **bilateral in 20 %** of cases.

TYPES:

- **intra-abdominal**
- **intra-canalicular**
- **extra-canalicular;** usually at the scrotal neck;
- **ectopic;** the most common site is within the superficial inguinal pouch which lies just inferior and medial to the superficial inguinal ring.

CONSEQUENCES:

1-Infertility

2-Malignancy : The most common cancer is a seminoma

3-Hernia

4-Testicular torsion

CLINICAL FEATURES

- During childhood, the testes are mobile and the cremasteric reflex is active.
- Should be differentiated from retractile testis
- A retractile testis can be gently milked from its position in the inguinal region to the bottom of the scrotum.
- A diagnosis of true incomplete descent should be made only if this is not possible.

When **the testis is impalpable**, ultrasound may be helpful in identifying the intracanalicular testis,

while **laparoscopy** may be needed to differentiate between the **abdominal testis** and a **truly absent testis**.

RETRACTILE TESTIS

■ Retractile testes should be differentiated from true undescended testes

■ This is most easily done with the child relaxed in a warm room

■ Retractile testes are more common than true undescended testes

■ Retractile testes require no treatment

SURGICAL TREATMENT

ORCHIDOPEXY

- Orchidopexy is usually performed before the boy reaches 12 months of age
- The testis and spermatic cord are mobilized and the testis is repositioned in the scrotum.
- Sometimes for a high undescended testis a two-stage surgical procedure is necessary.
- The testis is mobilized as far as possible and anchored with a suture and the mobilization is completed six months later.

INJURIES TO THE TESTIS

- The testis can be damaged either by blunt or by penetrating trauma.
- Injuries can range from simple bruising, intra-testicular haematomas to rupture of the tunica albuginea with very significant collections of blood within the tunica vaginalis (haematocele).
- for significant injuries including **testicular rupture**

1-clinical examination

2-Ultrasound examination

3-surgical exploration is indicated.

The objective is to **repair the tunica albuginea and preserve the testis**,but on occasions **orchidectomy** is necessary

ABSENT TESTIS

'Vanishing' testis describes a condition in which a testis develops but disappears before birth.

The most likely cause for this is prenatal torsion.

TORSION OF THE TESTIS

PATHOPHYSIOLOGY

Testicular torsion is a condition whereby the testicle twists in such a way that its blood supply becomes compromised.

If left untreated,lead to ischemia and the testicle dies.

The earlier the surgery > the better the results, a testicular salvage rate of 100 % if the testicle can be untwisted within 6 hours

20 % salvage rate if the surgery is delayed for 24 hours.

For torsion to occur, one of several abnormalities must be present:

1- **Inversion of the testis** is the most common predisposing cause.

2- High **investment of the tunica vaginalis** causes the testis to

hang within the tunica like a clapper in a bell.

3- **Separation of the epididymis** from the body of the testis

CLINICAL FEATURES

most common between 10 and 25 years of age.

Typically, there is sudden pain in the groin and the lower abdomen.

The patient feels nauseated and may vomit.

The testis seems high and the tender twisted cord can be palpated above it.

The cremasteric reflex is lost

-ve Prehn sign (relief of pain with elevation of the testicle).

+ve in epididymoorchitis

- lumps in the scrotal sac
  blood in the semen.
  Scrotal erythema.

Problems to be considered in the differential diagnosis of testicular torsion include the following:

- Torsion of testicular or epididymal appendage
  Epididymitis, orchitis, epididymoorchitis
- Hydrocele
- Testis tumor
- Idiopathic scrotal edema (occurs between the age of 4 and 12 years)
- The scrotum is very swollen but there is little pain or tenderness
- The swelling is usually bilateral
- It is thought to be an allergic phenomenon; occasionally there is eosinophilia.

The swelling subsides after a day
* Idiopathic testicular infarction
* Traumatic rupture
* Traumatic hematoma

MANAGEMENT

The management of the case should be determined primarily **on clinical grounds.**

If there is any doubt as to the diagnosis, then urgent scrotal exploration is indicated.

While **Doppler ultrasound scanning** can confirm the absence of the blood supply to

the affected testis, false positive results can be seen, so it **is not routinely recommended**

Exploration for torsion should be performed. **If the testis is viable** when the cord is untwisted it should be prevented from twisting again by **fixation** with nonabsorbable sutures between the tunica vaginalis and the tunica albuginea.

The other testis should also be fixed because the anatomical predisposition is likely to be **bilateral.**

An infarcted testis should be removed.

VARICOCELE

A varicocele is a varicose dilatation of the veins draining the testis.

SURGICAL ANATOMY

The veins draining the testis and the epididymis form the pampiniform plexus.

The left testicular vein empties into the **left renal vein**,

the right into the inferior vena cava

The testicular veins usually have **valves** near their terminations, but these are sometimes **absent.**

There is an alternative (collateral) venous return from the testes through the **cremasteric veins**, which drain mainly into the **inferior epigastrics**.

AETIOLOGY

Varicoceles are common, affecting perhaps 15–20 % of males.

90 % are left-sided, reflecting the proximal venous anatomy.

In some cases, the dilated vessels are cremasteric veins

 # The usual cause is absence or incompetence of valves in the proximal testicular vein.
 # most varicoceles are idiopathic,

obstruction of the left testicular vein **by a renal tumour or nephrectomy** is a cause of varicocele in later life; characteristically, in such cases the **varicocele does not decompress in the supine position.**

CLINICAL FEATURES

most varicoceles are **asymptomatic**,

those that are symptomatic tend to present an annoying dragging discomfort that is worse on standing at the end of the day.

When examined in the erect position, the scrotum on the affected side **hangs lower than normal**

on palpation the varicose plexus feels like a **bag of worms**.

There may be a **cough impulse**.

If the patient lies down the veins empty by gravity and is normal to palpation.

Ultrasonography can be helpful in the diagnosis of small varicoceles and in older men with an apparently recent onset of varicocele,

ultrasonography of the kidneys is important in excluding a left renal tumour.

VARICOCELE AND SPERMATOGENESIS

varicocele will tend to 'warm' the testis, which is usually around 2.5°C below rectal temperature, and there is conflicting evidence regarding the effect of this temperature difference upon spermatogenesis.

Unfortunately, there is little evidence that varicocelectomy improves semen quality or the rate of conception.

TREATMENT

Operation is not indicated for an asymptomatic varicocele.

When the discomfort is significant, **then embolization** of the gonadal veins is the usual **first line intervention**.
If this is **not possible**, or if the **varicocele recurs** (in 20 % after embolization), then **surgical ligation of the testicular veins** is the appropriate treatment, although recurrence can occur even after such surgery.

HYDROCELE

A hydrocele is an abnormal collection of serous fluid in a part of the processus vaginalis, usually the tunica vaginalis.

Acquired hydroceles are primary or idiopathic, or secondary to epididymal

or testicular disease.

AETIOLOGY

1 By **excessive production of fluid** within the sac, e.g. a secondary hydrocele (most frequently associated with **acute or chronic epididymo-orchitis**. torsion of the testis and with some testicular tumours)

2 By **defective absorption of fluid**; this appears to be the explanation for most primary hydroceles

3 By interference with **lymphatic drainage** of scrotal structures

4 By connection with the peritoneal cavity via a **patent processus vaginalis** (congenital).

 # Hydrocele fluid contains albumin and fibrinogen.

CLINICAL FEATURES

Hydroceles are typically
1-translucent

2- it is possible to 'get above the swelling' on examination of the scrotum

3-painless

4- it may reach a prodigious size before the patient presents for treatment

The condition is particularly common in hot countries.

ultrasound scan is necessary to visualise the testis if the hydrocele sac is tense.

Be wary of an acute hydrocele in a young man since there may be **a testicular tumour.**

the hydrocele may be **intermittent** :

Because of the hydrocele fluid may drain into the peritoneal cavity when the child is lying down

Ascites should be considered if the swellings are bilateral.

Encysted hydrocele of the cord is a smooth oval swelling near the spermatic cord, which is liable to be mistaken for an inguinal hernia.

Hydrocele of the canal of Nuck is a similar condition in females.

The cyst lies in relation to the round ligament and is always at least partially within the inguinal canal.

TREATMENT

Congenital hydroceles are treated by herniotomy if they do not resolve spontaneously

Small acquired hydroceles do not need treatment.

If they are sizeable and bothersome for the patient, then surgical treatment is indicated.

Aspiration of the hydrocele fluid is simple, but the fluid always reaccumulates within a week or so. It may be suitable for men who are unfit for scrotal surgery

Testicular malignancy is an uncommon cause of hydrocele that can be excluded by ultrasound examination

Filarial hydroceles and chyloceles

Filarial hydroceles and chyloceles account for up to **80 per cent** of hydroceles in **tropical countries** where the parasite **Wucheria bancrofti** is endemic.

HAEMATOCELE

The most common cause of a haematocele is vessel damage during needle drainage of a hydrocele. Prompt refilling of the sac associated with pain, tenderness and reduced transillumination will confirm the diagnosis.

other cause testicular trauma

Cysts associated with the epididymis

■ Lie posterior to and separate from the testis and they transilluminate

■ Diagnosis can be confirmed by ultrasound examination

■ Can be treated conservatively unless large or uncomfortable

ACUTE EPIDIDYMO-ORCHITIS

■ In young men usually arises secondary to a sexually transmitted genital infection

■ In older men usually arises secondary to urinary infection

■ May be a complication of catheterisation or instrumentation of the urinary tract

■ May need aggressive treatment with parenteral antibiotics (doxycycline or a quinolone)

All patients should drink plenty of fluid.
Local measures including scrotal support and analgesia are helpful.
Antibiotic treatment should continue for at least 2 weeks or until the inflammation has subsided. If suppuration occurs, drainage is necessary.

RETROPERITONEUM

SURGICAL ANATOMY

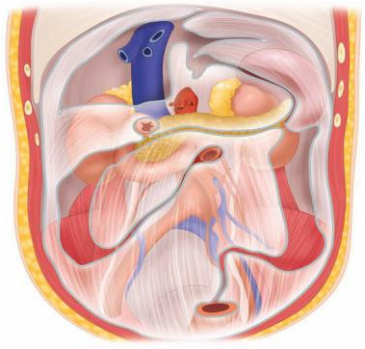


Figure 189

The retroperitoneum is defined as the space between the posterior envelopment of the peritoneum and the posterior body wall (Fig.1).

The retroperitoneal space is bounded superiorly by the diaphragm, posteriorly by the spinal column and iliopsoas muscles, and inferiorly by the levator ani muscles.

Although technically bounded anteriorly by the posterior reflection of the peritoneum, the anterior border of the retroperitoneum is quite convoluted, extending into the spaces in between the mesenteries of the small and large intestine. Because of the rigidity of the superior, posterior, and inferior boundaries, and the compliance of the anterior margin, retroperitoneal tumors tend to expand anteriorly toward the peritoneal cavity

RETROPERITONEAL INFECTIONS

The posterior reflection of the peritoneum limits the spread of most intra-abdominal infections into the peritoneum.

Accordingly, the source of retroperitoneal infections is usually an organ contained within or abutting the retroperitoneum. Retrocecal appendicitis, contained perforation of duodenal ulcers, iatrogenic perforation with esophagogastroduodenoscopy or endoscopic retrograde cholangiopancreatography, and complicated pancreatitis may all lead to retroperitoneal infection with or without abscess formation

RETROPERITONEAL (PSOAS) ABSCESS

Psoas abscess is a relatively uncommon diagnosis, psoas abscess was mainly caused by TB of the spine (Pott's disease). With the decline of *M. tuberculosis* as a major pathogen in resource-rich countries, a psoas abscess was mostly found secondary to direct spread of infection from the inflamed ± perforated digestive or urinary tract. In recent years a primary psoas abscess due to haematogenous spread from an occult source is more common,

especially in immunocompromised and older patients, as well as in association with intravenous drug misuse.

Clinical presentation is with back pain, lassitude and fever. A swelling may point to the groin as it tracks along iliopsoas. Pain may be elicited by passive extension of the hip or a fixed flexion of the hip evident on inspection.

Radiological investigation is via CT scanning **(Figure 2)** and treatment usually by percutaneous CT-guided drainage and appropriate antibiotic therapy

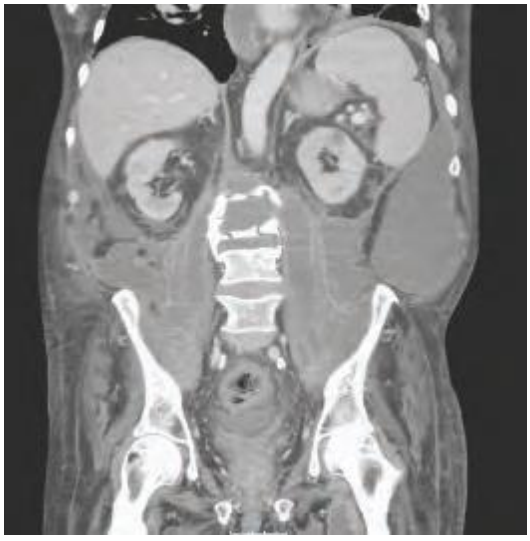


Figure 190 Representative sagittal computed tomography reconstruction

RETROPERITONEAL FIBROSIS

Retroperitoneal fibrosis is a class of disorders characterized by hyperproliferation of fibrous tissue, in

the retroperitoneum relatively rare diagnosis characterised by the development of a flat grey/white plaque of tissue, which is found first in the low lumbar region but then spreads laterally and Histological appearances vary from active inflammation with a high cellular content interspersed with bundles of collagen through to one of acellularity and mature fibrosis/calcification.

Its aetiology is obscure in most cases (idiopathic) being allied to other fibromatoses (others being Dupuytren's contracture and Peyronie's disease). In other patients the cause is known.

The clinical presentation may be one of ill-defined chronic backache or occur as a result of compromise to involved structures, e.g. lower limb or scrotal oedema secondary to venous occlusion, or chronic renal failure secondary to ureteric obstruction. Treatment will be directed to the cause, the modification of disease activity when appropriate, e.g. immunomodulation with steroids, tamoxifen and restoration upwards to encase the common iliac vessels, ureters and aorta. of flow in affected structures, e.g. ureteric stenting

CAUSES OF RETROPERITONEAL FIBROSIS

BENIGN

- Idiopathic (Ormond's disease)
- Chronic inflammation
- Extravasation of urine
- Retroperitoneal irritation by leakage of blood or intestinalcontent
- Aortic aneurysm (inflammatory type)
- Trauma
- Drugs (chemotherapeutic agents and previouslymethysergide)

MALIGNANT

- Lymphoma
- Carcinoid tumours
- Secondary deposits (especially from carcinoma of stomach ,colon,breast and prostate

RETROPERITONEAL TUMOURS

Although swellings in the retroperitoneum may include abscess, haematoma, cysts and spread of malignancy from retroperitoneal organs (kidney, ureter, adrenal), the term retroperitoneal tumour is usually confined to primary tumours arising in other tissues in this region e.g. muscles, fat, lymph nodes and nerves. The management of such tumours is now frequently by referral to a specialist centre and this should be done before biopsy which may compromise subsequent surgical cure. The two commonest are briefly described.

RETROPERITONEAL LIPOMA

The patient may seek advice on account of a swelling or because of indefinite abdominal pain. Women are more often affected. These swellings sometimes reach an immense size.

Diagnosis is usually by ultrasonography and CT scanning. A retroperitoneal lipoma sometimes undergoes myxomatous degeneration, a complication that does not occur in a lipoma in any other part of the body. Moreover, a retroperitoneal lipoma is often malignant (liposarcoma) (see below) and may increase rapidly in size

RETROPERITONEAL SARCOMA

Retroperitoneal sarcomas are rare tumours accounting for only 1–2% of all solid malignancies (10–20% of all sarcomas are retroperitoneal). The peak incidence is in the fifth decade of life, although they can occur at almost any age.

The most common types of retroperitoneal soft-tissue sarcomas in adults vary from study to study. However, in most studies, the most frequently encountered cell types are:

- liposarcoma;
- leiomyosarcoma;
- malignant fibrous histiocytoma(MFH)

CLINICAL FEATURES

Patients with sarcomas present late, because these tumours arise in the large potential spaces of the retroperitoneum and can grow very large without producing symptoms.

Moreover, when symptoms do occur, they are nonspecific, such as abdominal pain and fullness, and

are easily dismissed as being caused by other less serious processes. Retroperitoneal sarcomas are, therefore, usually very large at the time of presentation

INVESTIGATION

Detailed multiplanar imaging (CT + MRI) with reconstructions is required not only for tumour detection, staging and surgical planning, but also for guiding percutaneous or surgical biopsy of these tumours. Such biopsies have a greater role than for other sarcoma

TREATMENT

The definitive treatment of primary retroperitoneal sarcomas is surgical resection. Chemotherapy and radiotherapy without surgical debulking have rarely been beneficial, when used alone or in combination A multidisciplinary treatment approach with imaging review will be required when assessing operability (based on adjacency or involvement of vital structures) and approach. Up to 75% of retroperitoneal sarcoma resections involve resection of at least one adjoining intraabdominal visceral organ (commonly large or small bowel or kidney).

The most common types of vascular involvement precluding resection are involvement of the proximal superior mesenteric vessels or involvement of bilateral renal vessels.

PROGNOSIS

In the vast majority of sarcomas, cell type has no impact on treatment and long-term survival. Survival rates are in general poor, even after complete resection, being in the order of 35–50% (excluding low-grade liposarcomas, which may frequently be cured by resection

PRINCIPLES IN PEDIATRIC SURGERY

INTRODUCTION

Children are not small adults. They suffer from different disorders and their physical and psychological responses are different. Their capacity for adaptation is greater but they must endure any consequences of disease and its management for longer. In contrast to adults they rarely have comorbidity from degenerative diseases or lifestyle problems but they can suffer the unique consequences of congenital malformations. Children must be treated within the context of their families. This lecture focuses on aspects of pediatric surgery relevant to general surgery.

Common terms used in pediatric patients assessment are shown in the table below.

Common terms	
Preterm	<*37* completed weeks of gestation
Full term	Between *37* and 42 completed weeks of gestation
Neonate	Newborn baby up to 28 days of age
Infant	Up to 1 year of age
Child	Ail ages up to 16 years but often divided into
	preschool child (usually < 5 years), child and adolescent (puberty up to 16 years)

PROGNOSIS

Pediatric patients may present by either obvious congenital anomalies, or symptoms and signs of an underlying disease, however, some patients may remain with no presentation until late in adult life and so an estimate of the birth rate may give the surgeon a clue about what screening test to be taken. Antenatal diagnosis using techniques as amniocentesis, ultrasound, blood chemical analysis, and genetic or chromosomal analysis may detect chromosomal abnormalities, genetic abnormalities or structural malformations. This is important as it

may assist in early detection and thus warning the patient Preparing for surgery

mother, or if the condition is sever, to be avoided with termination of pregnancy. It also may help the surgeon to save the newborn baby life by planned deliver. Early detection may also direct the surgeon to transfer the baby.

Thermoregulation is important in children undergoing surgery. The body surface area to weight ratio decreases with age and small children Therefore lose heat more rapidly. Babies have less subcutaneous fat and immature peripheral vasomotor control mechanisms. The operating theatre must be warm and the infant's head (which may account for up to 20% of the body surface area compared ' with 9% in an adult) should be insulated. Infusions and respiratory gases may need to be warmed. The central temperature should be monitored and a warm air blanket is advisable during lengthy operations.

Infants undergoing surgery are vulnerable in other ways, Impaired gluconeogenesis renders them more susceptible to hypoglycaemia; blood glucose must be monitored and maintained above 2.5 mmol |-1. Newborns are at risk of clotting deficiencies and should be given intramuscular. vitamin K before major surgery. They are less able to concentrate urine or conserve sodium and have a greater obligatory Water loss to excrete a given solute load. Fluid and sodium requirements are relatively high. Infants are prone to gastro-oesophageal reflux and have less well-developed protective reflexes, rendering them more at risk of pulmonary aspiration; adequate nasogastric aspiration is essential in those with gastrointestinal, obstruction. Immaturity of the immune system increases the risk of infection; which can present with non-specific features such as poor feeding, vomiting and listlessness.

into more sophisticated pediatric departments before the baby is born (also known as in-utero transfer). The availability of effective surgical skills, equipment’s, arid nursing services along with early detection and early transport, may determine the prognosis of any congenital anomaly.

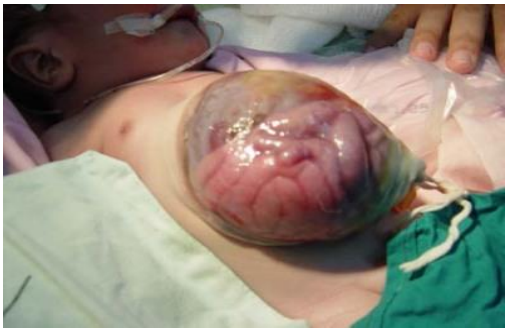
COMMON 'OBVIOUS' DEVELOPMENTAL ANOMALIES

EXOMPHALOS (OMPHALOCELE)

During embryonic life, the structures of the mid gut develop in the extracoelomic cavity through the umbilical cord, this, is called physiological herniation. At the 11th week of gestation these abdominal structures will reduce to the intraembryonic cavity in the counterclockwise direction. FaiIure of these organs to return causes herniation through the umbilical ring. This condition is distinguished from Gastroschisis by the presence of a membrane around the herniated viscera. lf the diameter of the sac exceeds 5 cm, its termed major exomphalos which usually involves herniation of the stomach, duodenum and parts of the liver. Otherwise its termed minor exomphalos, exomphalos occurs in 2.5\10,000 births and sometimes associated with cardiac and neural tube abnormalities.

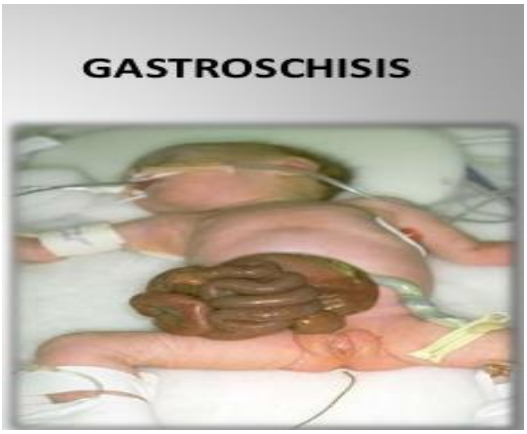
To surgeons it's considered as a spot Diagnosis but is often detected through alpha-FP screening or a detailed fetal ultrasound. Genetic counseling and genetic testing such as amniocentesis is usually offered during the pregnancy.

Reduction of the herniated mass may introduce a direct pressure to what is already a narrow abdominal cavity, thus it may cause compression on the diaphragm, causing respiratory distress. This method can be used only if the infant is to be put on a ventilator. However, it still may compress the inferior vena cava and thus can cause cardiovascular insufficiency, especially in those with cardiac abnormalities, and so methods like using to strengthen the wall of the sac are used. Until the baby grows old enough to manage the condition as a ventral hernia. Myocutaneos mobilization involves the covering the herniated sac with skin and muscle tissue, and thus, this may result in an increase in the size of the abdominal cavity.



GASTROSCHISIS

Gastroschisis, (also called paraomphalocele) represents a congenita! defect characterized by a defect in the anterior abdominal wall through which the intestinal contents freely protrude. There is no overlying sac and the size of the defect is usually. less than 4 cm The abdominal wall defect is located at the junction of the umbillicus and normal skin, and is a؛most always to the right of the umbilicus. During the late embryonic life, the herniated viscera are located in the amniotic cavity, thus the viscera are irrigated with amniotic fluid and therefore, Are edematous, and so this may also require ventilation if reduction is to ba done. A silastic sheath can be used to cover the herniated viscera until reduction is operable without the use of assisted ventilation. Myocutaneos mobilization can also be useful in this condition.



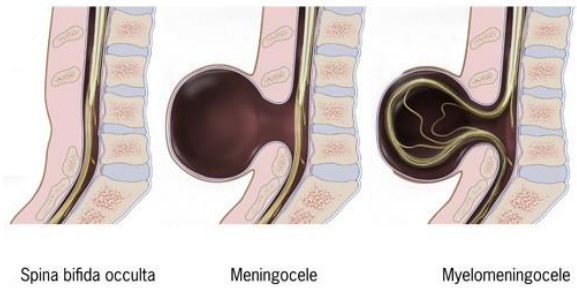
MENINGOCELE AND MYELOMENINGOCELE

MENINGOCELE

Meningocele mean's herniation of the meninges covering the sacroiliac part the spinal cord (most commonly the cuada equine) through a defect formed due to incomplete closure of the posterior wall of the vertebral canal, and covering fascias. Herniation of parts of the spinal cord is called myelomeningocele. The defect is called spina bifida and is either open also known as spina bifida aperta (i.e. apparent), or closed which is also known as spina bifida occulta (i.e. hidden). The difference is that the former has complete agenesis of the wall while the later still has some covering fascias.

MYELOMENINGOCELE

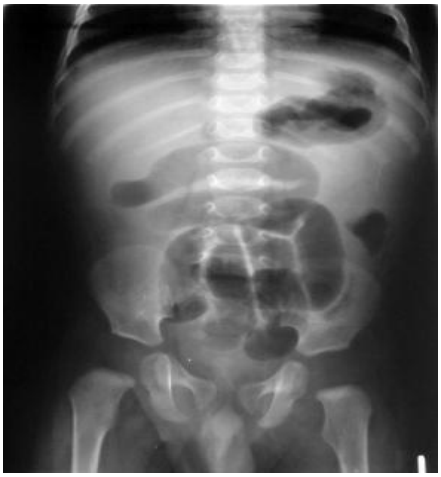
is most commonly seen in spina bifida aperta and is diagnosed from birth as these children have an ap- parent spinal defect. These children may also have numbness and weakness in the parts of the body below the level of the lesion. They also may have a neuropathic bladder ad paralysis of the anal sphincter. Children with spina bifida occulta may present in life when the spinal cord tethers through the defect, however, it can be diagnosed from birth owing to the characteristic overlying skin lesions (lipomas, hairy patches, dimples and sinuses).



INTESTINAL OBSTRUCTION

Infants with intestinal obstruction commonly. presents with bile stained vomiting, abdominal distention and failure to pass meconium. Meconium is normally passed within the first 24 hours in 80% of babies and delay beyond this is a cause for concern. other presentations are shown in the table below.

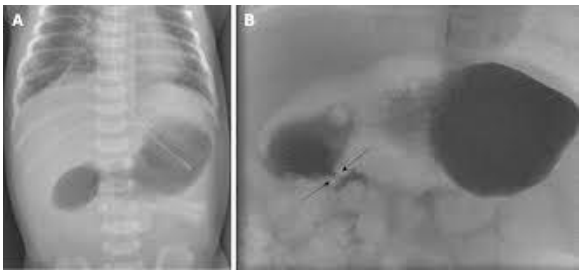
Causes of intestinal obstruction in children
Mechanical
Intrinsic • Atresia • stenosis
Intraluminal • Meconium ilues
Extrinsic • Malrotation • Diaphragmatic hernia • volvulus
Neurogenic • Agnagliosis • Hirschsprung's disease
Functional • Necrotizing enterocolitis • Sepsis • Prematurity • Drugs in labour (morphine) • peritonitis



DUODENAL OBSTRUCTION

Duodenal obstruction can be either extrinsic or intrinsic. Extrinsic duodenal obstruction may be due to compression by an annular pancreas or a subhepatic cecum in volvulus neonatorum. Intrinsic can be due to stenosis or prenatal ultrasound scan findings of a 'double bubble' in the fetal abdomen together with maternal polyhydramnios, There is an association with Down's syndrome (30%). Postnatally, the infant develops bilious vomiting if the atresia is distal to the ampulla (most common form 85%). A plain abdominal radiograph is usually diagnostic Repair is by duodenoduodenostomy. Occasionally, there is a duodenal membrane with a small central perforation (duodenal stenosis) which may delay the onset of obstructive symptoms until later childhood

atresia. Duodena! atresia (atresia = no lumen) may take the form of a completely obstructing membrane or the proximal and distal duodenum may be completely separated.

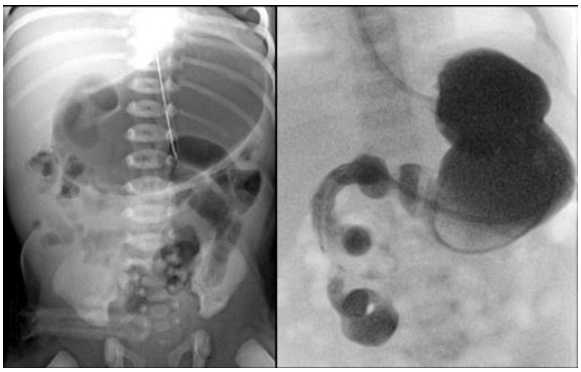


VOLVULUS NEONATORUM

By the 12th week of gestation, the mid-gut has returned to the fetal abdomen from the extra-

embryonic coelom and has begun rotating counter clockwise around the Superior mesenteric artery axis. In classical intestinal malrotation, this process fails; the duodenojejunal flexure lies to the right of the midline and the caecum is central, creating a narrow base for the small bowel mesentery,

which predisposes to mid-gut volvulus Malrotation with volvulus is life-threatening and typically presents with bilious vomiting. Bile-stained vomiting in the infant is a sign of intestinal obstruction until proved otherwise. As the gut strangulates, the baby may pass bloodstained stools and becomes progressively sicker. An upper gastrointestinal contrast study confirms the malrotation. Barium meal may show absence of the C-shaped duodenum. Resuscitation and urgent surgery are needed to untwist the volvulus, widen the base of the small bowel mesentery, straighten the duodenum and position the bowel in a non-rotated position (Ladd's. procedure) the appendix within the abdomen.is usually removed to avoid leaving it in an abnormal site



BOWEL ATRESIA

The anatomy of jejunal ileal atresia varies from an obstructing membrane through, to widely-separated blind- ended bowel ends associated with a mesenteric defect. Atresia's may be single (90%) or multiple (10%), most commonly in the jejunum but rare in the colon, and are probably secondary to a prenatal vascular or mechanical insult causing sterile infarction of a segment of gut and are associated with meconium ileus, cloacal extrophy and abdominal wall defects. They present with intestinal obstruction soon after birth and diagnosed by abdominal films which may show multiple fluid levels down to the site of obstruction. The proximal bowel is often extremely dilated and needs to be tapered prior to anastomosis to the distal bowel.

MECONIUM ILEUS

Infants with cystic fibrosis have characteristic pancreatic enzyme deficiencies and abnormal chloride secretion in the intestine that result in the production of viscous, water-poor meconium. Meconium ileus occurs when this thick, highly viscous meconium becomes impacted in the ileum and leads to high-grade intestinal obstruction. Meconium ileus most commonly affects the ileum, and can be either uncomplicated, in which case there is no intestinal perforation, or complicated, in which case prenatal perforation of the intestine has occurred, causing meconium peritonitis or vascular compromise of the distended ileum develops, causing atresia and volvulus and predisposing to perforation portion of the ileum under fluoroscopic control. Since these contrast agents act partially by absorbing fluid from the bowel wall into the intestinal lumen, maintaining adequate hydration of the infant during this manoeuvre is extremely important. The enema may be repeated at 12- hour intervals over several days until all the meconium is evacuated. Failure to reflux the contrast into the dilated portion of the ileum signifies the presence of an associated atresia or complicated meconium ileus, and thus warrants exploratory laparotomy. if surgical intervention is required because of failure of contrast enemas to relieve obstruction, resection of the distended terminal ileum is performed and the meconium pellets are flushed from the distal small bowel. At this point, ileostomy and mucous fistula may be created from the proximal and distal ends, respectively

CLINICAL FEATURES

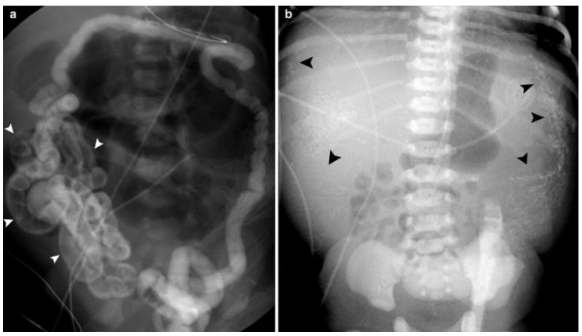
These infants present shortly after birth with progressive abdominal distention and failure to pass meconium with intermittent bilious vomiting and palpable abdominal masses.

INVESTIGATION

Abdominal radiographs show dilated loops of intestine. Because the enteric contents are so viscous, air-fluid levels do not form, even when obstruction is complete. Small bubbles of gas become entrapped in the inspissated meconium in the distal ileum, where they produce a characteristic "soap bubble" appearance on radiograph.

The diagnosis of meconium ileus is confirmed by a contrast enema, which typically demonstrates a

microcolon. In patients with uncomplicated meconium ileus, the terminal ileum is filled with pellets of meconium. in patients with complicated meconium ileus, intraperitoneal calcifications form, producing an eggshell pattern on plain abdominal x-ray and sometimes showing air under the diaphragm.



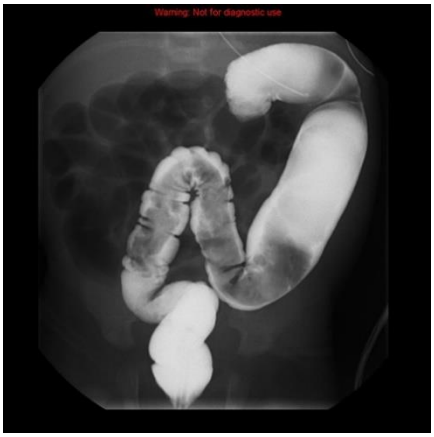
MANAGEMENT

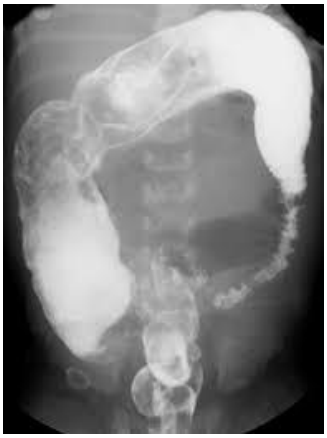
The treatment strategy depends on whether the patient has complicated or uncomplicated meconium ileus. Patients with uncomplicated meconium ileus can be treated non operatively. Dilute water-soluble contrast (gastrografine) is advanced through the colon into the dilated.

HIRSCHSPRUNG'S DISEASE

Hirschsprung's disease is characterised by the congenital absence of intramural ganglion cells (aganglionosis) and the presence of hypertrophic nerves in the distal-large bowel (figure). The absence of ganglion cells is due to a failure of migration of vagal neural crest cells into the developing gut. The affected gut is tonically contracted causing functional bowel obstruction. The aganglionosis is

restricted to the rectum and sigmoid colon in 75% of patients (short segment), involves the proximal colon in 15% (long segment) and affects the entire colon and a portion of terminal ileum in 10% (total colonic aganglionosis). A transition zone exists between the dilated, proximal, normally innervated bowel and the narrow, distal aganglionic segment.





CLINICAL PRESENTATION

Hirschsprung's disease typically presents in the neonatal period with delayed passage of meconium, abdominal distension and bilious vomiting (complete functional obstruction) but it may not be diagnosed until later in childhood on even adult life, when it manifests as severe chronic, constipation (partial functional obstruction).

INVESTIGATIONS

Definitive diagnosis of Hirschsprung's disease depends on histological examination (acetylcholine stains) of an adequate rectal biopsy by an experienced pathologist. A contrast enema may show the extent of the aganglionic segment.

MANAGEMENT

Surgery aims to remove the aganglionic segment and bring down healthy ganglionic bowel to the anus; these 'pull-through' operations (e.g. Swenson, Duhamel, Soave and transanal procedures) can be done in a single stage or in several stages after first establishing a proximal stoma in normally

innervated bowel. Most patients achieve good bowel control but a significant minority experience residual constipation and/or faecal incontinence.

NECROTIZING ENTEROCOLITIS (NEC)

lethal gastrointestinal disorder affecting the intestine of enteral feeding, bacterial infection, intestinal ischemia.

Necrotizing enterocolitis (NEC) is the most common and the stressed, preterm neonate. Multiple risk factors have been associated with the development of NEC. These include prematurity, low birth weight (<1.5 Kg), initiation of resulting from birth asphyxia, persistence of a patent ductus arteriosus, cyanotic, heart disease, and maternal cocaine abuse. NEC may involve single or multiple segments of the intestine, most commonly the terminal ileum followed

by the colon but it can be multiple and sometimes may even affect the whole intestines. The gross findings in NEC include bowel distention with patchy areas of necrosis, haemorrhage, mucosal ulceration, thinning, pneumatosis (subserosal gas collection), gangrene, or frank perforation. The microscopic features include the appearance of a "bland infarct" characterized by full- thickness necrosis (pan-necrosis).

PATHOGENESIS

The exact mechanisms that lead to the development of NEC remain incompletely understood. However, current thinking suggests that in the setting of an episode of perinatal stress, such as respiratory distress syndrome, the premature infant suffers a period of intestinal hypoperfusion. This is followed by a period of reperfusion, and the combination of ischemia and reperfusion lead to mucosal injury. The damaged intestinal mucosa can then be readily breached by indigenous microorganisms that translocate across it. The translocated bacteria then initiate an inflammatory cascade that involves the release of various pro-inflammatory mediators, which in turn may be responsible for further epithelial injury and the systemic manifestations of NEC.

CLINICAL FEATURES

In the earliest stage of the disease, infants present with formula intolerance. This is manifested by vomiting or by finding a large residual volume from a previous feeding in the stomach at the time of the next feeding. Following appropriate treatment, which consists of bowel rest and intravenous antibiotics, many of these infants will not progress to more advanced stages of NEC. Infants with late stages of the disease have established NEC that is not immediately life threatening. Clinical findings include abdominal distention and tenderness bilious nasogastric aspirate, and bloody, stools, which indicate the development of intestinal ileus and mucosal ischemia. Abdominal examination may reveal a palpable mass indicating the presence of an inflamed loop of bowel, diffuse abdominal tenderness, cellulitis, and edema of the anterior abdominal wall. The infant may appear systemically ill, with decreased urine output, hypotension, tachycardia, and non- cardiac pulmonary edema.

INVESTIGATIONS

Hematologic evaluation reveals either leukocytosis or leukopenia, an increase in the number of bands, and thrombocytopenia. An increase in the blood urea nitrogen and plasma creatinine levels with metabolic acidosis on blood gas analysis may be found, which signify the development of renal dysfunction.

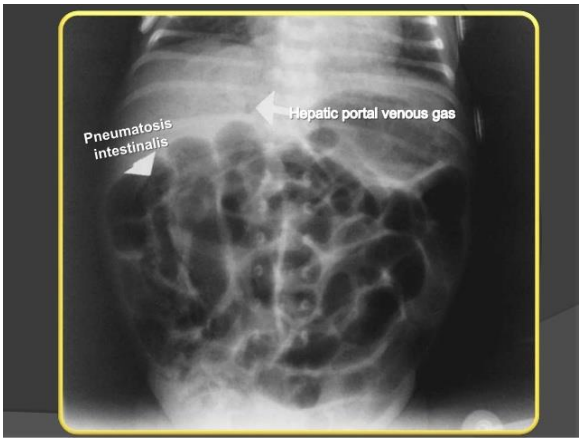
DIAGNOSIS

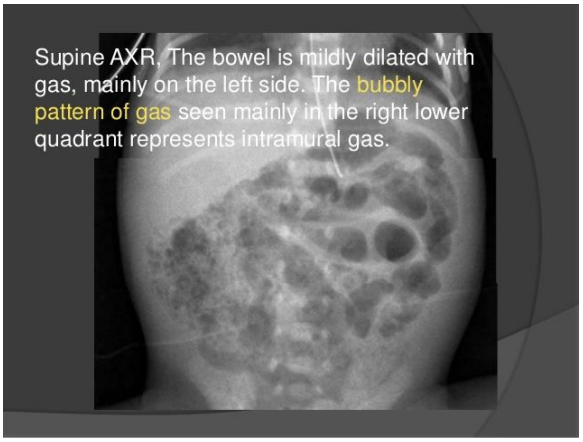
The diagnosis of NEC may be confirmed by abdominal radiography. The pathognomonic radiographic finding in NEC is pneumatosis intestinalis (figure 10), which represents invasion of the ischemic mucosa by gas-producing microbes. Other findings include the presence of ileus, portal venous gas, pneumoperitoneum, or ascites. Earlier stages of the disease may normally heal upon management with antibiotics and I.V fluids, either with no complications (by re-epithelization) or with complications causing scars and strictures (also known as NEC scar).

TREATMENT

In all infants suspected of having NEC; feedings are discontinued, a nasogastric tube is placed, and broad- spectrum parenteral antibiotics are given. The infant is resuscitated, and inotropes are administered to maintain perfusion as needed.

Intubation and mechanical ventilation may be required to maintain oxygenation. Total parenteral nutrition is started. Subsequent treatment may be influenced by the particular stage of NEC that is present. The presence of air in the abdomen along with clinical deterioration, erythema of the abdominal wall, presence of a fixed loop sign, abdominal masses and progressive acidosis are all indications for surgical intervention.





ANORECTAL MALFORMATIONS

The anus is either imperforate or abnormally sited. Associated malformations of the sacrum arid genitourinary tract are common. In boys, a perineal fistula (a 'low' defect) or an imperforate anus with a rectourethral fistula ('high' defect) is most commonly seep. In girls, an anterior anus (low defect) or an imperforate anus with a fistula opening in the posterior vestibule (not vagina) is most common (low defect). Cloacal malformations, in which the rectum and genitourinary tract share a common outflow channel, are also seen in girls (recto-vaginal fistula\ high defect). These children mainly present with symptoms of intestinal obstruction, however, they may present with fistulas without any symptom

	Male	Female
High	• Anorectal agenesis • With recto-prostatic urethral fistula • Without fistula • Rectal atresia	• Anorectal agenesis • With recto-vaginal fistula • Without fistula • Rectal atresia
Intermediate	• Rectobulbar-urethral fistula • Anal agenesis without fistula	• Rectovestibular fistula • Rectovaginal fistula • Anal agenesis without fistula
Low	• Anocutaneous fistula • Anal stenosis	• Anovestibular fistula • Anocutaneous fistula • Anal stenosis
Rare Malformations		• Cloaca

INVESTIGATIONS

Diagnosis of a low malformation is usually possible by inspection of the infant's perineum alone. A lateral prone radiograph at about 24 hours of age can help by showing the distance between the rectal gas bubble and the anal skin Other means of diagnosis include MRI and micturating cysto-urethrogram.

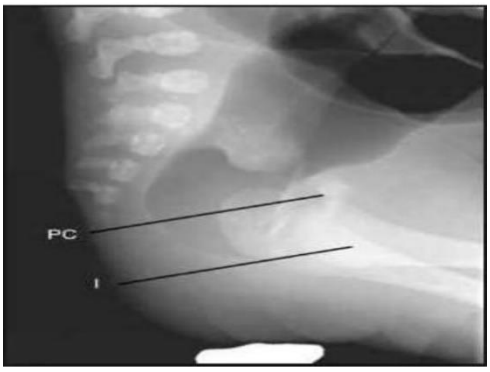
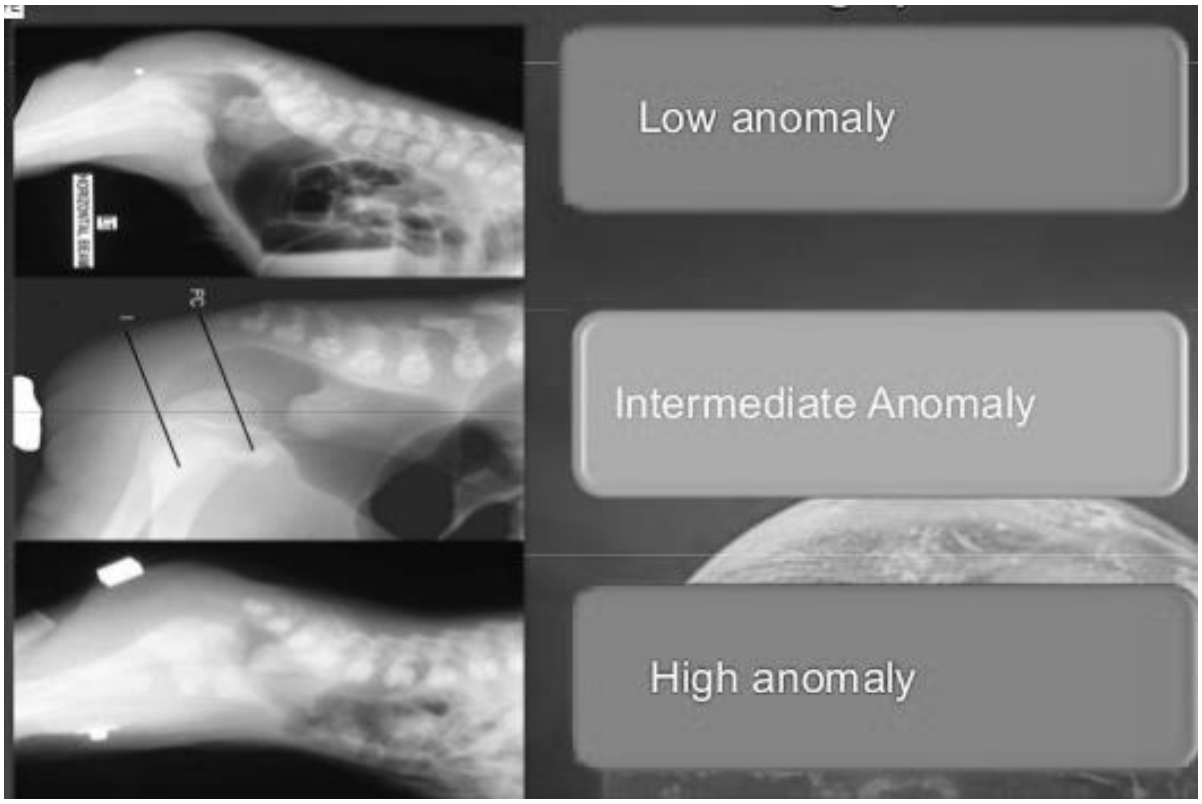


Figure If the caudal segments of the sacrum are deficient, the PC line can be developed by projection from the pubis through the same site on the ischium, which is approximately the junction between the upper quarter and the lower three-quarters.

TREATMENT

Most low malformations are treated by an anoplasty soon after birth. Higher, more complex defects need a temporary colostomy; after detailed investigations, reconstructive surgery is undertaken at a few months of age. In the posterior sagittal anorectoplasty (PSARP) dissection and reconstruction are performed through a midline sacroperineal incision Any fistula is divided, the distal rectai pouch is mobilised and placed within the pelvic muscles of continence (C, D), and an inversion anoplasty is fashioned. Functional outcome is related to the type of anorectal malformation (low defects are associated with constipation, higher defects with faecal incontinence) and the integrity of the sacrum and pelvic muscles.


Low anomaly
Intermediate Anomaly
High anomaly

DIAPHRAGMATIC HERNIA

DEVELOPMENT OF THE DIAPHRAGM

The anterior portion of the central tendon of the diaphragm develops from the septum transversum, the dorsolateral portions develop form lateral folds called the pleuroperitoneal membranes. The crura develop from the esophageal mesentery and the peripheral muscular portion arises from the thoracic intercostal muscle groups. Abnormalities in the development of the previously mentioned structures may result in diaphragmatic hernias.

DIAPHRAGMATIC HERNIAS

Diaphragmatic hernias are-one of the most common congenital anomalies associated with neonatal respiratory distress (others include pulmonary hypoplasia, TEF, cho anal atresia, and congenital heart diseases). It occurs in 1/4000 live births and most commonly (95%) results from failure of closure of the pleuroperitoneal canals resulting in a posterolateral defect (3), and may predispose what is called bochdalek hernia. Less than 5% are due a defect in the anterolateral portion (where the superior epigastric vessels enter through the foramen of Morgagni, and may result in what is called morgagni hernia. Herniation

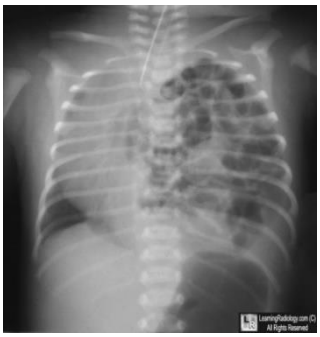
8% of neonates, who were born with diaphragmatic hernias, were born dead, however with the advent of antenatal diagnosis this percentage has been decreasing. Defects causing bochedalek hernias range from almost complete to agenesis and 80% of which occur in the left side, 19% occurring in the right side and 1% occurring bilaterally. Diaphragmatic hernias may present as either an intestinal obstruction with bilious emesis or more commonly, as respiratory distress symptoms including dyspnea, cyanosis, and tachycardia. The vomiting may indicate the presence of strangulation and is more commonly seen with morgagni type hernia. Examination may show a flat (or scaphoid) abdomen, shifting of the apex beat to the unaffected side, and reduced air entry and thus movement on the affected side. Bowel sounds are detected in the chest with shifting of heart sounds to the opposite side and absence of breathing sounds on the affected side.

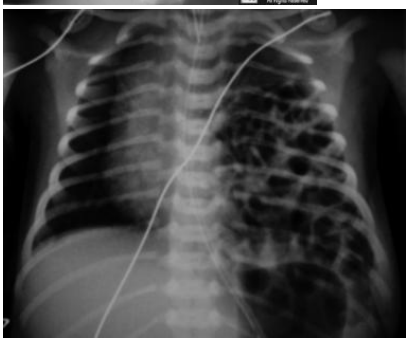
Chest x-rays will confirm the presence of gastrointestinal loops in the chest and may snow mediastinal shift of the stomach through the esophageal hiatus (2) may result in hiatus hernia, Weakness of the peripheral muscular portion of the diaphragm may result in diaphragmatic eventration. When diagnosing diaphragmatic hernias, it's important to assess the presence of malrotation in the gastrointestinal tracts (90%), CNS defects (spina befjda, hydrocephalus, and cerebral agenesis), cardiovascular defects(ASD, VSD, COO, and TOF) and the presence of chromosomal disorders (trisomy 18 and 21) as these conditions are closely associated with diaphragmatic hernias.

MANAGEMENT OF DIAPHRAGMATIC HERNIAS

Infants who present with respiratory distress and show typical features of diaphragmatic hernias on chest x-rays should not be ventilated with an ambu bag or rebreathing masks, as these may accumulate gas in a herniated stomach and cause further distress and strangulation, in addition to causing barotraumas and resultant pneumothorax on the unaffected site of the chest. Instead, positive pressure ventilation is used, keeping pressure at 30 cm H2O, can maintain blood PH at more than 7.5, however this is also associated with the risk of contralateral pneumothorax. A nasogastric tube is important as it may decompress the stomach and relief intra thoracic pressure. Ringer lactate should be given for intravascular resuscitation and correction of respiratory acidosis. Blood should be monitored by ABG's.

After resuscitation is complete, surgery is indicated. An abdominal approach is less invasive, using a subcostal incision. The herniated viscera are pulled back to the abdominal cavity and the defect is closed by suturing (if small) or by using a silastic mesh (if large). If there is malrotation, Ladd's procedure should be performed.



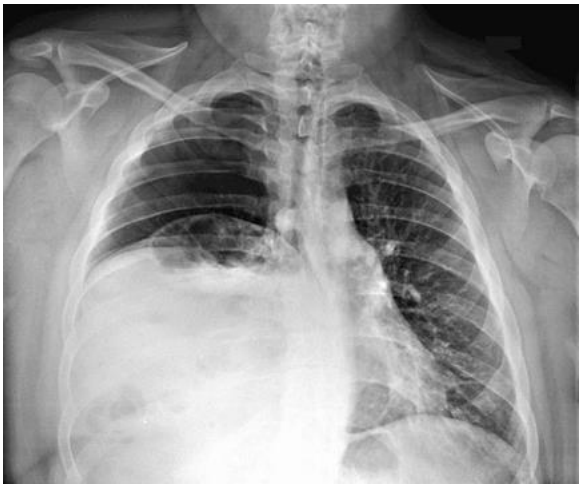


EVENTRATION OF THE DIAPHARGAM

Congenital anomalies resulting in absence of muscularization of the peripheral portion of the diaphragm, or weakness of that portion secondary to phrenic nerve injuries, may allow the abdominal viscera to push the diaphragm into the chest causing what is similar to diaphragmatic hernias. Chest x-rays may show the presence of bowel covered by a radiopaque layer, inside the chest. However, the diagnosis is complete by using fluoroscopy which may show paradoxical diaphragmatic elevation on inspiration. Respiratory distress and\or functional deficit are enough indications for surgery. Surgery involves strengthening the diaphragm by plication with non-absorbable sutures.

PROGNOSIS

Prognosis is related to the time of onset of symptoms and the degree of respiratory impairment. Generally poor prognosis is associated with early respiratory failure, presence of cardiac anomalies, lung agenesis and polyhydramnios, and presence of liver or stomach in the chest. Good prognosis is associated with early antenatal diagnosis.



MORGAGNI HERNIA

This uncommon condition may result from herniation of the abdominal viscera through the foramen of Morgagni, where the internal mammary and superior epigastric vessels pass. It's rarely symptomatic, but may present as bilious vomiting with coughing and choking secondary to pressure on mediastinal structures. It's closely associated with ectopic cordis, bifid sternum, Exomphalos, and cardiac anomalies. Chest X-ray may show the presence of intestinal loops in the chest and mediastinum, but is distinguished from hiatus hernia only by barium contrast on lateral chest films (anterior is Morgagni hernia and posterior is hiatus hernia)

HIATUS HERNIA

A hiatus hernia is an abnormal protrusion of the stomach through the oesophageal diaphragmatic hiatus into the thorax. There are two types: sliding (90%) and rolling (10%). A sliding hernia occurs when the stomach slides through the diaphragmatic hiatus, so that the gastroesophageal junction lies within the chest cavity. It is covered anteriorly by peritoneum, and posteriorly is extra-peritoneal . A rolling or para-oesophageal hernia is formed when the stomach rolls up anteriorly through the hiatus; the cardia remains in its normal position and therefore the cardio-oesophageal sphincter remains intact. Rolling and sliding hernias are caused by weakness of the muscles around the hiatus. They tend to occur in middle-aged and elderly patients. Women are affected more frequently than men and there is a higher incidence in the obese. However, of interest, is that which occurs in children.

CLINICAL FEATURES

Hiatus hernias are often asymptomatic, but can produce some of or all the following symptoms:

- *Heartburn and regurgitation* owing to an incompetent lower oesophageal sphincter, which is aggravated by stooping and lying flat at night, and can be relieved by antacids.
- *Oesophagitis* resulting from persistent acid reflux, which leads to ulceration, bleeding with anaemia, fibrosis and stricture formation.
- *Epigastric and lower chest pain,* especially in para-oesophageal hernias, as the herniated part of the stomach (usually the fundus) becomes trapped in the hiatus. This can be a surgical emergency owing to the obstruction and strangulation of the stomach.
- Palpitations and hiccups, symptoms caused by the mass effect of the hernia .in the thoracic cavity irritating the pericardium and the diaphragm. In patients with a large rolling hiatus hernia, displacement of the whole stomach may result in a volvulus into the chest, producing symptoms of vomiting from gastric outflow obstruction.

GASTROESOPHAGEAL REFLUX

It is caused by the retrograde flow of gastric acid through an incompetent cardiac sphincter into the lower esophagus. The cardiac sphincter usually prevents reflux by the following mechanisms:

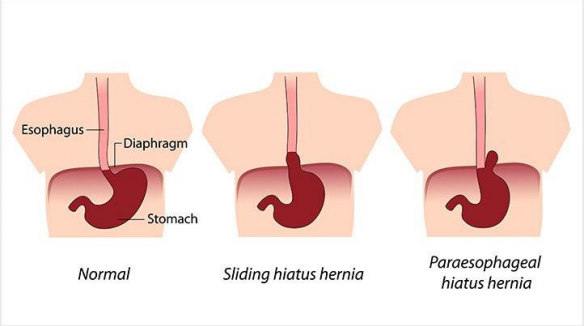
- a physiological high-pressure zone (not a true sphincter) in the lower end of the oesophagus
- the mucosal rosette at the cardia, which acts like a plug
- the angle at which the oesophagus joins the stomach between the left border of the oesopha¬gus and the fundus (angle of His). .
- the diaphragmatic sling (crura), which acts like a pinchcock at the lower end of the oesophagus
- The high-pressure area at the lower end of the esophagus, caused by the positive intraabdominal pressure.

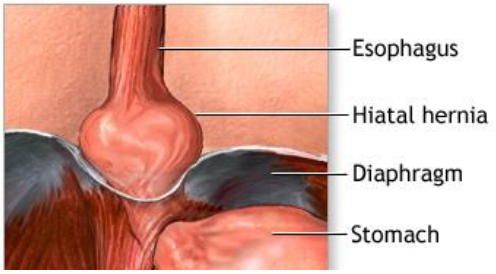
INVESTIGATIONS

The diagnosis of hiatus hernia may be confirmed by performing a barium swallow and meal, or by upper gastrointestinal endoscopy. A chest X-ray may show a fluid level behind the heart and a widened mediastinum.

MANAGEMENT

Conservative management in children includes feeding the baby while decreasing the angle of His by keeping the baby supine. Giving the baby solid food may decrease the associated reflux symptoms and gastric decompression is of value. Failure of conservative management, presentation of esophagitis, anemia, strangulation and intractable vomiting are all indications for anti-reflux surgery. The most common procedure currently performed is the Nissen's fundoplication, in which the fundoplication is taken posteriorly around the lower oesophagus and sutured to the left anterior surface of the left side of the proximal stomach as a 3608 wrap.





ESOPHAGEAL ATRESIA AND TRACHE-OESOPHAGEAL FISTULAS

Esophageal atresia (EA) and tracheoesophageal fistula (TEF) are among the most gratifying pediatric surgical conditions to treat. And include complete interruption of the esophageal lumen with or without the presence of a fistula with the tracheal lumen. It affects 1/3000-4000 live births.

The most commonly seen variety is EA with distal TEF (type C), which occurs in approximately 75—85% of the cases in most series. The next most frequent is pure EA (type A), occurring in 8 to 10% of patients, followed by TEF without EA (type E). This occurs in 5-8% of cases, and also is referred to as an H-type fistula, based on the anatomic similarity to that letter. EA with fistula between both proximal and distal ends of the esophagus and trachea (type D) is seen in approximately 1-2% of cases, and type B, EA with TEF between proximal segments of esophagus and trachea, is seen in approximately 1% of all cases.

EA-TEF may be associated with other congenital anomalies.

These defects are known by the acronyms VATER or VACTERRL syndrome, which refers to vertebral (missing vertebra) and anorectal (imperforate anus) anomalies, cardiac defects (severe congenital cardiac disease), tracheoesophageal fistula, renal anomalies (renal agenesis and renal anomalies), and radial limb hyperplasia. In nearly 20% of infants born with esophageal atresia, some variant of congenital heart disease is present.

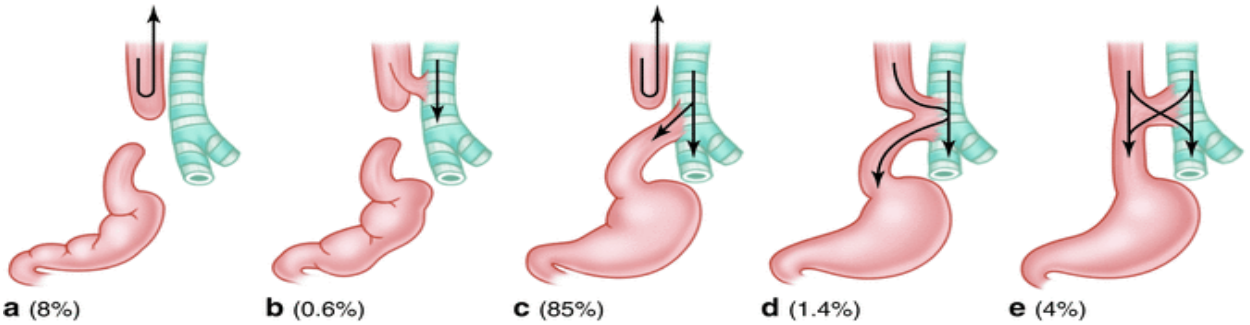
CLINICAL PRESENTATION

The anatomic variant of infants with EA-TEF predicts the clinical presentation. When the esophagus ends either as a blind pouch or as a fistula into the trachea (as in types A, B, C, or D), infants present with excessive drooling, followed by choking or coughing immediately after feeding. As a result, aspiration occurs through the fistula tract. As the neonate coughs and cries, air is transmitted through the fistula into the stomach, resulting in abdominal distention (type C and D).

As the abdomen distends, it be- comes increasingly more difficult for the infant to breathe. This leads to further atelectasis, which compounds the pulmonary dysfunction. In patients with type C and D varieties, the regurgitated gastric juice passes through the fistula, where it collects in the trachea and lungs and leads to a chemical pneumonitis, which further exacerbates the pulmonary status.

In many instances, the diagnosis actually made by the nursing staff, who attempt to feed the baby and notice the accumulation of oral secretions. Death is caused primarily by pulmonary complications but may also be due to prematurity or associated anomalies. Good prognosis is associated with full term babes who are generally in a good condition and in those who. were detected early. Early antenatal diagnosis may increase the survival rate up to 98% as it may decrease the incidence of pulmonary complications.

the presence of gas below the diaphragm, which confirms the presence of the tracheoesophageal fistula.



DIAGNOSIS

A diagnosis of EA-TEF can be suspected prenatally on ultrasound evaluation. Typical features include failure to visualize the stomach (collapsed because of inefficient swallowing) and the presence of polyhydramnios. These findings reflect the absence of efficient swallowing by the fetus. Other findings include lower than expected fetal weight and a distended esophageal pouch.

Neonates who have copious, fine white frothy bubbles of mucus in the mouth and nose with recurrence of secretion

The dilated upper pouch may occasionally be seen on a plain chest radiograph and sometimes associated with tracheal compression and deviation. If a soft feeding tube is used, the tube will coil in the upper pouch, which pro- vides further diagnostic certainty. The absence of a gastric bubble may indicate the absence of an associated fistula. Whenever there is any diagnostic uncertainty, a contrast study will confirm the diagnosis of EA and occasionally document the TEF. A 1-2 ml of barium is instilled through an 8F catheter placed in the esophagus.

after suctioning are suspected to have EA. History may tell the presence of drooling, dysphagia, regurgitation of milk with episodes of rattling respiration, coughing, choking and cyanosis and worsening of these symptoms after feeding. Physical examination may show resonant abdominal percussion and indicates the presence of a fistula. The diagnosis of esophageal atresia is confirmed by the inability to pass an orogastric tube into the stomach which may stop when the distance between its proximal pore and the baby's incisor teeth is 10 cm.

Chest radiographs are taken in the lateral decubitus position .as well as the antero-posterior position to detect spilling of contrast into the trachea. Although, these studies have high diagnostic accuracy they are well associated with the risk of aspiration pneumonitis and pulmonary injury and add minimal information to the diagnostic workup. Bronchoscopy may be performed to exclude the presence of additional, upper pouch fistulae in cases of esophageal atresia (i.e. differentiation of types B, C, and D), and identification of a laryngeotracheoesophageal cleft if the patient is to be operated.

In a child with esophageal atresia, it is important to identify whether coexisting anomalies are present. These include cardiac defects in 38%, skeletal defects in 19%, neurologic defects in 15%, renal defects in 15%, anorectal defects in 8%, and other abnormalities in 13%.

Examination of the heart and great vessels with echocardiography is important to exclude cardiac defects, as these are often the most important predictors of survival in these infants. The echocardiogram also demonstrates whether the aortic arch is left sided or right sided, which may influence the approach to surgical repair.

Vertebral anomalies are assessed by plain radiography, and a spinal ultrasound is obtained if any are detected. A patent anus should be confirmed clinically. The kidneys in a newborn may be assessed clinically by palpation.

An ultrasound of the abdomen will demonstrate the presence of renal anomalies, which should be suspected in the child who fails to make urine. the presence of extremity anomalies is suspected when there are missing digits, and confirmed by plain radiographs of the hands, feet, forearms, and legs. Rib anomalies may also be present. These may include the presence of a thirteenth rib.

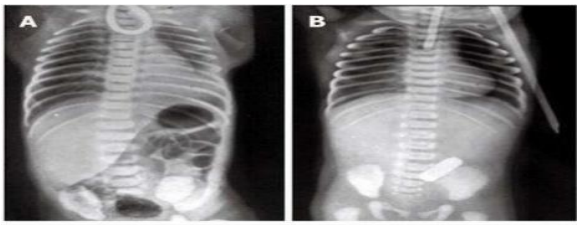


Fig (A) Orogastric catheter in the blind esophageal pouch & presence of air in the stomach.

Fig (B)The radiopaque tube in the blind esophageal pouch & absence of air in the stomach

INITIAL MANAGEMENT

The initial strategy after the diagnosis is confirmed is to place the neonate in an infant warmer with the head elevated at least 30 degrees. A sump catheter is placed in the upper pouch on continuous suction. Both of these strategies are designed to minimize the degree of aspiration from the esophageal pouch When saliva accumulates in the upper pouch and is aspirated into the lungs, coughing, bronchospasm, and desaturation episodes can occur, which may be minimized by ensuring the patency of the sump catheter, Intravenous antibiotic

therapy is initiated, and warmed electrolyte solution is administered. Where possible, the right upper extremity is avoided as a site to start an intravenous line, as this location may interfere with positioning of the patient during the surgical repair.

The timing of repair is influenced by the stability of the patient. Definitive repair of the EA-TEF is rarely a surgical emergency. If the child is hemodynamically stable and is oxygenating well, definitive repair may be performed within 1 to 2 days after birth. This allows for a careful determination of the presence of coexisting anomalies and for selection of an experienced anesthetic team.

PRIMARY SURGICAL CORRECTION

In a stable infant, definitive repair is achieved through performance of a primary esophagoesophagostomy. The infant is brought to the operating room, intubated, and placed in the lateral decubitus position with the right side up in preparation for a right posterolateral thoracotomy. If a right-sided arch was determined previously by echocardiography, consideration is given to performing the repair through the left chest, although most surgeons believe that the repair can be performed safely from the right side as well.

The. operative technique for primary repair is as follows . A retropleural (extraplueral) approach is generally used, as this technique prevents widespread contamination of the thorax if a postoperative anastomotic leak occurs. The sequence of steps includes:

1. Mobilization of the pleura to expose the structures in the posterior mediastinum. Using a right posterolateral thoracotomy approach may require division and ligation of the azygos vein.

2. Division of the fistula and closure of the tracheal opening by a mediastinal flap.

3. Mobilization of the upper esophagus sufficiently to permit an anastomosis without tension, and to determine whether a fistula is present between the upper esophagus and the trachea. Forward pressure by the anesthesia staff on the sump drain in the pouch can greatly facilitate dissection at this stage of the operation. Care must be taken when dissecting posteriorly to avoid violation of either the lumen of the trachea or esophagus.

4. Mobilization of the distal esophagus. This needs to- be performed judiciously to avoid devascularization, since the blood supply to the distal esophagus is segmental from the aorta. Most of the esophageal length is obtained from mobilizing the upper pouch, since the blood supply travels via the submucosa from above. Also care must be taken to avoid injury to branches of the vagus nerve supplying the esophagus;

5. Performing' a primary esophagoesosophageal anastomosis. Most surgeons perform this procedure in a single layer using 5-0 sutures. If there is excess tension, the muscle of the upper pouch can be circumferentially incised without compromising blood supply to increase its length, Many surgeons place a trans anastomotic feeding tube in order to institute feeds in the early postoperative period.

6. A retropleural drain is placed, and the incision is closed in layers.

If anastomosis is impossible, a staged procedure may be required, where by the surgeon suffices with dividing the fistula at this stage and a gastrostomy is performed, to allow the baby to feed. A cervical esophagostomy is required to allow the baby to learn breast feeding as its crucial at this stage of life. Continuous esophageal suction is important, and TPN is instituted. These conservative techniques are used for few months until anastomosis is feasible, if not, colonic or gastric flaps are used to replace the missing segment. Conservative management using the previously mentioned techniques can also be useful in inoperable patients; prematurity, low birth weight,

POSTOPERATIVE COURSE

The infant should be ventilated as needed. A gastrostomy feeding should be resumed. When a transanastomotic tube is placed, feeds are begun slowly in the postoperative period. Some surgeons institute parenteral nutrition for several days, using a central line. The retropleural drain is assessed daily for the presence of saliva, indicating an anastomotic leak. Many surgeons obtain a contrast swallow 1 week after repair to assess the caliber of the anastomosis and to determine whether a leak is

present. If there is no leak, oral feedings are started. After 3 weeks, an esophageal dilatation (24f) is performed to prevent future esophageal stenosis.

COMPLICATIONS OF SURGERY

Anastomotic leakage occurs in 10 to 15% of patients.

Early leakage is manifested by a new pleural effusion, pneumothorax, and sepsis, and requires immediate exploration. Anastomotic leakage that is detected after several days usually heals without intervention, particularly if a retropleural approach is used. Strictures are not infrequent (10 to 20%), particularly if a leak has occurred.

"Recurrent" tracheoesophageal fistula may represent a missed upper pouch fistula or a true recurrence. This may occur after an anastomotic disruption, during which the recurrent fistula may heal spontaneously. Otherwise, reoperation may be required.

Gastroesophageal reflux commonly occurs after repair of EA-TEF, potentially due to alterations in esophageal motility and the anatomy of the gastroesophageal junction. Brassy cough is not uncommon and may persist for 12-18 months after the operation, after which it disappears spontaneously as the trachea grows.

H-TYPE FISTULA

Patients with type E TEFs (also called H-type) most commonly present beyond the newborn period. Presenting symptoms include coughing that is aggravated by feeding, cyanosis, recurrent chest infections, bronchospasm, and failure to thrive. The abdomen is distended and filled with air and there is a high risk of peptic pneumonitis due to reflux of gastric acid. The diagnosis is suspected using barium esophagography, and confirmed by endoscopic visualization of the fistula. Surgical correction is generally possible through a cervical approach, and requires mobilization and division of the fistula. Outcome usually is excellent.

ABDOMINAL EMERGENCIES IN CHILDREN

The most common presentation in abdominal emergen¬cies is pain. The pain in newly born children is expressed by screaming and crying attacks, however, in older chil¬dren the pain can be expressed linguistically (symptoms) and with body language (signs). Identification of the type, pattern, and mechanism of the cause can all point the surgeon to the underlying pathology.

2 types of pain are known, visceral and somatic. Visceral pain is due to distention of the capsules (adventitia or serosa) surrounding abdominal organs. Nearly all types of pain start by a visceral pain, especially in those with pathological events that involves distention. Visceral pain is received by the autonomic nerves, and since these nerves only supply the capsule, the ability of the brain to localize the site of pain is Very poor, thus, visceral pain is always described as being in the midline or generalized. Somatic pain on the other side is received by somatic nerves, which only innervates the parietal peritoneum, it only occurs when the parietal peritoneum is involved by an inflammatory process. The number of nerves innervating the peritoneum is much more, comparing to those of the autonomic nerves supplying the viscera, thus, the pain is more localized. Pain can be caused by distention of the capsules, peritoneal inflammation, spasm, irritation, traction, ischemia or malignant infiltration. Processes that follow include acute inflammation, peritonitis, or strangulation.

It's important for the surgeon to exclude systemic causes of abdominal pain as these have special line of treatment. Systemic causes of pain in children are shown in the table below.

systemic causes of abdominal pain

- Septicaemia
- Heart failure
- Diabetes mellites (type 1)
- Sickle cell crises
- uraemia

he cardinal symptoms of an abdominal emergency are pain, vomiting, diarrhea. All presentations of abdominal pain lasting 3-4 hours or more, should be regard as an abdominal emergency until proven otherwise. On the other hand, vomiting and diarrhea are not included in this definition. However, they may point to the surgeon an underlying pathology, for example a persistent vomiting should raise the possibility of a small bowel obstruction, or 24-hour diarrhea should suggest the possibility of pelvic lesion.

It's of greater importance to establish wither pain is still present, becoming worse, or subsiding. Clinical monitoring should be done every 3 hours.

SIGNS THAT ARE USED TO MONITOR ABDOMINAL PAIN AND THEIR INTERPRETATION

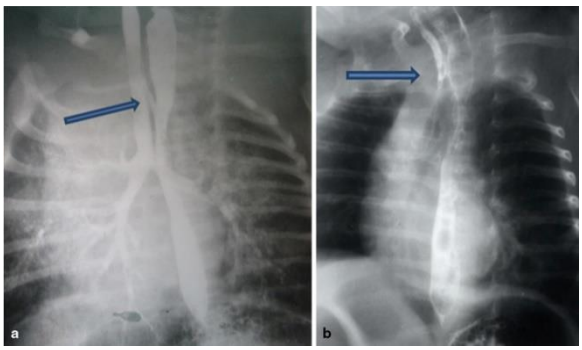
Referred tenderness; indicates the presence of distended or inflamed bowel. Its elicited by direct palpation on the opposite site of pain

Localized tenderness; found in

Excess fiatus
Overloaded colon
Hyperplastic lymph nodes
Acute appendicitis
Strangulated gut

Guarding; a variable degree of involuntary increased muscle resistance which indicates local peritonitis

Board like rigidity; indicates general peritonitis



HYPERTROPHIC PYLORIC STENOSIS (HPS)

Hypertrophic pyloric stenosis (HPS) is a condition that is associated with hypertrophy of the circular muscle of the pyloric canal of the stomach with increased length and wall thickness and decreased lumen diameter, thus causing outflow obstruction. Timely diagnosis and. treatment of infants with hypertrophic pyloric stenosis (HPS) is extremely gratifying. It is one of the few instances in surgery in which a relatively simple operation can have such a dramatic long-term effect. HPS occurs in approximately 1 in 300 live births, and classically presents in a first-born male between 2 and 6 weeks of age. However, children outside of this age- range also are commonly seen, and the disease is by no means restricted to either males or first-born children. The cause of HPS has not been determined. Studies have shown that HPS is found in several generations of the same family, suggesting a familial link.

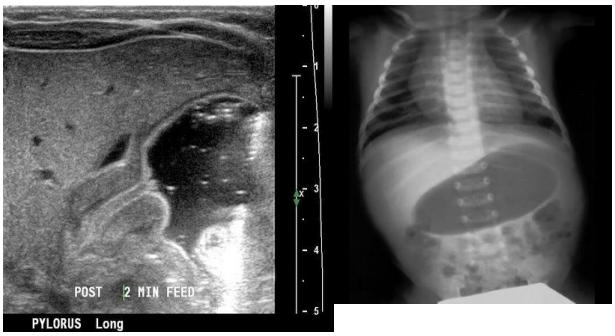
CLINICAL FEATURE

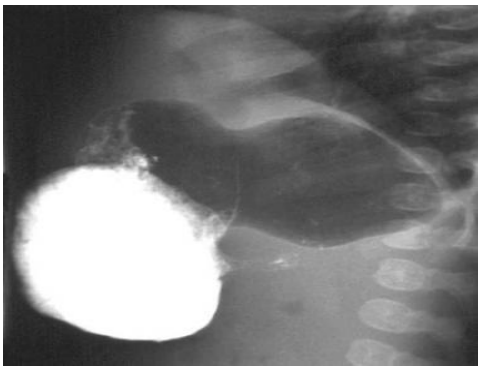
Infants with HPS usually present in the 2nd week of like, with recurrent non bilious vomiting that becomes increasingly projectile over the course of several days to weeks. The vomits usually contains elements of milk with gastric mucus. Despite the recurrent emesis, the child normally has a voracious appetite, leading to a cycle of feeding and vomiting that invariably results in severe dehydration if left untreated. The patient is usually crying and is restless due to pain and hunger. The child usually loses weight and fails to thrive. Particularly perceptive caregivers will mention that their infant is passing less flatus, which provides a further clue that gastric outlet obstruction is complete. A coffee ground vomits indicates the: development of gastritis.

Continuous cycles of feeding and vomiting may. cause dehydration and the infant may develop hypochloremia, hypokalemic metabolic alkalosis. Examination may reveal palpable "olive" like mass in the right upper quadrant, and the presence of visible gastric waves that pass from left to right on the abdomen. A feeding test usually allows better palpation of the mass, and may show better peristalsis.

DIAGNOSIS

The diagnosis of pyloric stenosis usually can be made on physical examination. When the olive cannot be palpated, ultrasound can diagnose the condition accurately in 95% of patients. Criteria for ultrasound, diagnosis include a channel length of over 16 mm and pyloric thickness over 3 mm. barium meal may also be of value in the diagnosis, but less accurate than ultrasound: The presence of a 'string 'sign with a filling defect at the proximal orifice of the canal ('shouldering'), is usually diagnostic.





Differential diagnosis for HPS is shown in the table below.

differential diagnosis for HPS
mismanagement of the feeding program (over feeding)
malrotation (annular pancreas or subepatic cecum)
gastroesophageal reflux
intracranial conditions (cerebral birth injuries, meningitis)
septicaemia

TREATMENT

Pyloric stenosis is never a surgical emergency, although dehydration and electrolyte abnormalities may present a medical emergency. Fluid resuscitation with correction of electrolyte abnormalities and metabolic alkalosis Is essential before induction of general anesthesia for surgery. After resuscitation, a Fredet-Ramstedt pyloromyotomy is performed. It may be performed using an open or laparoscopic approach. The open pyloromyotomy is performed through either an umbilical or a right upper quadrant transverse abdominal incision. The operation involves dividing the serosa and then splitting the pyloric muscle until the submucosa is seen bulging upward.

INTUSSUSCEPTION

Intussusception is the leading cause of intestinal obstruction in the young child. It refers to the condition whereby a segment of intestine becomes drawn into the lumen of the more proximal bowel. The process usually begins in the region of the terminal ileum (80%), and extends distally into the ascending, transverse, or descending colon. Rarely, an intussusception may intussuscept through 'the rectum. The pathologic results of such condition are compression on the mesenteric vessels with resultant congestion, strangulation, obstruction, gangrene and perforation.

The cause of intussusception is not clear, although current thinking suggests that hypertrophy of the Peyer's patches in the terminal ileum from an antecedent viral infection acts as the starting point. Peristaltic action of the intestine then causes the bowel distal to this point to invaginate into itself, this is called primary intussusceptions. Peak incidence of Primary intussusception is between 5 and 10 months of age, at which, the feeding habits of the baby usually is changed. Idiopathic intussusception (secondary intussusception) occurs in children between the ages of approximately 6 and 24 months. Beyond this age group, one should consider the possibility that a pathologic starting point may be present. These include polyps, malignant tumors such as lymphoma, enteric duplication cysts, or Meckel's diverticulum. Such intussusceptions are rarely reduced by air or contrast enema , and thus the starting point is identified when operative reduction of the intussusception is performed.

CLINICAL MANIFESTATIONS

Typically, the infant develops paroxysms of crampy abdominal pain, lasting 2-3 minutes, with intermittent vomiting (milky then becomes bilious) that is associated with pallor, exhaustion and drowsiness. Between attacks, the infant may act normally, but as symptoms progress, increasing lethargy develops. Bloody mucus (”red currant- Jelly" stool) may be passed per rectum. Examination may reveal the attacks of pain, as the patient draws up his. knees but relaxes when the spasm eases. A palpable sausage mass may be found anywhere around the umbilicus. Rectal examination may reveal blood or sometimes palpate the congested tip of the intussuscepted mass. It's important to distinguish between rectal prolapse and the tip of the intussuscepted mass, usually a rectal prolapse has a palpable attachment in the rectum, while intussusception will seem like an object hanging from a sealing; with no palpable attachment.

INVESTIGATIONS

Plain X-rays may show signs of small bowel obstruction and with soft tissue opacity. the diagnosis is confirmed by contrast barium enema which may show 'coiled spring' sign, a 'crescent or meniscus sign', or a 'first sign'. Ultrasound may also assist in the diagnosis and may show a kidney like mass or a target sign.

MANAGEMENT

Patients with intussusception should be assessed for the presence of peritonitis and for the severity of systemic illness. Following resuscitation and administration of intravenous antibiotics, the child is assessed for suitability to proceed with nonoperative versus operative reduction.

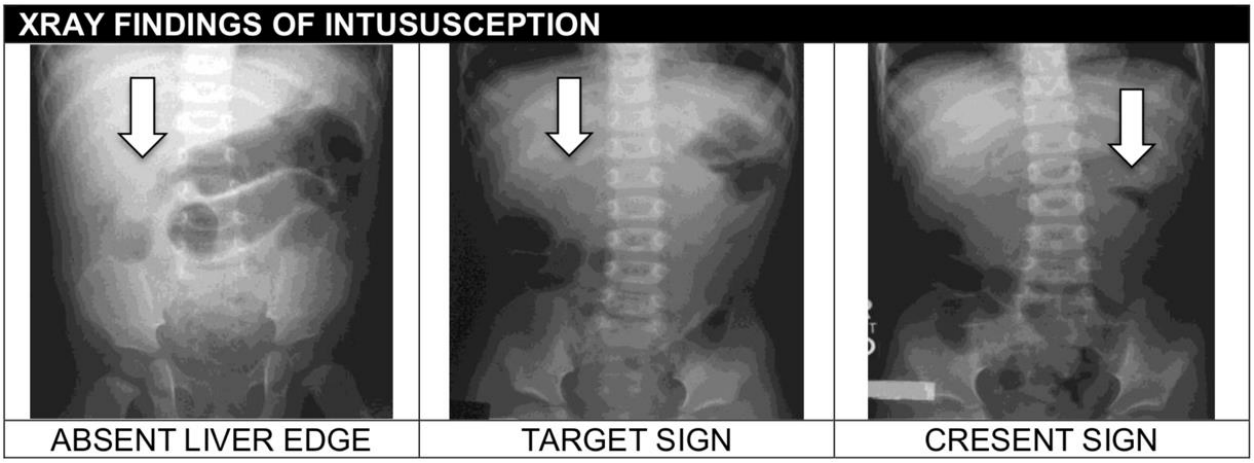
The presence of a pathologic leading point, presence of peritonitis, perforation or/and the presence of shock are contraindications for performing nonoperative reduction. In the stable patient, Treatment involves an attempt at radiographic or pneumatic reduction of the Intussusception using barium or air enema, respectively.

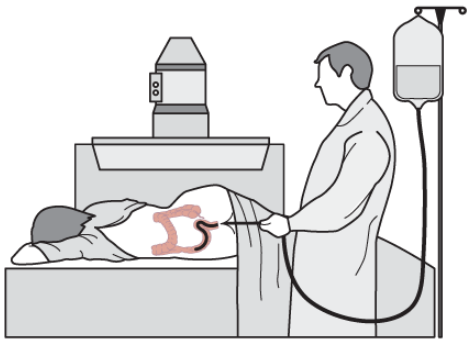
A small tube is passed into the rectum and barium or air is pumped in using a pressure-limited valve. The reduction is monitored using X-ray screening (fluoroscopy). A second or third attempt, after a delay of an hour or so between efforts, may succeed where the initial attempt fails. Successful reduction

is marked by free reflux of air into multiple loops of small bowel, and symptomatic improvement as the infant suddenly becomes pain free.

Unless both of these signs are observed, it cannot be assumed that the intussusception is reduced. Overall success rates for pneumatic reduction in excess of 80% have been reported, whilst that of radiographic reduction varies and is typically between 60 and 90%. Recurrent intussusception occurs in up to 10% of cases reduced non operatively.

Failure to reduce the intussusception in 3 attempts, presence of peritonitis, perforation, the presence of shock ,and the presence of a pathologic leading point, recurrence after non operative reduction are all indications of surgery. Surgery can be done open with manual reduction, or laparoscopic. An irreducible intussusception, or a strangulated segment can be treated by resection and anastomosis.





ACUTE APPENDICITIS

Acute appendicitis is the most common emergency in patients of child bearing age.

ETIOLOGY AND PATHOGENESIS

ETIOLOGY

Obstruction of the lumen is the dominant causal factor in acute appendicitis. Fecaliths are the usual cause of appendiceal obstruction. Less-common causes are hypertrophy of lymphoid tissue, tumors, vegetable and fruit seeds, and intestinal parasites. The frequency of obstruction rises with the severity of the inflammatory process. Some cases of Appendicitis result from causes other than obstruction, like those occurring in the. course of pneumonia and tonsillitis, in which direct hematogenous spread of micro-organisms to the lumen may take place.

PATHOGENESIS

There is a predictable sequence of events leading to eventual appendiceal rupture. The proximal obstruction of the appendiceal lumen produces a closed-loop obstruction, and continuing normal secretion by the appendiceal mucosa rapidly produces distention. Distention of the appendix stimulates nerve endings of visceral afferent stretch fibers, producing vague, dull, diffuse pain in the mid-abdomen or lower epigastrium.

Peristalsis is also stimulated by the rather sudden distention, so that some cramping may be superimposed on the visceral pain early in the course of appendicitis.

Distention continues from continued mucosal secretion and from rapid multiplication of the resident

bacteria of the appendix. Distention of this magnitude usually causes reflex nausea and vomiting, and the diffuse visceral pain becomes more severe. As pressure in the organ increases, venous pressure is exceeded.

Capillaries and venules are occluded, but arteriolar inflow continues, resulting in engorgement and vascular congestion. The inflammatory process soon involves the serosa of the appendix and in turn parietal peritoneum in the region, producing the characteristic shift in pain to the right lower quadrant. The mucosa of the gastrointestinal tract, including the appendix, is susceptible to impairment of blood supply, thus its integrity is compromised early in the process, allowing bacterial invasion.

As progressive distention encroaches upon first the venous return and subsequently the arteriolar inflow, the area with the poorest blood supply suffers most: ellipsoidal infarcts develop in the antimesenteric border. As distention, bacterial invasion, compromise of vascular supply, and infarction progress, perforation occurs, usually through one of the infarcted areas on the antimesenteric border.

Perforation generally occurs just beyond the point of obstruction rather than at the tip because of the effect of diameter on intraluminal tension.

PRESENTATION

Periumbilical pain that localizes few hours later to the right lower quadrant, followed by nausea, vomiting, fever, and localized peritoneal irritation in the region of McBurney's point. Alternating constipation and. diarrhea may ensue. History may reveal symptoms typical of tonsillitis or pneumonia. Generalized lymphadenopathy may also be present in the history.

Some forms of gastroenteritis may progress into appendicitis and thus, vomiting and diarrhea may sometimes precede pain. on Examination, pyrexia and tachycardia may appear and most certainly indicates an infective cause.

Generally, the child is reluctant to move or cough, as these may aggravate the pain. On palpation there is a localized tenderness on the right iliac fossa, with positive psoas sign, obturator sign (both are due to muscle irritation), Rovsing's sign, and rebound tenderness. There is a sever muscle guarding and rigidity at the site of the inflamed appendix. Active observation and repeating abdominal examination, prevents many unnecessary explorations.

INVESTIGATIONS

Blood counts may depict neutrophils leukocytosis. Abdominal X-rays may show small bowel dilatation or feacolith calcification. Ultrasound may show a noncompressible tubular mass (that doesn't change its diameter after compression by the radiologist), fluids, mesenteric thickening, or a peri appendicular abscess the use of CT scan in the diagnosis of appendicitis is sometimes unnecessary. The differential diagnosis for acute appendicitis in children is shown in the table below.

Differential diagnosis for acute appendicitis in children
• Mesenteric adenitis
• Meckel's diverticulitis
• Primary peritonitis
• Ruptured ovarian cyst
• Torsion of an ovarian cyst or ovary
• Torsion of an omentum
• Suppurating deep iliac lymph nodes

TREATMENT

It's well accepted that any case of suspected appendicitis that shows typical features on ultrasound should go through appendicectomy (laparoscopic or open). A perforated appendix should be treated as a case of peritonitis. If there is a periappendicular abscess, it must be drained. Some of the complications of acute appendicitis are the formation of an appendicular mass secondary to walling off of the inflamed appendix by an omentum, which may or may not be associated with an abscess. This complication may resolve spontaneously in 80% of cases but if not should undergo interval appendicectomy after 8-12 weeks.

ACUTE MESENTERIC ADENITIS

Acute mesenteric adenitis is the disease most often confused with acute appendicitis in children. Almost invariably, an upper respiratory infection is present or has recently subsided. The pain is usually diffuse, and tenderness is not as sharply localized as in appendicitis. Voluntary guarding is sometimes present, but true rigidity is rare. Generalized lymphadenopathy may be noted. Laboratory procedures are of little help in arriving at the correct diagnosis, although a relative lymphocytosis, when present, suggests mesenteric adenitis. Observation for several hours is in order if the diagnosis of mesenteric adenitis seems likely, because mesenteric adenitis is a self-limited disease. However, if the differentiation remains in doubt, immediate exploration is the safest course of action.

ABDOMINAL MASSES IN CHILDREN

An abdominal mass is a common symptom in children and may be due to abnormalities in the gastrointestinal tract, gastrointestinal tracts associated viscera, urinary system, spleen, or reproductive organs.

CLINICAL DIAGNOSIS OF ABDOMINAL MASSES

Clinical diagnosis stand's superior to other means of diagnosis in the case of an abdominal mass, as it's the main guidance to investigations and management. Assessment of a mass in the abdomen should focus on the site, size, consistency, and mobility. The site may give a clue about the organ of origin (see the table on the side). Pathologic masses have certain ranges of sizes, thus, once the mass size is assessed, it can give a clue about the underlying pathology. Consistency may identify weather the mass is cystic (soft) or solid (firm) in nature. Mobility may help identify to which structure the mass is fixed (e.g.; an enterogenous cyst only moves with the intestines). Once these parameters are assessed, the age of the patient, the length of the complaint and the type of the main and associated symptom can further help in the process of. diagnosis.

The gender of the patient is very important in assessing lower abdominal masses, Patients who are of female gender with a lower abdominal mass should be suspected to have an imperforate hymen, an ovarian cyst or an ovarian tumor, thus a vaginal inspection should be performed once a lower abdominal mass is detected. If there is an imperforate hymen the mass is probably due to accumulation of vaginal secretions in the bladder through a vesicovaginal fistula and is called hydrocolpos (if presentation was delayed until menarche the mass is called hemocolpos). Ovarian cysts and tumors may present with abdominal pain, a palpable abdominal mass, evidence of urinary obstruction, symptoms of bowel obstruction, and endocrine imbalance.

It's important for you to take into consideration some of the common masses in children which might be of non-pathological origin, for example, the liver is usually palpable below the costal margin in patients under the age of 3-4 years. Faecal impaction is common among children under 1 year of age and may occur secondary to changes in feeding programs. It may cause an abdominal mass secondary to obstruction and accumulation of gasses. Fecal impaction can be excluded by using abdominal films. Before abdominal examination, always make sure that the patient's bladder is empty, as a full bladder may cause an abdominal mass.

Abdominal masses in children
Masses related to the G.l tract 1. Appendicular mass (right iliac fossa) 2. Congenital hypertrophicpyloric stenosis (epigastrie sausage like mass) 3. Volvulus (central ill-defined mass (formed of aggregated loops)) 4. Enterogenous cyst or mesenteric cyst (anywhere in the gastrointestinal tract) 5. intestinal lymphoma 6. Crohn's disease
Lower abdominal masses *1.* Imperforate hymen *2.* Ovarian cyst *3.* Ovarian tumor
Right subcostal 1. Hepatomegaly 2. Biliary atresia 3. Choledochal cyst 4. Hydatid cyst 5. hepatoblastoma
Left subcostal 1. Splenomegaly (thalassemia) 2. Hydatid cyst
Loin masses 1. Wilm's tumor 2. Hydronephrosis 3. neuroblastoma
Others Retroperitoneal sarcomas and teratomas

CLINICAL FEATURES

Usually the mass itself is the presenting feature, which is typically discovered by the mother while drying the child's abdomen after a bath. However, masses can be painful and the baby may present

with screaming attacks, where by a mass can only be detected after examination. When doing an examination, palpation should be restricted as it may promote embolizing metastasis of certain types of tumors; the most common is wilm's tumor.

INVESTIGATIONS

Plain films of the abdomen may show calcifications within a soft tissue mass, or displacement of bowel loops in volvulus neonatorum. Ultrasound is useful in identifying hydronephrosis, cysts, multilocular cysts, solid masses and the origin of any type of these masses. Intravenous pyelography can be used to assess calceal distortion and the function of the contralateral kidney after hydronephrosis is detected by ultrasound. Barium studies are useful in identifying masses arising in the G.l tract. C.T scan and MRI are useful for grading of tumors in the abdominal cavity.

INTESTINAL DUPLICATIONS

Duplications represent mucosa-lined structures that are in continuity with the gastrointestinal tract. Although they can occur at any level in the gastrointestinal tract, these inguinal anomalies are found most commonly in the ileum within the leaves of the mesentery.

Duplications may be long and tubular, but usually are cystic masses (enterogenous cysts). In all cases, they share a common wall with the intestine. Symptoms associated with enteric duplication cysts include recurrent abdominal pain, emesis from intestinal obstruction, or hernatochezia. Such bleeding typically results from ulceration in the duplication, or in the adjacent intestine if the duplication contains ectopic gastric mucosa. On examination, a palpable mass is often identified. Children may also develop intestinal obstruction. Torsion may produce gangrene and perforation.

MESENTERIC CYSTS

Mesenteric cysts are similar to duplications in their location within the mesentery. However, they do not contain any mucosa or muscular wall. Mesenteric cysts can cause intestinal obstruction or may present as an abdominal mass.

BILIARY ATRESIA

The most important surgical cause of jaundice in the newborn period is biliary atresia. The incidence of this disease is approximately 1 in 20,000. This disease is characterized by an obliterative process of the extrahepatic bile ducts, and is associated with hepatic fibrosis.

The etiology is Unknow. The obliterative process involves the common duct, cystic duct, one or both hepatic ducts, and the gallbladder, in a variety of combinations.

Jaundice, a constant finding, is usually present at birth or shortly thereafter, but may go undetected or may be regarded as physiologic until the child is 2 or 3 weeks old. The infant demonstrates acholic, grey-appearing stools, secondary to obstructed bile flow. Infants with biliary atresia also manifest progressive failure to thrive, and if untreated, progress to develop stigmata of liver failure and portal hypertension, particularly splenomegaly and esophageal varices.

WILM'S TUMOR

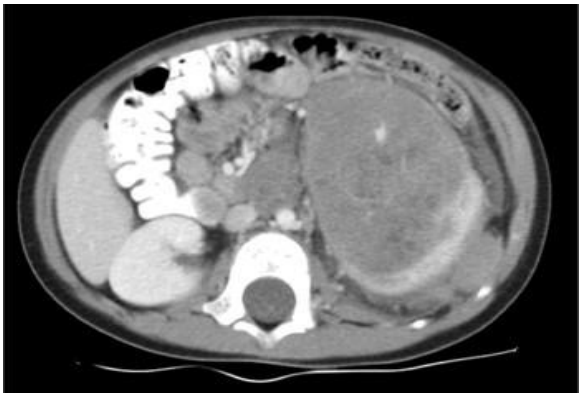
Wilms' tumor is the most common primary malignant tumor of the kidney in children and occurs in 1\10,000 live births. Pathologically, the tumor is formed of renal tissue with various degrees of differentiation, producing a mixed histological picture of epithelial cells, resembling renal tubules in 3 mesenchymal tissue. Germline mutations in primitive embryonic mesenchymal cells are probably the cause.

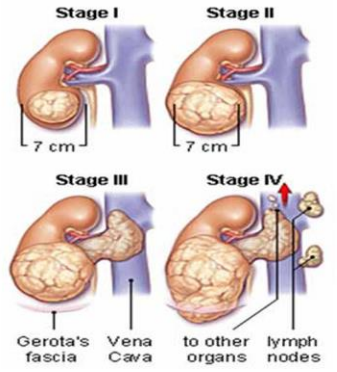
CLINICAL FEATURES

the tumor usually presents in children under the age of 3 years (60%) with symptoms as hypertension, hematuria, obstipation, weight loss. But the majority (90%) presents as an asymptomatic mass in the flank or upper abdomen. In 10% of patients the mass is bilateral. 40% have metastatic spread at presentation, but with a good prognosis.

DIAGNOSIS

Diagnosis can be achieved by using ultrasound which may also detect renal vein or inferior caval extension. CT scan may aid in provisional diagnosis by characterizing the mass, identifying the presence of metastases, and providing information on the opposite kidney.





MANAGEMENT

The goals of surgery include complete removal of the tumor. It is crucial to avoid tumor rupture or injury to contiguous organs. A sampling of regional lymph nodes should be included, and all suspicious nodes should be sampled. Typically, a transverse abdominal incision is made, and a transperitoneal approach is used. The opposite side is carefully inspected to ensure that there is no disease present. A radical nephroureterectomy is then performed with control of the renal pedicle as an initial step. If there is spread above the hepatic veins, an intrathoracic approach may be required. If bilateral disease is encountered, both lesions are biopsied, and chemotherapy is administered, followed by a nephron sparing procedure. The cure rate and prognosis depend on the stage, e.g. stage 1 (localized to the kidney) has more than 80% 3 years survival rate and more than 90% cure rate while stage 4 (heamatogenous spread) has less than 30% 3 years survival rate.

NEUROBLASTOMA

Neuroblastoma is most highly malignant and the third most common pediatric malignancy, and accounts for approximately 10% of all childhood cancers. Incidence is about 1 in 8000 live births. These tumors arise from neu neural crest cells either in the adrenal medulla or the sympathetic ganglia and show a range of malignancy from benign ganglioneuroma to malignant neuroblastoma. 75% of these tumors arise in the abdomen.

CLINICAL FEATURES

Clinical presentation depends on the site of tumor and presence of metastasis. 30% of patients presents as an abdominal mass. Symptoms related to metastasis include weight loss and pallor. Pulmonary and bone metastasis is common.

INVESTIGATIONS

Urine sample analysis may show an increase in the level of VMA (Vanillylmandelic acid) and MHMDA (m-hydroxymandelic acid). Plain abdominal films may show speckled calcification Diagnosis can be confirmed by ultrasound and CT scan (. CT scan can also be used for staging of the tumor. Bone scans or bone scintigraphy can detect bone metastasis.

MANAGEMENT

The goal of surgery is complete resection. However, this is often not possible due to the extensive locoregional spread of the tumor at the time of presentation. Under these circumstances, biopsy is performed and preoperative chemotherapy is provided based on the stage of the tumor.

After neoadjuvant treatment been administered, surgical resection is performed. The principal goal of surgery is to obtain at least a 95% resection, without compromising major structures. Abdominal tumors are approached through a transverse incision. Thoracic tumors may be approached through a posterolateral thoracotomy or through a thoracoscopic approach. These may have an intraspinal component. Post-operative radiotherapy is indicated in late stages. Prognosis is best in children presenting before 2 years of age, but mainly depends on the stage, e.g. stage 1 (localized) has 3 years survival rate of more than 90%, while a stage 4 (hematogenous spread has 3 years survival rate of less than 30%.

NEONATAL HYDRONEPHROSIS

Hydronephrosis refers to dilatation of the renal pelvis and calyces, with accompanying atrophy of the parenchyma, Fetal urinary tract abnormalities occur in up to 1% of all pregnancies, and hydronephrosis accounts for half of these. It's 5 times more common in males than in females. it can be uni- or bilateral and can be either mild moderate or severe. Evaluation and management depend on the severity and whether it is unilateral or bilateral. - Most mild and moderate cases resolve spontaneously; Routine prenatal ultrasound often identifies this. Antenatal hydronephrosis may be caused by pelviureteric junction obstruction, vesicoureteric junction obstruction or reflux, multicystic kidney, primary obstructive megaureter and posterior urethral valves. The urgency and type of investigation depends on the size of the hydronephrosis. Small unilateral hydronephrosis requires no action, whereas larger lesions require ultrasound, micturating cystography and perhaps isotope renal scans repeated at intervals to decide if surgery is needed. In bilateral severe hydronephrosis, early investigation and surgery is essential.

INGUINOSCROTAL CONDITIONS

The most common reasons for non-acute surgical referral are hernias and associated problems, abnormalities of testicular descent and foreskin problems.

EXAMINATION OF THE GROIN AND SCROTUM

Examination check list for the groin and scrotum is shown in, the table below.

Examination of the groin and scrotum
Principles • Always examine both sides • Examine the patient while lying supine and standing erect • Cough impulse is not feasible in babies and is re- placed by crying or straining • The examination involves inspection (look) and palpation (feel)
Look for • mass • Cough impulse (crying) • Redness • Sinuses or scars • Transillumination
Feel • Cough impulse (crying) • The mass,The groin, testes, epidymi, and spermatic cords assessing • tenderness • induration • consistency • weather you can get above the mass • relation to the testis (above, below, within) • weather it moves with the testis

EMBRYOLOGY

The indifferent gonad (i.e. ovary or testis) begins to develop at the fifth week of intrauterine life in the gonadal ridge. This is part of the urogenital ridge derived from intermediate mesoderm that will also form the kidney and ureter and the genital ducts in the male or the uterus and uterine tubes in the female. At the lower pole of the putative testis, a strand of mesenchyme develops into the cord-like gubernaculum (the equivalent in females is the round ligament of the uterus). At about the eighth week,

a prolongation of peritoneum known as the processes vaginalis appears along the gubernaculum (or round ligament), and extends down into the labioscrotal fold. The testis then migrates distally along the peritoneal canal. The processes vaginalis normally closes spontaneously soon after birth. Persistence causes three very common problems in boys

1. **inguinal hernia**: persistence of the processus vaginalis can allow abdominal viscera to easily herniate through the Internal inguinal ring. Inguinal hernia can occur in 3 forms, (A) bubonocele: in which the sac doesn't extend beyond the external ring (B) funicular sac: in which there is an obliterated segment that intervenes between the tunica vaginalis and the herniating sac. It may extend beyond the external inguinal ring. (C) Complete or scrotal: the mass extends beyond the external inguinal ring into the scrotum and is in communication, with the tunica vaginalis.
2. **hydrocele**: patency of the processus vaginalis can result in accumulation of fluid from the peritoneal cavity into the cord of the processus and/or the tunica vaginalis. When the tunica vaginalis is involved without involvement of the cord, the condition is termed non-communicating hydrocele, otherwise its termed communicating hydrocele. Sometimes the fluids accumulate in a loculus along the course of the cord of the processus without involving the tunica vaginalis, this is called encysted hydrocele of the cord.
3. **combined abnormalities**: this indicates the presence of an inguinal hernia and a distal hydrocele. In girls, the canal of nuck undergoes the same obliteration of the processus vaginalis in boys. Failure of the obliteration process can result in hydroceles of the canal of Nuck. These conditions all present as inguinal or scrotal swellings usually in babies and pre-school children.

HYDROCELE

Hydrocele can be either primary or secondary. primary hydrocele in which the cause reside in a

problem with obliteration of the processus vaginalis are mainly (90%) communicating.

Secondary hydrocele is most commonly non-communicating and can follow testicular trauma or torsion, epididymitis or rarely, a testicular tumour.

The mass is cystic and painless. On examination it is irreducible, with no expansile or palpable impulse after coughing, crying or straining. The most important criteria

In the clinical diagnosis of hydrocele is positive transillumination.

The presentation depends on the age of the child. Infantile hydrocele occurs secondary to accumulation of fluid in the tunica vaginalis (non-communicating) or due to patency of the processus vaginalis (communicating) and may be uni- or bilateral. They have a strong tendency to close and reabsorb spontaneously in 90% of cases.

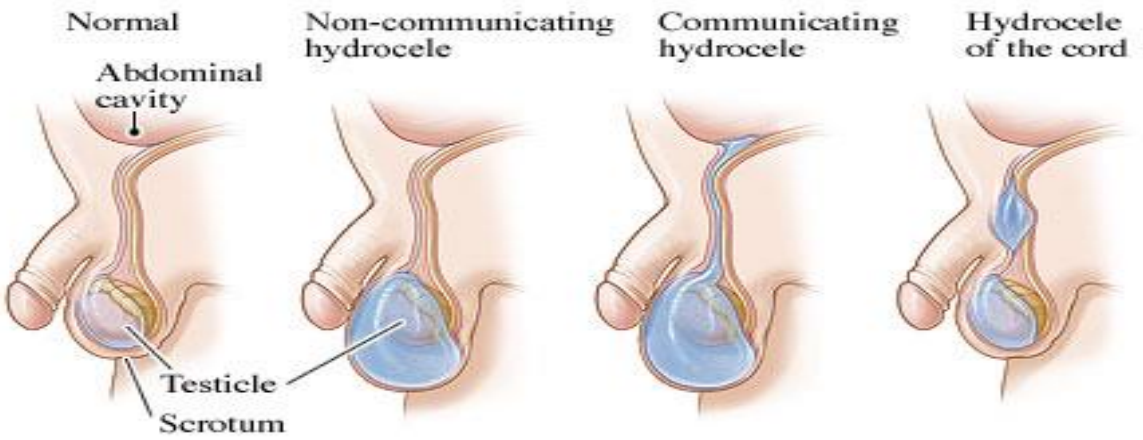
Virtually all infantile hydrocele has disappeared by the age of 1 year, but if they were tense and painful, they should undergo surgery. hydrocele in older children are most commonly communicating and have diurnal variation in size. They rarely disappear spontaneously and may require surgery.

INGUINAL HERNIAS

inguinal hernias in children arise because the processus vaginalis fails to close after testicular descent; they are therefore true congenital abnormalities. Anatomically, they are much the same as indirect inguinal hernias in adults except that there is rarely a substantial abdominal wall defect, contents are either loops of small bowel omentum, or ovary or fallopian tube in girls. The incidence in infants ranges from 1 to 2% (1/50-100 live births) with a male preponderance of 4:1; 98% of these hernias are indirect. The incidence peaks at the age of 1-3 months. The risk of incarceration increases in the 1st 6-12 months of life. 60% of inguial hernias presents on the right side, 30% on the left and 10% are bilateral.

DIAGNOSIS

A hernia usually presents as a lump at the external inguinal ring that appears when the child cries or strains at stool but reduces spontaneously in between. Examination is the key for diagnosis, which is also confirmed by history; positive expansile and palpable impulse on straining, coughing or crying. Sometimes there is no obvious hernial mass on examination inspite of the presentation; this is due to spontaneous reduction of the mass. In this case sensation of rubbing two pieces of peritoneum ('silk glove sign') indicates the patency of the processus vaginalis. Differential diagnosis for inguinal hernias in children is shown in the table below.



Differential diagnosis for inguinal hernias in children

- Hydrocele
- Retractile testes
- Undescended testes
- Inguinal lymphadenopathy

The description incarcerated means a hernia has become acutely irreducible whereas the term strangulated implies there is also impairment of the blood supply to the hernia contents. Strangulation can follow incarceration of a hernia but luckily is uncommon in young children. When a hernia incarcerates it becomes painful, tender and irreducible. Classically, a mother discovers a firm lump in the groin of her crying child. He may have vomited once or twice but the diagnosis is usually made before intestinal obstruction becomes established. On examination, the child is usually well. There is an obvious, irreducible lump in the groin which may extend into the scrotum. If the bowel becomes obstructed, fluid accumulates in the bowel and vomiting follows, causing fluid depletion and electrolyte disturbances. The blood supply of the incarcerated segment of intestine may become obstructed causing bowel infarction. Pressure on the spermatic cord may cause vascular obstruction to the testis, and rapid treatment is needed to avoid testicular infarction and irreversible damage. In girls, incarceration of the ovary may result in strangulation. Differential diagnosis for strangulated hernias is shown in the table below.

Differential diagnosis for strangulated inguinal hernias

- Encysted hydrocele of the cord
- Torsion of an undescended testes
- Torsion of fully descended testis (testis redux)
- Lymph adenitis

MANAGEMENT

Asymptomatic inguinal hernias in children should be electively repaired without delay to prevent acute obstruction. Incarcerated and strangulated hernia can be reduced manually ('TAXIS') this requires giving the child analgesia (opiates) or to distract the child with a bottle of feed.

An attempt at reduction includes surrounding the external inguinal ring with the fingers of one hand loosely, while gently pushing the fundus of the hernia inside with the fingers of the other hand. Reduction en mass should be avoided. If there is successful reduction, the operation should be performed within 24-48 hours until the edematous sac regresses. If the reduction is unsuccessful, the patient is admitted to the operation immediately.

The standard operation is inguinal herniotomy and this is one of the most common general paediatric surgical procedures. In babies and children, the procedure involves isolating the peritoneal sac from the cord (or round ligament), ligating it at the external ring and removing it (inguinal herniotomy). Complications of surgery include bleeding, infection, injury to the cord structures, iatrogenic cryptorchidism and recurrence of hernia.

NORMAL DESCENT OF THE TESTES

In the seventh and eighth months of intrauterine life, the testicle descends along the inguinal canal into the upper scrotum, and with its progress the processus vaginalis is formed and pulled along with the migrating testicle. At birth, approximately 95% of infants have the testicle normally positioned in the scrotum. The normal mechanism of descent is not fully understood but appears to occur in two phases. Migration from the gonadal ridge to the internal inguinal ring depends on shortening of the gubernaculum, whilst descent from the internal ring to the scrotum is driven by circulating androgens. Arrest of descend along this pathway may result in what is called 'undescended testes'.

EMPTY SCROTUM

Empty scrotum means the absence of a testicle inside the scrotum, it can either be unilateral or bilateral. Causes of an empty scrotum are summarized in the table below. During the examination of a patient with an empty scrotum, you should always look for an associated ectopic testis, or the presence of a testis at the superficial inguinal pouch.

You should also bear in mind the presence of a retractile testis, which can be reduced back to the scrotum. Assessment of associated hernia, general development of the baby, and other congenital anomalies is of value.

conditions associated with empty scrotum

Palpable testis

- Retractile testes
- Ectopic testis

- Undescended testis (superficial inguinal pouch)

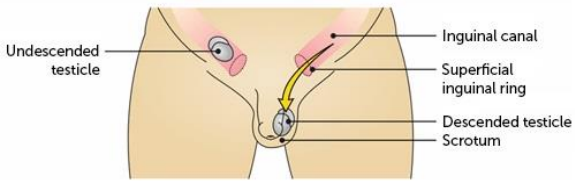
Impalpable testis

- Anorchia (absent testis)
- Dysplastic testis (small and very abnormal testis)
- Undescended testis (intra-abdominal)

UNDESCENDED TESTES

The term undescended testis (cryptorchidism; crypto=hidden) refers to the interruption of the normal descent of the testis into the scrotum. The testicle may reside in the retroperineum, in the internal inguinal ring, in the inguinal canal, or even at the external ring (the superficial inguinal pouch).

A distinction should be made between the undescended testicle and the ectopic testily described later. 90% of cases of Undescended testis are found unilateral, 70% of those are found in the right side. There is high incidence among premature infants (25%) comparing to those of full-term (7%=2% at birth 5% at 1 year of age).



DIAGNOSIS

For diagnosis, the child should be examined in the supine position, where visual inspection may reveal an empty scrotum. A unilateral undescended testicle usually can be palpated in the inguinal canal or in the upper scrotum. The testicle is relatively immobile and has short cord. Ultrasound, C.T scan, MRI and laparoscopy are used to Iocate retroperitoneal testis.

COMPLICATIONS

1. Torsion-incompletely descended tastes are abnormally mobile. Torsion of the testis, which is actually torsion of the spermatic cord, causes strangulation of the testicular blood supply, resulting in necrosis and later atrophy.
2. Trauma palpable undescended testis are liable to direct injury.
3. Subfertility-undescended testes exhibit incomplete maturation of the seminiferous tubules, leading to the production of sperm abnormal in quantity, form or motility. 90% of men have achieved fertility if their orchiopexy had been performed before 2 years of age.
4. Neoplasia-maldescent is associated with up to 5 times the normal risk of later testicular malignancy (although the risk is still small).

MANAGMENT

orchiopexy means mobilization of the undescended testis into the scrotum. its best performed at 6-24 months of age. This can also be useful to repair any associated hernia (10-20% of undescended testes cases are associated with inguinal hernia). Treatment may improve future fertility, place the testis in an easy palpable position/ and afford cosmetic and psychological benefit.

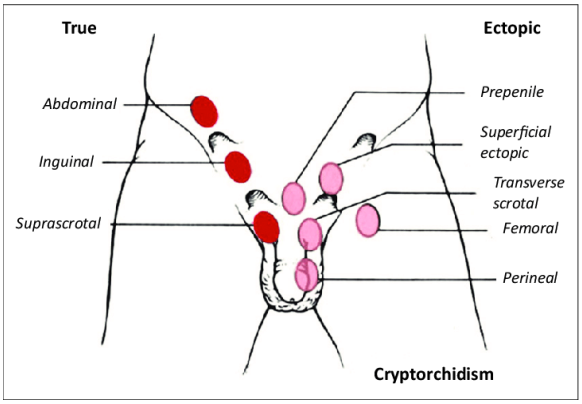
ECTOPIC TESTES

Ectopic testes accounts for 10% of empty scrotum associated conditions.

By definition, an ectopic testis is one that has passed through the external ring in the normal pathway, and then has come to rest in an abnormal location secondary to the presence of an abnormal pouch along the pathway of testicular descent.

This is mainly the superficial inguinal pouch (a facial barrier that prevents entry of the testis into the scrotum). When this pouch extends to the perineum (perineal), to the base of the penis (suprapubic), or to the contralateral scrotum (contralateral), an ectopic testis may form Other possible sites include the femoral triangle at the medial aspect of the thigh.

The testis is usually normal in size and the cord is of good length so that there is no vascular compromise



RETRACTILE TESTES

These are normally descended testes, but retract into the upper scrotum or groin due to hyperactive cremasteric response. They can be manipulated back into the scrotum, where it should lie' without tension or restriction.

With age the testes spontaneously reside for longer periods in the scrotal pouch and thus no further intervention is required, however, re-examination should be done a few months later of the 1st visit so that the doctor can confirm the condition.

ACUTE SCROTUM

Acute scrotum mean's the acute presentation of scrotal pain in a child due to an underlying cause. The child usually refuses to walk or walk with an abnormal gait abducting the thigh.

Causes of acute scrotum

- strangulated inguinal hernia.
- torsion of the testis.
- torsion of one of the scrotal appendages (hydatid
- of morgagni)
- epididymis-orchitis
- idiopathic scrotal edema
- inguinal lymph adenitis

TESTICULAR TORSION

Testicular torsion is an acute presentation that occurs due to twist of the testis upon the spermatic cord, this twist may involve and obstruct the venous plexus surrounding the cord, leading to edema in the cord and the testis, which may end in arterial obstruction and gonadal necrosis. Testicular torsion is the commonest cause of acute scrotum among all age groups (incidence 1\4000), but the incidence peaks in late childhood and early adolescence.

Predisposing abnormality (e.g. undescended testis) is almost always present.

CLINICAL FEATURES

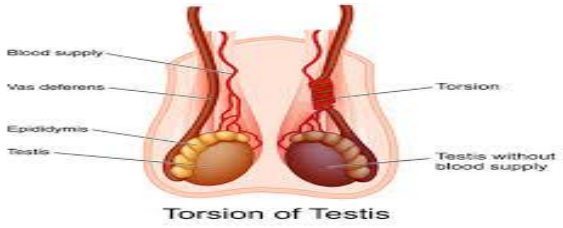
Presentation is typical of that of acute scrotum, associated with lower abdominal pain, and acute unilateral scrotal swelling.

On examination, the testis is elevated and palpation of the spermatic cord-can reveals a thickening and irregular surface.

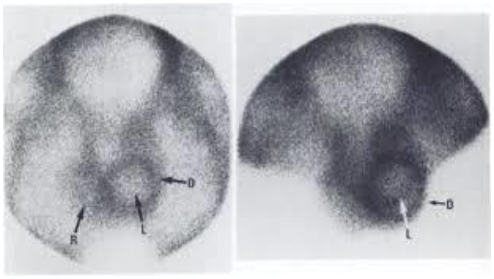
INVESTIGATIONS

Ultrasound with Doppler blood flow monitoring may be helpful in cases where the clinical picture makes torsion unlikely as it is reassuring to know there is good blood flow to the testis

Radioisotope testicular scan may also be of use in the diagnosis of testicular torsion. It shows a 'halo sign'



Torsion of Testis



MANAGEMENT

When a diagnosis of torsion cannot be excluded, the only safe option is to carry out urgent surgical exploration of the testis. If torsion is present and if the testis is viable, it should be untwisted and fixed to prevent re-torsion. Delays should be avoided; maximal success rate is obtained when surgery is performed within 10 hours of onset. A non-viable testis should be removed and the contralateral testis should also be fixed at the same time since the attachment abnormality is usually bilateral.

GASTRO-INTESTINAL BLEEDING IN CHILDREN

Gastrointestinal bleeding in infants and children is a fairly common problem, accounting for 10%-20% of referrals to pediatric clinics. Clinically, there are many types of gastrointestinal bleeding; these are anorectal bleeding, **hemorrhage with other clinical features, hemorrhage in the neonatal period arid massive hematemesis or melena.** The diagnosis of the underlying cause always starts by classifying the presentation under one of the previously mentioned types and depends on the age of the patient, the type and quantity of bleeding, and the associated symptoms. Gl bleeding can also be classified as being small (<20 ml) or large (>200 ml).

ANO-RECTAL BLEEDING

Anorectal bleeding implies passage of small volume of blood with stool, most commonly from the anal canal, or less commonly from the rectum or colon. Sometimes the cause is bleeding from the stomach, duodenum, small bowel or even the esophagus due to small transit time. On history you should always determine whether the blood was in streaks or mixed with stool. Examination with inspection and digital examination is used to evaluate the diagnosis. Causes of anorectal bleeding are shown in the table below.

Causes of anorectal bleeding in children
Common causes • Anal fissure • Juvenile polyp • Rectal prolapse • Meckel's diverticulum
Rare causes • Adenomatous Familial polyposis (FAP) • Haemangiomas • Ulcerative colitis • Multiple polyposis • Malignancy

According to the nature of the blood, anorectal bleeding can be either one of the following

1. **Fresh blood**; the pathology is most certainly between the anal margin and the lower sigmoid colon.
2. Blood clots or Cherry red blood; this indicates a pathology in the colon.
3. meleana or Altered blood; black tarry greasy offensive stool. Occurs due to hemorrhage from the G.l tract between the esophagus and the small bowel.
4. Occult bleeding, bleeding that occurs somewhere along the whole gastrointestinal tract but is not grossly visible on stool. it can be detected with a haemoccult stool test. It's usually associated with recurrent anemia, and most certainly indicates malignancy.

ANAL FISSURES

Anal fissures are the most common cause of ano rectal fresh blood in children of 1-10 years of age. it's almost confined to infants and toddlers. An anal fissure is characterized by a linear anal ulcer, often with a hypertrophied internal sphincter visible in the base, affecting the anal canal below the dentate line from the anal transition zone to the anal verge. There is often little in the way of granulation tissue in the ulcer base. Owing to failed attempts at healing, there may be a tag of skin (caused by edema and hypertrophic response to inflammation) at the lowermost extent of the fissure, known as a 'sentinel pile'. At the proximal extent of the fissure there may be a hypertrophied anal papilla.

Most fissures are idiopathic, idiopathic Anal fissures in children are extremely confined to the posterior midline of the anal canal. Fissures elsewhere occur in ulcerative colitis, crohn's disease and in immunocompromised patients. Fissures associated with these conditions are usually recurrent in course. Anal fissures are either acute or chronic. Acute fissures may settle spontaneously, but chronic anal fissure is defined as an ulcer that has been present for at least 6 weeks.

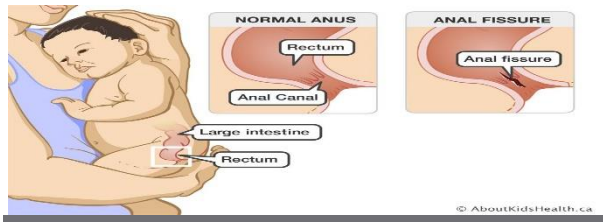
CLINICAL FEATURES

Children with anal fissures may presents with a painful defecation, where by the mother say's that her child cries on passing motion. The mother or the child may notice the presence of few streaks or drops of bright blood on the surface of the stool

(ano-rectal bleeding). The constipation is caused by spasm of the internal sphincter which is caused by pain. Accumulation of stool inside the rectum urges the child to defecate, however this may aggravate pain and thus increases sphincter spasm and causes more constipation.

MANAGEMENT

After ruling out IBD's and immunodeficiency as a cause of the fissure, treatment is carried through by interfering with the viscous circle of spasm, pain, and constipation. Acute fissures are treated by stool softeners (decreases pain and constipation), sitz path (reliefs the spasm, and promotes healing by improving the circulation) and gentle dilatation (overcomes anal sphincter spasm). Chronic fissure is treated by botox (botulinum toxin reliefs anal spasm). Surgery is indicated where medical treatment fails. Lateral internal sphincterotomy is the most common operation performed and involves controlled division of the lower half of the internal sphincter at the lateral position.



POLYPS AS A CAUSE OF ANO-RECTAL BLEEDING

There are many types of polyps, only the commonly associated with ano-rectal bleeding are discussed here. These include juvenile polyps, Peutz-Jeghers Syndrome, adenomatous polyp's multiple polyposis syndrome and familial adenomatous polyposis.

JUVENILE POLYPOSIS SYNDROME (JPS)

This rare disease occurs in children of ages less than 10 years. The formed polyps are once thought to be the result of cyst formation secondary to obstruction of intestinal crypts, however, it's now agreed that these are hamartomas in nature. Polyps ulceration may lead to bleeding and the occurrence of blood with stool. In 93% of cases the bleeding is bright (fresh) and intermittent, and patients describes the presence of blood streaked feces at the end of defecation.

PEUTZ-JEGHERS SYNDROME

Peutz-Jeghers syndrome is an autosomal dominant inherited disorder that is rare but is an 'easy to diagnose' cause of ano-rectal bleeding. The clinical manifestations include gastrointestinal polyps and melanin pigmentation at mucocutaneous junctions and occasionally on the dorsum of the hands and feet. Polyps occur most commonly in the jejunum. Most cases present in childhood or adolescence. There are usually dark brown or bluish spots on the lips and inside the mouth. The face, palms, soles, arms and perianal region can also be affected The usual presentation is with abdominal pain or obstruction due to intussusception of a polyp, but rectal bleeding following ulceration and iron deficiency anaemia are also common.

OTHER CAUSES OF ANORECTAL BLEEDING

include multiple polyposis syndrome which by definition means the presence of more than 5 polyps without any family history of polyposis syndrome, or the presence of any number of polyps with a positive family history of polyposis syndrome. A common variant of this is familial adenomatous polyposis, Is associated with a high risk of malignancy during adult life.

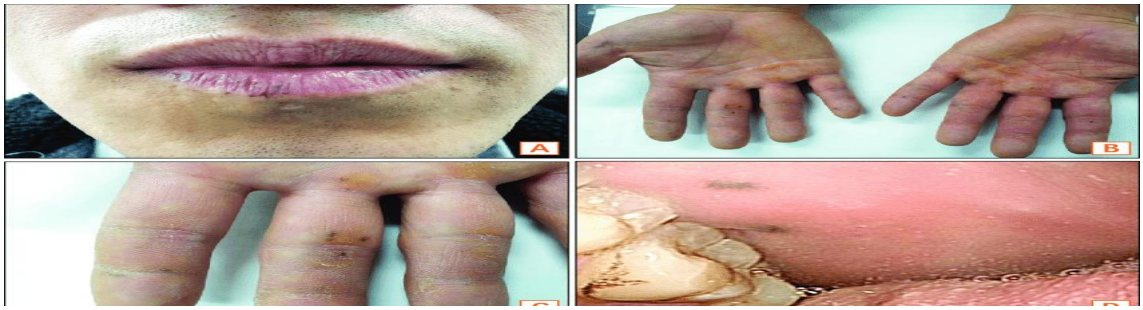


Figure 191 typical presentation of PJS

RECTAL PROLAPSE

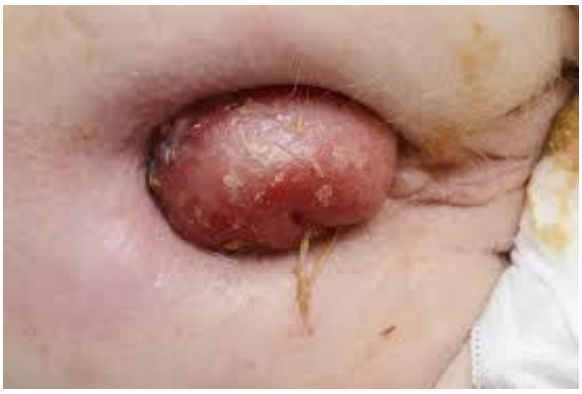
ETIOLOGY

Rectal prolapse means passage of part of the rectal wall through the anal canal, it's more common in children and is predisposed by prolonged straining during defecation in a child with constipation. It's commonly seen in hyper- kinetic children, whom because of the act of ill training prolong the attempts to defecate producing excessive straining without constipation, it's also seen in frequent diarrhea as a part of malabsorption syndrome, cystic fibrosis, and celiac disease. In cystic fibrosis it's coughing, which increases intra-abdominal pressure,' is the cause rather than diarrhea.' In children who are malnourished and marasmic, loss of fat supporting the rectal wail can cause rectal prolapse. Other causes of rectal prolapse include paralysis of the sphincter muscles in myelomeningocele and sacral agenesis, and divarication of the puborectalis muscle secondary to separation of the symphysis pubis.

CLINICAL FEATURES

Rectal prolapse presents in children of ages between 1-3 years with a history of a painless prolapsing mass from the anus during defecation, which reduces spontaneously. Some presentations state that they will have to reduce it manually. Constipation is a constant feature in rectal prolapse and is always suspected as the cause. Rectal prolapse may also present as ano-rectal bleeding. Digital examination may reveal a decreased sphincter tone and an inside bulge that have a palpable attachment (bn the contrary of intussusceptions). Differential diagnosis for rectal prolapse is shown in the table below.

Differential diagnosis for rectal prolapse
• **Rectal polyps**
• **Intussusception External**
• **hemorrhoids**



MANAGEMENT

Management of rectal prolapse is presented by 6 S's:

1. Stool; use stool softeners to treat the constipation or treat malabsorption.
2. Seat; squatting position may aid in stretching the anal sphincters
3. Sedation; small doses of any sedative drug is helpful in training hyperkinetic children to defecate properly.
4. Strapping; a transverse strap applied to the buttocks is used to prevent prolapse from the anus.
5. Seclerosing; injection of 5 ml 5% phenol in almond oil or hypertonic glucose, may promote fibrosis and thus can strengthen the rectal wall.
6. Suture; if the previous methods don't work, surgery is indicated, and it aims to stabilize the rectum by fixing it to the sacral wall using sutures (chromic cat gut). (thiersch procedure)

MECKEL'S DIVERTICULUM

PATHOLOGY

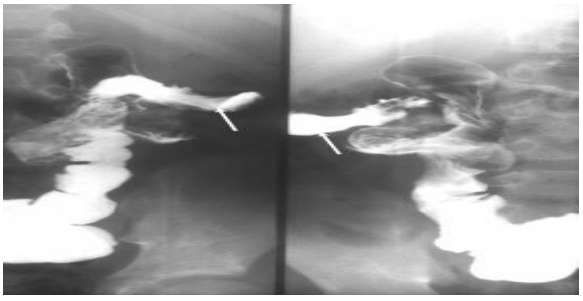
Meckel's diverticulum is a remnant of a portion of the embryonic omphalomesenteric (vitelline) duct. Failure of' regression of the vitelline duct may result in various Anomalies illustrated in figure 4. It is located on the antimesenteric border of the ileum, usually within 2 feet of the ileocecal valve. Meckel's diverticulum is about 2 inches in length with approximately 2 cm in diameter. It usually contains either one of 2 types of heterotopic tissue, pancreatic or gastric. The latter is more liable to ulcerate and bleed. It affects 2% of the population with a male to female ratio of about 2:1 and generally is symp¬tomatic before the age of 2. See the table below.

the rule of 2 in meckel's diverticulum
2 feet from the ileocecal valve
2 inches in length
2 cm in diameter.
2 heterotopic tissue
2% of the population
2:1 male to female ratio
Symptomatic before the age of 2

Congenital anomalies commonly associated with meckel's diverticulum are shown in the table below

Congenital anomalies commonly associated with meckel's diverticulum
Cardiac anomalies
Congenital diaphragmatic hernia
Duodenal atresia
Esophageal atresia
Imperforate anus
Gastroschisis Omphalocele
Malrotation
Hirschsprung's disease
Down's syndrome

Perforation of a Meckel's diverticulum may occur if the outpouching becomes impacted with food, leading to distention and necrosis. Occasionally, bands of tissue ex- tend from the Meckel's diverticulum to the anterior abdominal wall, and these may represent starting points around which volvulus may develop. Sometimes meckel's diverticulum may become a pathological leading point and cause intussusceptions. Similarly, to duplications, ectopic gastric mucosa may produce ileal ulcerations that bleed and lead to the passage of maroon-colored stools.



CLINICAL FEATURES

The presentation of meckel's diverticulum depends on the age. Symptoms common to all age groups include abdominal pain, nausea, vomiting, ano-rectal bleeding and abdominal distention. Newborns commonly present with symptoms of intestinal obstruction due to an under lying volvulus or intussusception. In infants and young children, the presentation is mainly a painless lower gastrointestinal bleeding. In older children meckel's diverticulum may undergo inflammation and be confused a appendicitis. Thus, one of the differential diagnoses c meckel's diverticulitis is acute appendicitis. Diagnosis may be made by technetium pertechnetate scans when the patient presents with bleeding.

TREATMENT

Treatment is surgical. If the base is narrow and there is no mass present in the lumen of the diverticulum, a wedge resection of the diverticulum (diverticulectomy) with transverse closure of the ileum can be performed.

When a mass of ectopic tissue is palpable, if the base is wide, or when there is inflammation, it is preferable to perform a resection of the involved bowel and end-to-end ileoileostomy. Incidental finding of meckel's diverticulum is an indication for resection, especially in children less than 8 years of age and in those containing heterotopic tissues.

HEMORRHAGE ACCOMPANIED BY OTHER CLINICAL FEATURES

Causes of hemorrhage accompanied by other clinical feature which are likely to be the presentation. They areshown in the table below. This form is managed according to the underlying condition.

causes of H.A.P.O.F	
cause	Associated symptom
• Intussusception	1. lntestinal obstruction 2. Redacurrent jelly
• Ulcerative colitis	1. Bloody diarrhea 2. Anaemia and weight loss

• Gastroenteritis	1. Bloody diarrhea 2. Dehydration
• FAP	1. Ano-rectal bleeding 2. Anemia 3. Mucocutaneos pig-mentation ;

HEMORRHAGE IN THE NEONATAL PERIOD

This encompasses acute conditions associated with loss of blood from the gastrointestinal tract in the newly born babies. The conditions are shown in the table below.

Conditions associated with hemorrhage in the neonatal period	
cause	mechanism
Hypoprothrombinaemia and thrombocytopenia	Increased tendency to bleed due to defective coagulation
Infantile hypertrophic pyloric stenosis	Development of gastritis (coffee ground blood)
Gastro-esqphageal reflux dis ease	Development of peptic esophagitis (coffee ground blood)
Volvulus neonatorum	Strangulation
Necrotizing enterocolitis	Ulceration and hemorrhage

Ingestion of blood from the birth canal during child birth or from a cracked nipple during feeding is sometimes confused as being hemorrhage from the gastrointestinal tract.

MASSIVE HAEMATEMESIS OR MELAENA

This means the presence of heamatamesis or melaena either together or separately, in large amounts. Causes are shown in the table below. heamatamesis means vomiting or regurgitation of blood from the mouth.

Causes of massive melaena or heamatamesis

Esophageal varices in portal hypertension
Meckel's ulceration

Necrotizing enterocolitis

Stress ulcerations in

- Burns (curling's ulcer)
- Intracranial injury (Cushing's ulcer)
- Sever toxic infection

NECK MASSES

Neck masses are found either in the midline or lateral compartments. See the below table.

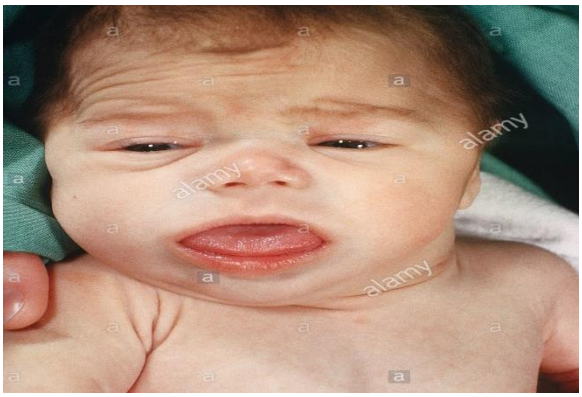
Neck masses
Anterior midline • Thyroid associated lumps • Ectopic thyroid • Thyroglossal cyst • Parathyroid • Dermoid cysts ...
Anterior triangle • Branchial cyst • Carotid aneurysm
Posterior triangle • Lymph nodes. • Innominate or subclavian aneurysms • Lymph nodes (Virchow's nodes)
Submandibular area • Submandibular salivary glands
Parotid area • Parotid gland
Others • Cystic hygroma • Extra-angular dermoid

The diagnosis of neck masses is mainly determined by examination, thus other than knowing the site of the presenting mass, its important for the surgeon to identify other characters of the mass. The characteristics that are usually required are shown in the table below.

charecteristics of a lump
Look 7 S's Site, Size (in two directions), Shape, Surface (presence of a scar or sinus), Skin color (at rest and with pressure), Surrounding regional lymph nodes, Shine a light (transluscency)
Feel 5 T's Tenderness, Tempreture, Texture, To press on it, To feel its edge, To get above it.
Move 3 D's Does the lump move spontaneously? Does the skin move over the lump? Does the lump move over the underlying structures?
Listen for bruit

NEONATAL GOITER (CRETINISM)

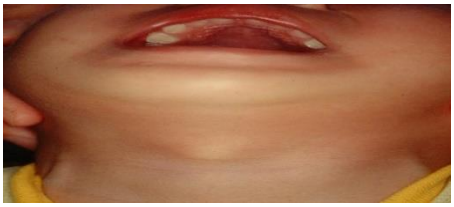
This means diffuse enlargement of the thyroid gland in the newly born child, that if left untreated may lead to hypothyroidism and cause a syndrome called cretinism. The child may have symptoms of respiratory distress with or without obstruction of the upper respiratory tracts. the cause is probably the ingestion of iodine containing compounds like thiouracil. When iodine is removed, the gland may start to regress in size. Compensatory thyroxine may be required. In cases of respiratory distress and upper respiratory obstruction, partial thyroidectomy is required and in emergency presentations tracheostomy is required.



THYROGLOSSAL DUCT CYST

PATHOLOGY

The thyroid gland buds off the foregut diverticulum at the base of the tongue in the region of the future foramen cecum at 3 weeks of embryonic life. As the fetal neck develops, the thyroid tissue becomes more anterior and caudal until it rests in its normal position. The "descent" of the thyroid is intimately connected with the development of the hyoid bone. Residual thyroid tissue left behind in the migration may persist and subsequently present in the midline of the neck as a thyroglossal duct cyst. The thyroglossal cyst may occur at any site along the line of descent, but the majority is found at the level of the thyrohyoid membrane under the deep cervical fascia.



CLINICAL FEATURES

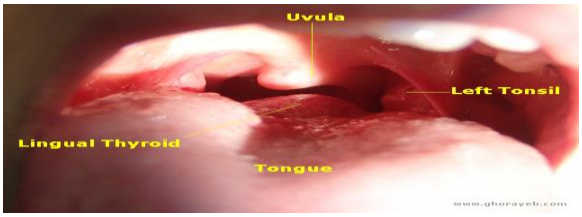
a mass is most commonly appreciated in the 2- to 4-year-old child when the baby fat disappears and Irregularities in the neck become more readily apparent. Occasionally they may appear after an upper respiratory Infection (as they became enlarged and painful). Usually the cyst is small, round and encountered in the midline at or below the level of the hyoid bone. On examination, the mass is painless, fluctuant, and movable, that moves up and down with swallowing or with protrusion of the tongue. If the duct retains its connection with the pharynx, infection may occur, in which the mass become reddened and drainage of the mucus may sometimes occur under the skin. Differential diagnosis of thyroglossal cyst is shown in the table below.

Differential diagnosis for thyroglossal cyst

- **Dermoid cysts**
- **Thyroid neoplasm (teratoma) Sebaceous cyst**
- **Ectopic thyroid**
- **Lipoma**
- **Submental lymph nodes**

DIAGNOSIS

Diagnosis is usually made by examination, but if there Is any question regarding the diagnosis or if the thyroid gland cannot be palpated in its normal anatomic position, it is advisable to obtain a nuclear scan to confirm the presence of a normal thyroid gland.



MANAGEMENT

Infected cysts should be treated with antibiotics. If the cyst presents with an abscess, treatment should consist of drainage and antibiotics. Following resolution of the inflammation, resection of the cyst in continuity with the central portion of the hyoid bone and the tract connecting to the pharynx, in addition to ligation at the foramen cecum **(the Sistrunk operation)** is curative. Lesser operations result in unacceptably high recurrence rates, especially those which does not remove the hyoid bone.

LINGUAL THYROID

A lingual thyroid represents a failure of the median thyroid diverticulum to descend normally and may be the only thyroid tissue present. Its four times more common in females. They are found at the posterior aspect of the tongue and ranges between less than a centimeter to more than 4 cm in size. Large glands can cause obstructive symptoms such as choking, dysphagia, airway obstruction, and hemorrhage. Diagnosis can be achieved through examination alone. Provisional diagnosis using CT scans and radioactive iodine scan is of use before the surgery.

ECTOPIC THYROID

Normal thyroid tissue may be found anywhere in the central neck compartment, including the esophagus, trachea, and anterior mediastinum. Thyroid tissue has been observed adjacent to the aortic arch, in the aortopulmonary window, within the upper pericardium, and in the interventricular septum. Often, "tongues" of thyroid tissue are seen to

extend off the inferior poles of the gland and are particularly apparent in large goiters.

BRANCHIAL CYSTS AND FISTULAS

Paired branchial clefts and arches develop early in the fourth gestational week. The first cleft and the first, second, third, and fourth pouches give rise to adult organs.

The embryologic communication between the pharynx and the external surface may persist as a fistula (branchial fistula). A fistula is seen most commonly with the second branchial cleft, which normally disappears, and' extends from the anterior border of the sternocleidomastoid muscle superiorly, inward through the bifurcation of the carotid artery', and enters the posterolateral pharynx just below the tonsillar fossa. Branchial cysts may form at a remnant of the branchial cleft. The cyst is located at the site of the remnant; at the carotid triangle. approximately 10% are bilateral and contains thick yellowish creamy fluid (cholesterol crystals). It may become Infected. The treatment's surgical, and complete removal of the cyst and tract is necessary for cure. There is recurrence rate of less than 7% for branchial cysts.

CYSTIC HYGROMA

Cystic hygroma (lymphangioma) occurs as a result of sequestration or obstruction of developing lymph vessels. Occasionally unilocular cysts occur, but more often there are multiple cysts. The cysts are fluctuant and are lined by endothelium and filled with lymph (crystal clear), but some may contain cavernous haemangiomatous vessels. Thus, some may consider these to be hamartomas of the lymphatic tissue.

CLINICAL FEATURES

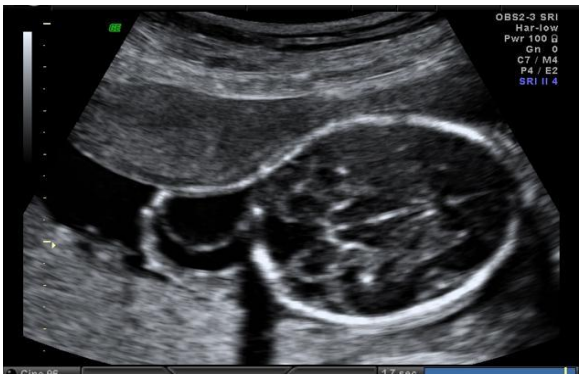
The presentation depends on the site and size; Sites possible for cystic hygroma and their frequency are shown in the table below.

Site where cystic hygroma might be
• **In the neck 70%** • **Axillary region 20%** • **Superior mediastinum Mesentery Retroperitonuem Pelvis and groin**

Cystic hygroma in the neck may.be found at the floor of the mouth or the peripharyngeal area and may endanger breathing airways or cause dysphagia. Cavernous haemangiomas containing cystic hygroma may bleed and cause rapid enlargement which may be present as an emergency. Infection within the cysts may occur and may also cause enlargement.

DIAGNOSIS

Diagnosis can be made prenatally using ultrasound, pert- natal diagnosis can be done by ultrasound, CT scan and MRI.



TREATMENT

Surgical excision is the treatment of choice for cystic hygromas. Total removal may. not be possible because of the extent of the hygroma and its proximity to, and intimate relationship with, adjacent nerves, muscles, and blood vessels.

لجنة الطب والجراحة

ولأن كل عمل خير يقف وراءه
أعين سهرت لنشره تتوجه
لجنة الطب والجراحة

بالشكر الجزيل لكل من ساهم في تحقيق هذا الإنجاز
ونخصُّ في شكرنا الفريق الأكاديمي اللي قدم هذا العمل على مصلحته و راحته.

لو أنني أنشدت ألف قصـــيدة
لوجدتها في حقّكم لا لن تفي

* * *

سيروا الى العلياء واقتادوا المنى
وامضوا إلى الإبداعِ دون توقفِ