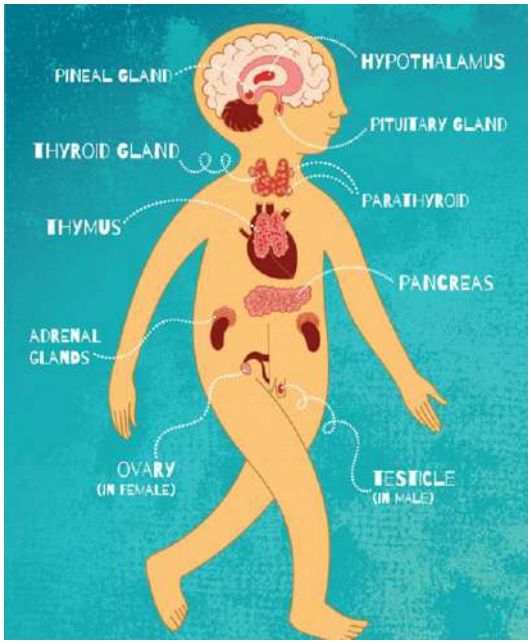
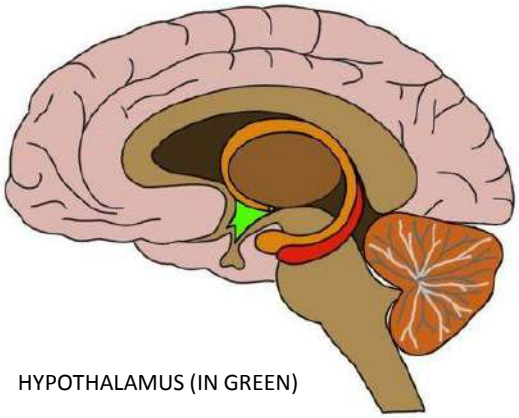
Endocrinology / pituitary gland

Introduction

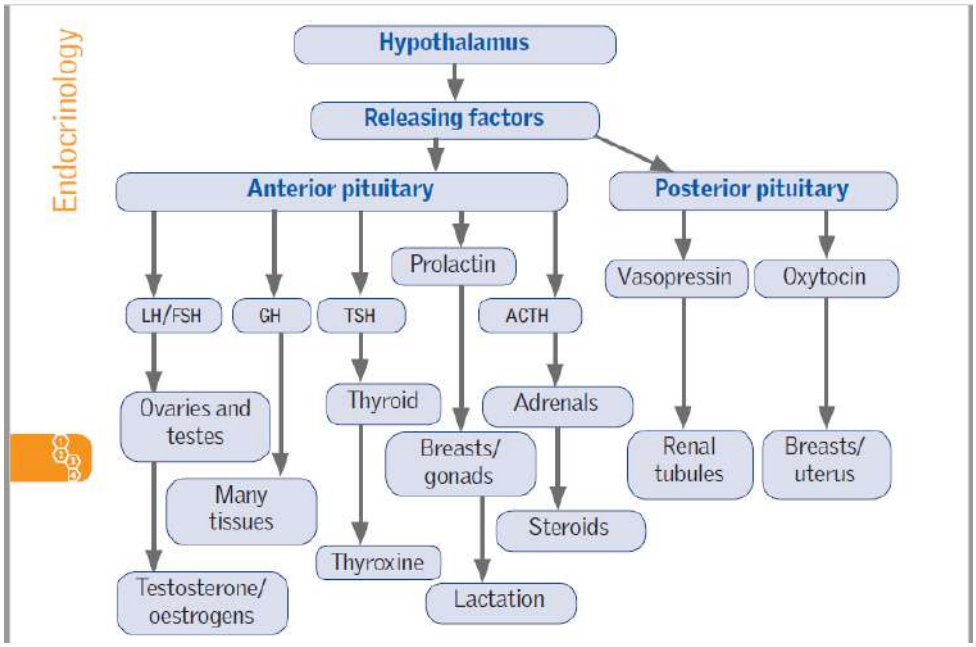
Endocrine and metabolic disorders are relevant to anesthetics because some disorders present systemic manifestations and require especial management modifications.

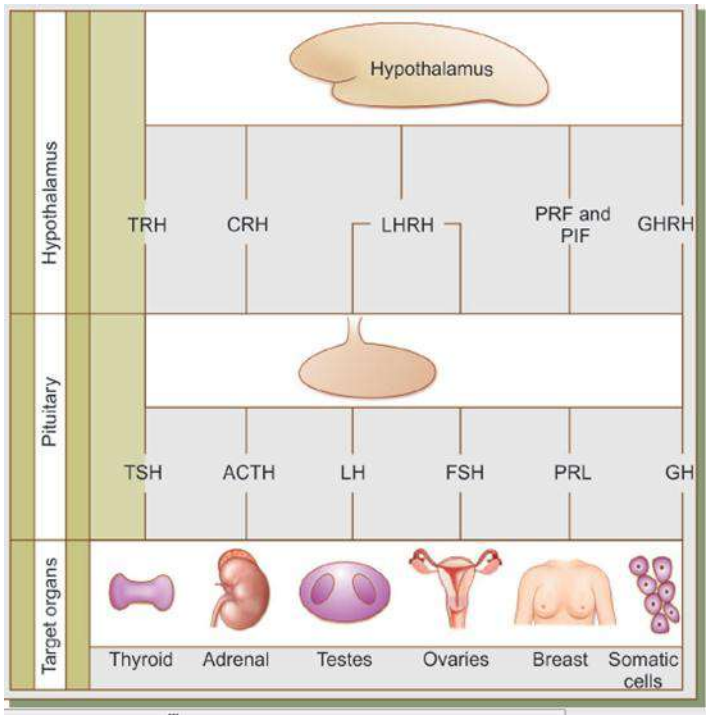




HYPOTHALAMUS (IN GREEN)

The hormones are produced at three levels, i.e. hypothalamus (releasing hormones), pituitary (trophic or stimulating hormones) and target organ such as thyroid, parathyroid, adrenals, gonads etc. (active hormones or prohormones) Figure 1.





The hormones released by the hypothalamus include

Vasopressin, or antidiuretic hormone (ADH): causes water reabsorption in the kidneys, maintains blood pressure

Oxytocin: "the cuddle/love hormone," regulates social interaction and sexual reproduction

Growth hormone (GH): in children, acts on several parts of the body to promote growth; in adults, maintains body structure, metabolism, and maintenance of blood glucose levels

Prolactin: plays a role in lactation, maintenance of the reproductive system, behavior, and regulation of the immune system

Corticotrophin-releasing hormone (CRH): controls the body's response to stress

Growth hormone-releasing hormone (GHRH): (no, there wasn't a typo – this is a real, redundant-sounding hormone) can you guess what this does? That's right! This hormone stimulates the release of the growth hormone

Somatostatin: this hormone inhibits the secretion of pancreatic and gastrointestinal hormones

Gonadotrophin-releasing hormone (GnRH): this is released from the nerve cells in the brain, controlling the production of luteinizing hormone and follicle-stimulating hormone

Thyrotrophin-releasing hormone: regulates the production and secretion of thyroid-stimulating hormone and prolactin

Common Symptoms in Endocrine Diseases

In endocrine diseases, symptoms of over- or under-secretion of hormones are encountered. Some general symptoms include weight loss or gain, excessive hair loss or growth, skin pigmentation, fertility or menstrual problems, dwarfism, slow mental activity, disturbances in the heart rate and rhythm, gigantism, and chronic fatigue.

Common Investigations in Endocrine Diseases

Investigations into endocrine diseases include thyroid function tests, isotope scanning, fine needle cytology, plain x-rays of the appropriate anatomic region, ultrasound, CT scan, MRI, biochemical investigations such as estimation of levels of hormones, blood glucose, serum cortisol levels, urea and electrolytes, vitamin D levels, serum calcium and phosphate levels.

Diseases of the Pituitary Gland

- **Hypopituitarism**

Definition/description: A condition characterized by the deficiency of anterior or posterior pituitary hormones is called hypopituitarism.

Pituitary dwarfism

Hypopituitarism in children typically results in short stature with normal proportions, and slow growth due to a deficiency in the growth hormone. This condition is called **pituitary dwarfism.**

Causes include anterior pituitary tumours, surgery on the pituitary for tumours, past head injury, tuberculosis, sarcoidosis, and radiation.

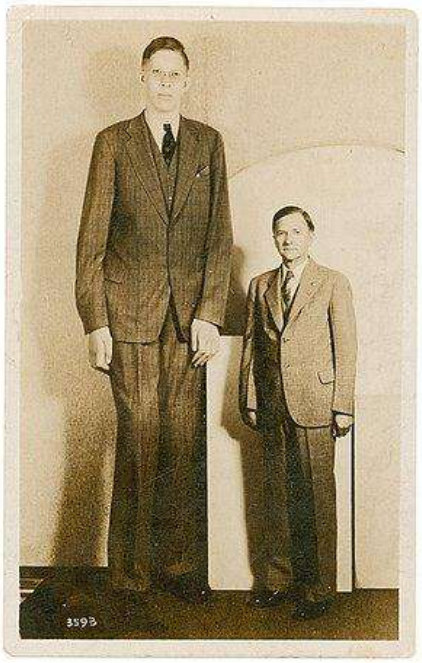
Symptoms and signs include myxoedema, infertility, amenorrhoea, depression, signs of hypoglycaemia, muscle weakness, and short stature.

Investigations include CT scan, MRI, glucose, T4 and TSH levels, prolactin, gonadotrophins, cortisol and testosterone levels.

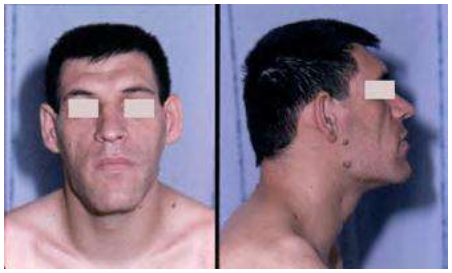
Management: Hormone replacement therapy; recombinant growth hormone (GH) is recommended for children with a deficiency of growth hormone.

- **Acromegaly and gigantism**

Definition/description: Acromegaly and gigantism are syndromes that result when the pituitary gland produces excess growth hormone (GH). Nearly always this is due to a pituitary adenoma. If this occurs after epiphyseal plate closure at puberty, the condition is called **acromegaly**; if before the closure of the epiphyses, the result is **gigantism**.



Symptoms and signs in acromegaly: Excess secretion of growth hormone usually starts between the ages of 20 and 40. Headache is common due to the pituitary tumour. Other features include coarsening of the facial features, enlargement of the extremities, a husky voice, excessive sweating and offensive body odour, hypertension, joint symptoms of degenerative arthritis, peripheral neuropathy, impaired glucose intolerance, menstrual irregularities and heart failure. These patients are at a higher risk of developing gastrointestinal cancers.

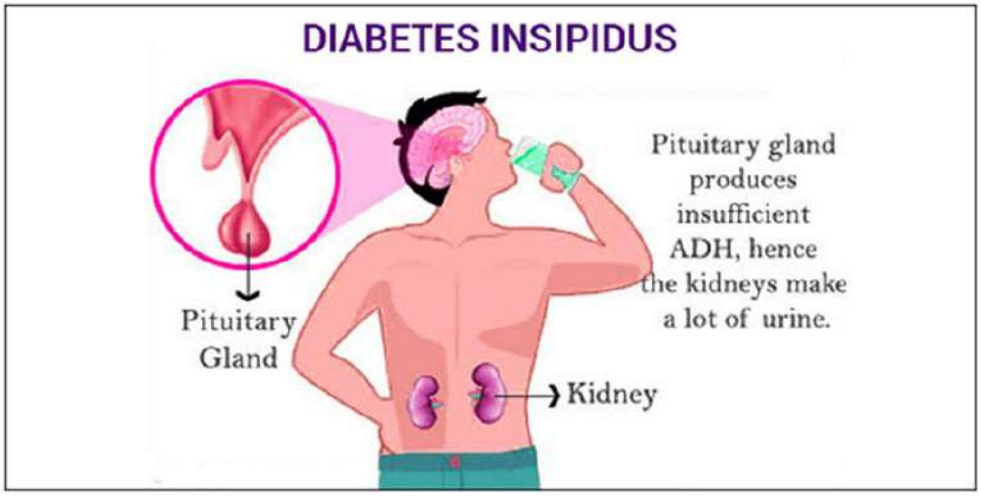


TREATMENT

Treatment for acromegaly is aimed at restoring normal growth hormone levels. The preferred initial therapy for active acromegaly is microsurgical removal of the pituitary tumor with preservation of the gland.

- **Diabetes insipidus (DI)**

Definition: Diabetes insipidus (DI) is characterised by an inability to produce concentrated urine due to the complete or partial deficiency of antidiuretic hormone (ADH), or renal resistance to the ADH action. These are respectively known as cranial diabetes insipidus and nephrogenic diabetes insipidus.



Causes: Causes of cranial DI can be idiopathic, a head injury, or sarcoidosis. Nephrogenic DI may be caused by drugs, renal disease and glycosuria.

Symptoms and signs: These include polyuria resulting in large volumes of pale coloured urine. Frequent urination, nocturia and polydipsia and dehydration are other common symptoms. Loose teeth are another feature of DI.

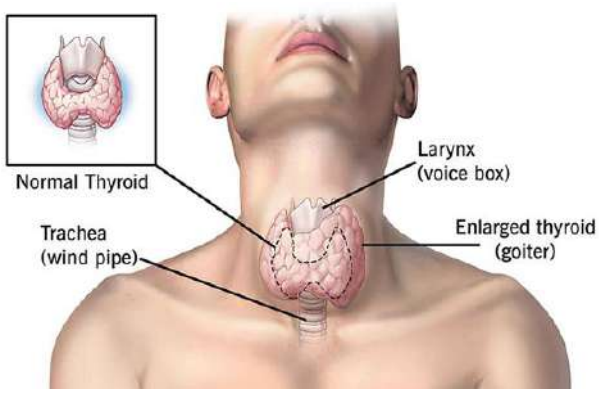
Investigations: Estimation of urine osmolality and a 24-hour urine output is required to confirm polyuria.

Management includes administration of desmopressin for cranial DI, and treatment of the underlying cause for nephrogenic DI. Underlying causes should be identified and treated.

Diseases of the Thyroid Gland

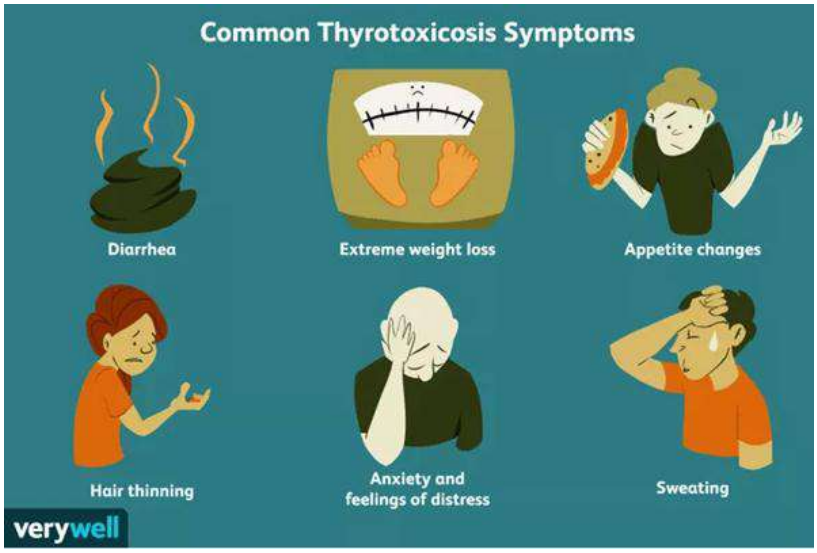
- **Hyperthyroidism (Thyrotoxicosis)**

Definition/description: Also known as thyrotoxicosis, this disorder is characterized by over-production of thyroid hormone. Thyroid hormone exists in two main forms: thyroxin (T4) and triiodothyronine (T3).

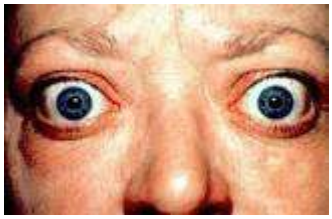
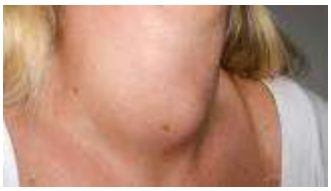
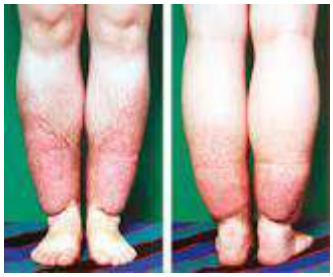


Cause: A common cause of hyperthyroidism is an autoimmune disorder (Graves' disease), where antibodies stimulate the thyroid to secrete excess hormone. Less often, a nodule within the multinodular goiter, or a thyroid adenoma producing excessive thyroxin is responsible.

Symptoms: Common symptoms include sweating, heat intolerance, sleep disturbances, irritability, amenorrhea, palpitations, weight loss, increased appetite, and anxiety.



Signs include tachycardia, atrial fibrillation, exophthalmos, fine tremor, goiter, and pretibial myxoedema.

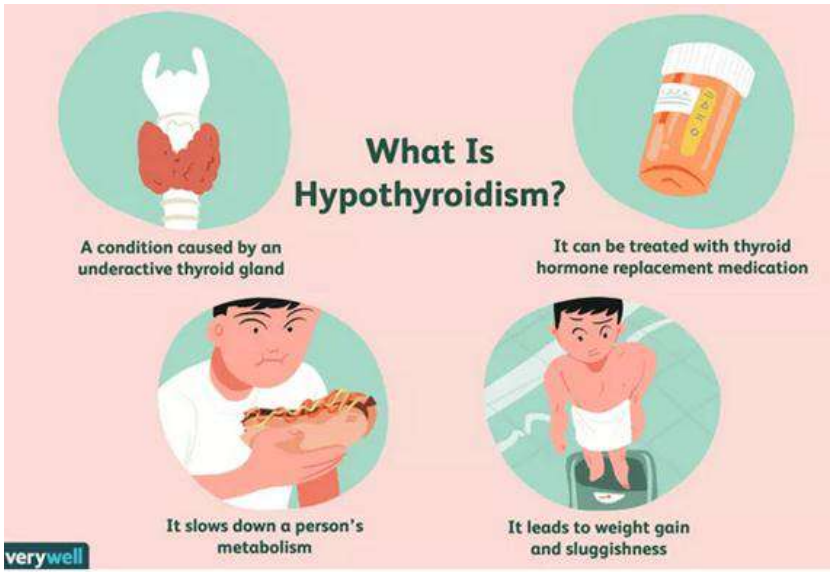




Investigations: T3 is raised (80-220 ng/dL). T4 or (fT4) is also usually raised (5.0 to 12.0µg/dL). Thyroid Stimulating Hormone (TSH) (0.5 to 5.0 mIU/L) may be suppressed in those with multinodular goiter. The presence of thyroid autoantibodies may be detected.

Management includes antithyroid drugs (Carbimazole or propylthiouracil) and surgery

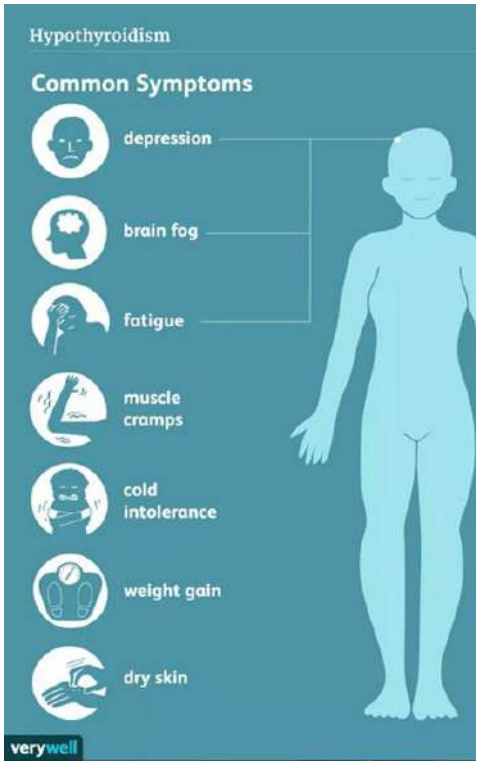
- **Hypothyroidism**

Definition/Description: Underproduction of thyroid hormone results in hypothyroidism.

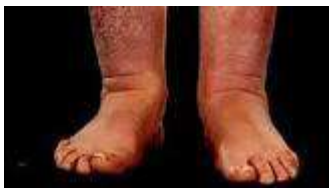


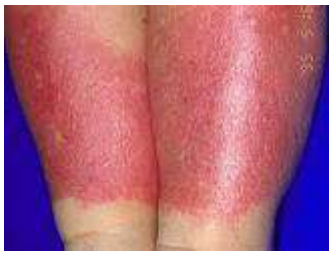
Causes include **an** iodine deficiency, (the most common cause worldwide) or an autoimmune disorder (Hashimoto's disease). Thyroidectomy or radiation of the gland, and occasionally hypopituitarism, may be etiologically associated.

Symptoms include weight gain, cold intolerance, depression, tiredness and constipation.



Signs include the slow relaxation of tendon reflexes, myxoedema (deposition of subcutaneous mucopolysaccharides), hair loss, a hoarse voice, cold skin and bradycardia.



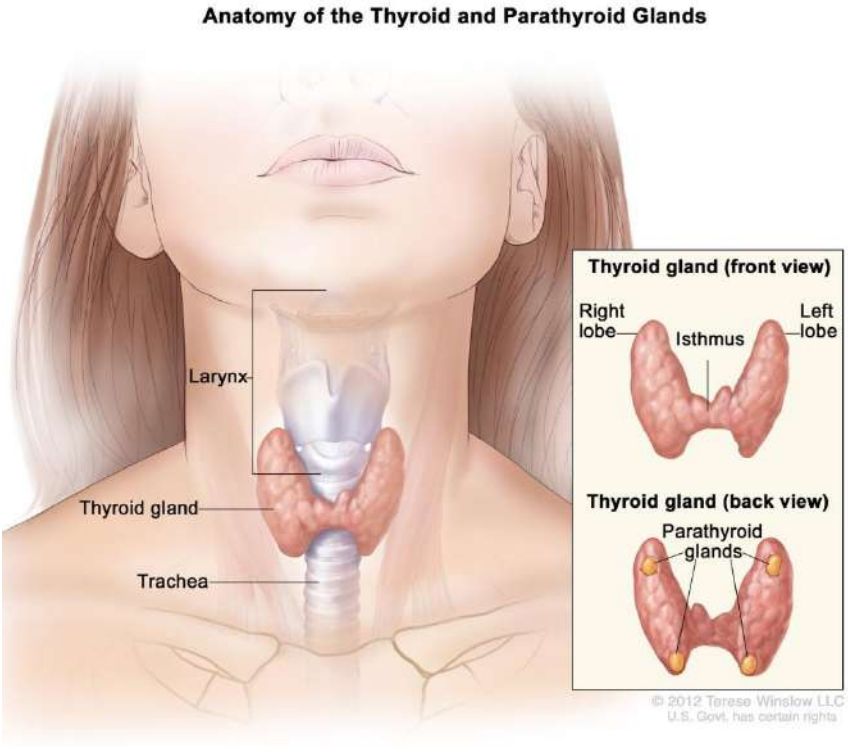


Investigations include estimation of T4 (low) and TSH (high) levels. Tests for cholesterol (hypercholesterolemia) and anemia are necessary. Thyroid autoantibodies in the autoimmune variant can be detected.

Management: Thyroxin replacement is the treatment of choice.

Parathyroid gland & calcium balance

Diseases of the parathyroid glands include primary hyperparathyroidism, secondary hyperparathyroidism, hypoparathyroidism and pseudohypoparathyroidism.



- **Primary hyperparathyroidism**

Definition/description: High levels of parathyroid hormone (PTH) (14 to 65 pg/mL) due to glandular pathology results in primary hyperparathyroidism.

Cause: Adenoma or hyperplasia of the parathyroid gland.

Symptoms and signs: Often, patients are asymptomatic. When symptomatic, features include polyuria, excessive thirst (due to hypercalcaemia), anorexia, weakness, constipation, vomiting, renal colic, backache, hypertension, renal stones, peptic ulceration, giant cell tumour of the bone, and pancreatitis.

Investigations: These include serum calcium (>10.5 mg/dL,), phosphate (raised), alkaline phosphatise (raised), bicarbonate, vitamin D, x-rays of the hands and skull (‗pepper-pot' like erosions of skull), abdominal x-rays for renal stones, ultrasound of the neck, radioactive thallium-technetium subtraction scan and immunoassays for PTH.

Management: This includes surgery for adenoma and hyperplasia of the parathyroid gland. In addition, administration of vitamin D in severe cases may be necessary.

- **Secondary hyperparathyroidism**

Definition/Description: Prolonged hypocalcaemia associated with renal failure and deficiency of dietary vitamin D can lead to secondary hyperparathyroidism. Stimulated PTH can also result in secondary hyperparathyroidism. Treatment includes correction of the underlying cause.

- **Hypoparathyroidism**

Definition/description: Hypoparathyroidism may be either primary due to autoimmune disease, or secondary as a result of thyroid surgery.

Symptoms and signs include peri-oral and peripheral paraesthesia, cramps, and abnormalities of the hair, nails and teeth in chronic cases. Tetany in acute cases is characterized by tingling in the extremities, spasms in the hands, facial twitching.

Investigations include estimation of serum calcium (low), phosphate (raised), and alkaline phosphatase (normal) levels. Skull x-ray shows basal ganglion calcifications, and plasma PTH levels are low.

Management includes IV administration of calcium gluconate and in acute cases, long term alfacalcidol.

Pseudohypoparathyroidism

Definition/description: This is an inherited disorder with resistance to PTH.

Symptoms/signs include a short stature, mental retardation, ‗moon' face, cerebral calcifications, short 4th and 5th metacarpals and hypothyroidism.

Management: Treatment is as for primary hypoparathyroidism.

Disorders of Calcium Metabolism

Introduction

Calcium is required for the proper functioning of muscle contraction, nerve conduction, hormone release and blood coagulation. It also helps regulate several enzymes. Calcium metabolism depends upon a host of factors which include the interplay between dietary calcium, its absorption and excretion; and hormonal interactions of parathormone, vitamin D and calcitonin. Maintenance of calcium is

dependent on dietary calcium intake, its absorption from the GIT and excretion by the kidneys. Ninety-nine percent of body calcium is in the bones as hydroxyapatite crystals. The recommended daily allowance of dietary calcium for healthy individuals is 1 gm. Normal total plasma calcium levels range from 8.8 to 10.4 mg/dL. Disorders of calcium metabolism occur when the body has too little or too much calcium.

- **Hypocalcaemia**

Definition/description: Hypocalcaemia refers to total plasma calcium levels below 8.8 mg/dL in the presence of normal protein levels.

Causes: These include: hypoparathyroidism, vitamin D deficiency, renal disease, magnesium depletion, acute pancreatitis, hypoproteinaemia, septic shock, hyperphosphataemia, and drugs such as anticonvulsants (phenytoin) and rifampin.

Symptoms and signs: Hypocalcaemia may be asymptomatic. When symptomatic, neuromuscular irritability resulting in muscle cramps in the back and legs are common.Prolonged hypocalcaemia may cause cataracts. When plasma levels of calcium reach below 7 mg/dL, tetany may develop. Laryngospasm or generalised seizures are also seen in these patients. Tetany is characterized by paraesthesia of the lips, tongue, fingers and feet, carpopedal spasm, generalized muscle aching, and spasms of the facial musculature.

Other observable changes in hypocalcaemia include dry and scaly skin, brittle nails and coarse hair.

Investigations: These include measurement of plasma Ca levels. Hypocalcaemic patients should also be tested for renal function such as blood urea nitrogen (BUN) and creatinine. Other tests include estimations of serum phosphate, magnesium and alkaline phosphatase levels.

Management: In tetany, intravenous calcium gluconate is administered. In chronic hypocalcaemia, oral Ca and occasionally vitamin D supplements are recommended.

- **Hypercalcaemia**

Definition/description: Hypercalcaemia is characterised by plasma calcium levels above 10.4 mg/L.

Cause: Hypercalcaemia results from excessive bone resorption usually caused by cancer. Parathyroid hormone excess (hyperparathyroidism) also can cause excessive bone resorption and hypercalcaemia. Other causes of hypercalcaemia include excessive absorption of calcium in the GIT and elevated plasma protein

concentrations. Immobilization with prolonged complete bed rest can cause bone resorption and hypercalcaemia.

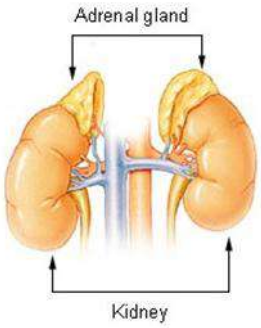
Symptoms and signs: Mild hypercalcaemia may be asymptomatic. Clinical manifestations include constipation, anorexia, abdominal pain, nausea and vomiting. polyuria, nocturia and polydipsia are common. In severe cases confusion, delirium, psychosis and coma may occur. Renal damage includes acute renal failure or chronic damage due to kidney stones.

Investigations include plasma calcium levels, renal function tests, and appropriate tests for hyperparathyroidism.

Management: Management strategies include a decrease in calcium intake, an increase in calcium excretion (IV fluids and diuretics), decrease in bone resorption, and removal of excess calcium through dialysis. In patients with vitamin D toxicity, the use of corticosteroids is effective in reducing the intestinal absorption of calcium. Calcitonin (Miacalcin), controls calcium levels in the blood.

Diseases of the Adrenal Gland

The adrenal glands comprise two major 'functional units' – the cortex and the medulla. The cortex consists of three zones: an 'inner' zona reticularis (secreting androgens, e.g. dehydroepiandrosterone sulphate (DHEAS)), a 'middle' zona fasciculate (secreting glucocorticoids, e.g. cortisol) and an 'outer' zona glomerulosa (secreting mineralocorticoids, e.g. aldosterone).



Cortisol has many vital metabolic and immunomodulatory effects and is important in the maintenance of normal circulatory function. Aldosterone promotes renal sodium retention and potassium excretion.

- **Primary hyperaldosteronism**

Primary hyperaldosteronism is an important treatable cause of hypertension in the young to middle-aged.

Aetiology

Many cases are caused by benign aldosterone producing adenomas (so-called Conn's adenomas), but bilateral adrenal hyperplasia/nodular disease is also found in a significant number of patients.

Clinical presentation

Most cases come to light during investigation of hypertension or unexplained hypokalaemia. Nonspecific symptoms including weakness, lassitude and polyuria (hypokalaemia may be associated with nephrogenic DI) are reported by some patients. Evidence of end organ damage (e.g. hypertensive retinopathy and nephropathy) may be seen in longstanding, inadequately treated cases.

Investigation

. Creatinine and electrolytes – the classical picture is one of hypokalaemic alkalosis: the accompanying serum sodium level is typically normal to high.

. Urinary potassium and sodium – hypokalaemia is associated with an inappropriate kaliuresis.

. Plasma renin and aldosterone – the hallmark of primary hyperaldosteronism is excessive autonomous production of aldosterone, occurring in the face of renin suppression.

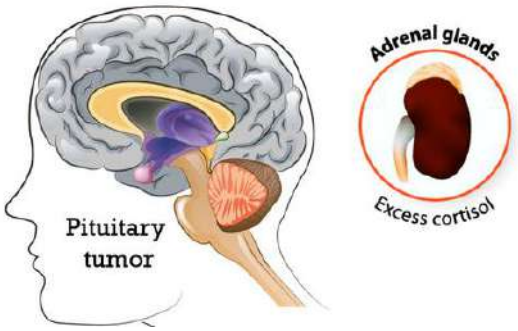
. CT/MRI of the adrenals may help to distinguish unilateral adenoma from bilateral hyperplasia.

Management

Spironolactone is the medical treatment of choice by virtue of its ability to block the action of aldosterone at the mineralocorticoid receptor.

- **Adrenocortical Excess (Cushing's syndrome); hyperadrenalism**

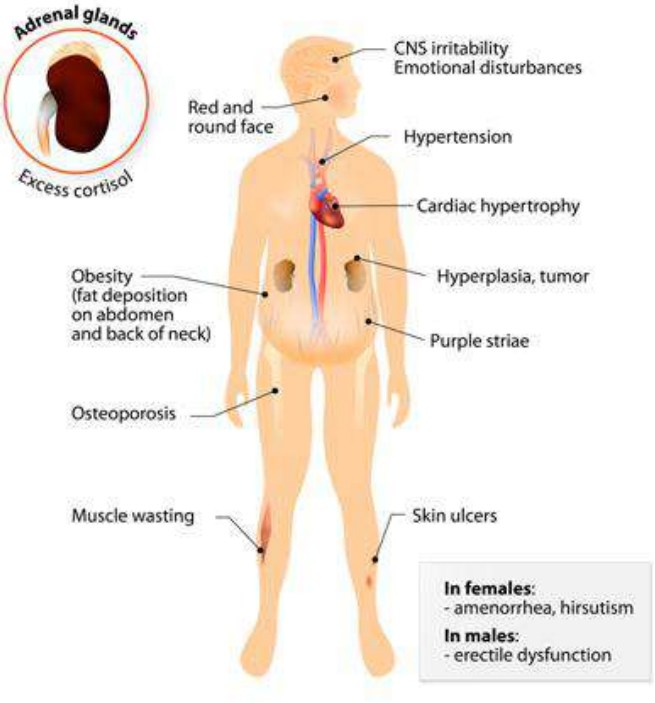
Definition/description: Cushing‘s syndrome refers to the clinical picture resulting from circulating cortisol excess from any cause, whereas **Cushing‘s disease** results from hyperfunction of the adrenal cortex from pituitary Adrenocorticotropic Hormone (ACTH) excess, usually due to pituitary adenoma.

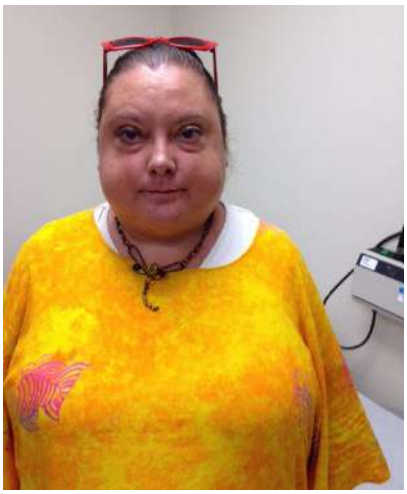


Cause: Hyperfunction of the adrenal cortex due to ACTH-dependent or ACTH–independent causes. ACTH–dependant causes include hypersecretion of ACTH from the pituitary gland, or from a non-pituitary tumour such as small cell carcinoma of the lung (ectopic ACTH syndrome), or by administration of exogenous ACTH.

ACTH-independent hyperfunction usually results from therapeutic administration of corticosteroids, or from adrenal adenomas or carcinomas.

Symptoms and signs: These include wasting of tissues, myopathy, thin skin, osteoporosis, easy bruising, truncal obesity with prominent supraclavicular and dorsal cervical fat pads, head and neck fat accumulation (buffalo hump), thin extremities, moon facies, hirsutism (in which women have too much unwanted hair), increased susceptibility to infections, poor wound healing and purple striae on the abdomen.





Investigations: Urine is to be tested for free cortisol and electrolytes (hypocalaemia). Other tests include a glucose intolerance test, measurement of blood pressure (hypertension), chest x-rays (to exclude bronchial carcinoma), estimation of ACTH levels (high in pituitary disease), serum cortisol levels and CT scans of the pituitary and adrenal glands.

Management includes surgery on the pituitary tumours or adrenal glands where appropriate, and drugs to inhibit cortisol levels.

- **Adrenal insufficiency: Primary adrenal insufficiency (Addison's disease) and secondary adrenal insufficiency**

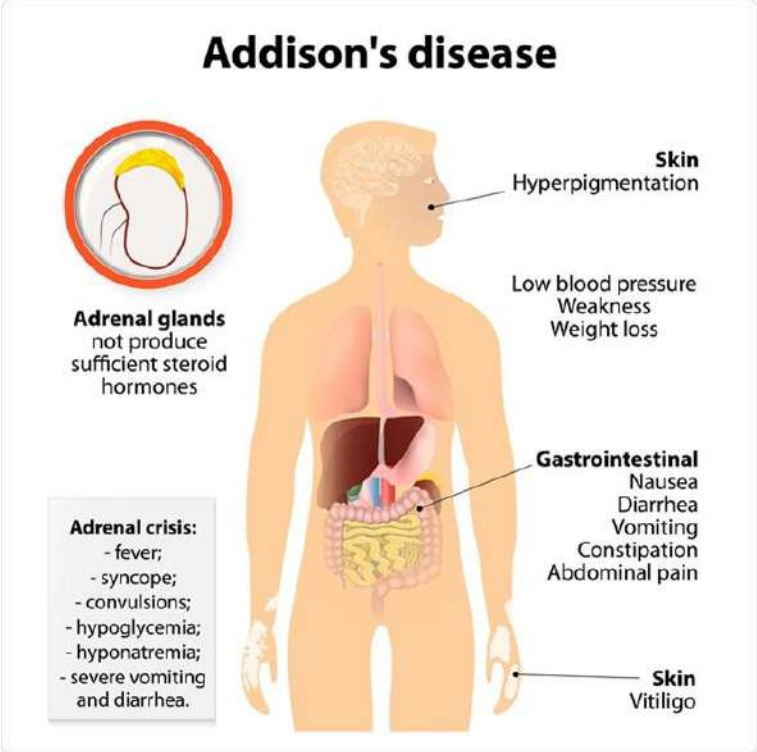
Definition/Description: A disease of the adrenal glands causing primary adrenal insufficiency is called **Addison‟s disease**.

Cause: Primary adrenal insufficiency involves autoimmune destruction of the glands in about 80% of cases. Other causes include TB, metastatic disease and hypoparathyroidism, diabetes mellitus and Graves‘ disease.

Secondary adrenal insufficiency results from panhypopituitarism, a lack of ACTH, and in those patients who are receiving steroids.

Symptoms/Signs: In primary adrenal insufficiency (Addison‘s disease), lack of cortisol and aldosterone exists. Clinical features include nausea, shock, and bowel

disturbances. Other features of adrenal insufficiency include weakness, apathy, anorexia, weight loss, abdominal pain, infrequent periods and constipation. Hypotension, vitiligo, hyperpigmentation of mucous membranes (lips, rectum and vagina, for example), and those areas exposed to sunlight (called ―bronzing‖) and pressure (such as bony prominences). Black freckles are common on the forehead, face, neck and shoulders. Increased pigmentation is due to increased levels of ACTH. In secondary adrenal insufficiency, most clinical features are similar to those of primary adrenal insufficiency, however, muco-cutaneous pigmentation is absent.



Weight loss/anorexia

Pigmentation of feet

Treatment

Addison’s disease is treated with medications that serve to replace the hormones that the body cannot produce. These drugs include hydrocortisone, which is used to replace cortisol, and mineralocorticoid, which is used to replace aldosterone.

Diabetes mellitus: complications, management, preparation for operation

Definition/description: Diabetes mellitus (DM) is a group of disorders characterized by persistent hyperglycaemia due to a deficiency of endogenous insulin, or resistance to insulin action.

Two types of DM exist: type 1 and type 2 DM.

Type 1 is insulin dependent. Usually it is found in children, who are often prone to ketosis. Approximately 5-10% of the people who have diabetes have type 1.

Type 2 is non-insulin dependent, and usually occurs in obese older adults.

Cause: An autoimmune process resulting in β-cell destruction of the pancreas is the cause of type1 DM (that stops your body from making insulin). Obesity and genetic component are associated with type 2 DM (your body doesn't use insulin well and can't keep blood sugar at normal levels). Other factors associated with type 2 DM include drugs such as corticosteroid therapy and thiazides, as well as pancreatic disease, Cushing's disease, acromegaly and thyrotoxicosis.

Symptoms and signs include irritability, tiredness, thirst, a dry mouth, weight loss, nocturia, blurring of vision, hyperphagia (excessive hunger and eating), dehydration, ketonuria, hyperventilation, ketone breath, obesity, lethargy, increased susceptibility to infections (such as pruritus vulvae), and delayed wound healing. These are presenting symptoms of type 2 DM. Polyphagia, polydypsia (excessive thirst) and polyuria (the three _P's) are classic symptoms of Type 1 DM.

Investigations for DM include:

1. Urine examination by glucose strips or dipstick method. If glycosuria is detected, the patient should be tested for blood glucose levels.

2. Random blood glucose and fasting blood glucose tests. Typical blood glucose levels that people with diabetes aim at:

•Before eating food: 80 to 130 mg/dL

• About 2 hours after eating food: less than 180 mg/dL

3.An oral glucose tolerance test (OGTT) is indicative in borderline cases. [This measures your blood sugar before and after you drink a liquid that contains 75 g oral glucose]. At 2 hours (140 mg/dL normal, 140 to 199 mg/dL prediabetes, and 200 mg/dL or higher indicates diabetes.].

4. HbA1c (glycosylated haemoglobin) measurements, measures the amount of blood sugar (glucose) attached to hemoglobin (which provide an accurate measurement of glycaemic control over a period of three months).

Normal: HbA1c below 5.7%

Prediabetes: HbA1c between 5.7% and 6.4%

Diabetes: HbA1c of 6.5% or higher.

5. Tests for the **evaluation of serum** cholesterol (for hypercholesterolemia), creatinine, microalbuminuria for renal disease, **fundoscopy** for diabetic retinopathy, hypertension and peripheral neuropathy.

Complications of DM: DM can damage a variety of organs.

o **Kidney Damage:**

o **Ocular damage**:

o **Heart:** chronic heart disease (CHD) is common (diabetic cardiomyopathy).

o **Circulation:** atheroma of large vessels. Small vessel disease may cause distal gangrene.

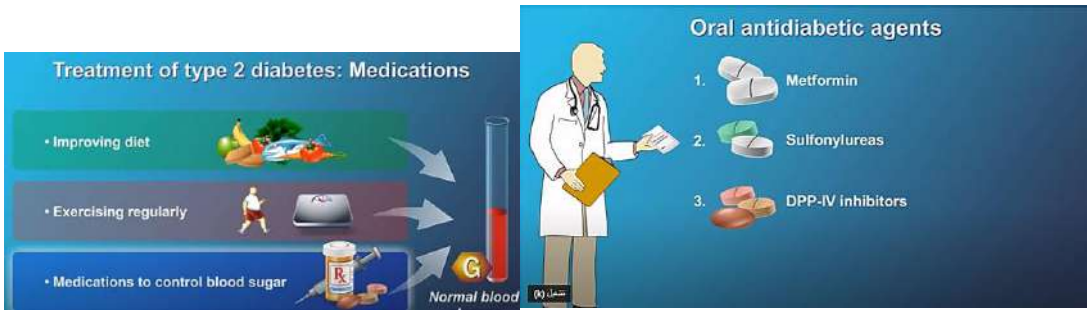
o **Nervous system**: peripheral neuropathy

Transient diabetes as a complication of pregnancy is known as gestational diabetes.

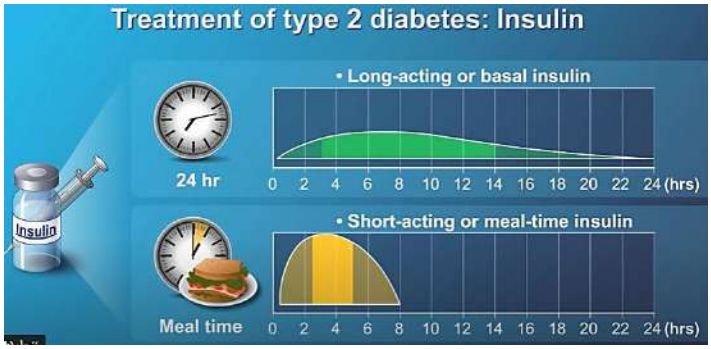
Management:

Type 1 DM: Short acting insulin preparations (e.g., Humalog) before meals and long acting (e.g., Humulin I) before bedtime are recommended.

Type 2 DM: dietary control, physical exercise, reduction in weight and restricted carbohydrate intake are essential.



Dipeptidyl peptidase-4 (DPP-4) inhibitors (gliptins) are a relatively new class of oral diabetes drugs. include sitagliptin, saxagliptin, linagliptin, and alogliptin.



PREOPERATIVE ASSESSMENT

• History–regarding

– Duration of the disease and drug therapy the patient is receiving, i.e. oral hypoglycemic agents or insulin.

– Adequacy of control of blood sugar.

– Symptoms suggestive of cardiovascular disease.

– Frequency and severity of hyperglycemic and/or hyperglycemic episodes.

– Neurological symptoms- stroke, tingling/ numbness, mononeuropathy, glove-and stocking paresthesia, etc.

– Diarrhea after meals, reflux esophagitis.

• Examination:

– Weight, body habitus

– Blood pressure in sitting and standing positions

– 'Prayer' sign (The patient is typically unable to straighten the interphalangeal joints of the fourth and fifth fingers.)

– Airway assessment

– Neurological examination to document any deficit

– Anatomy of spine for feasibility of spinal anesthesia

• Relevant preoperative investigations

– The hallmark of diabetes is a fasting blood sugar greater than 126 mg/dl or a random blood sugar of more than 200 mg/dl.

– Glycosylated hemoglobin (Hb AIC) levels reflect adequacy of control over the preceding 1-3 months. Levels between 5-7% of total hemoglobin are normal whereas more than 9% indicates poor long term glucose control.

– Laboratory investigations include determination of blood sugar, blood urea, serum creatinine, serum electrolytes and urine analysis for glucose, ketones and proteins.

• Goals of optimization

– To avoid hypoglycaemia as well as hyperglycaemia and maintain blood sugar between 120-180 mg/dL.

– Identification of associated end-organ disease and initiation of interventions to limit cardiovascular, renal and metabolic complications.

OPTIMAL ANESTHETIC MANAGEMENT

Pre-anesthetic Instructions:

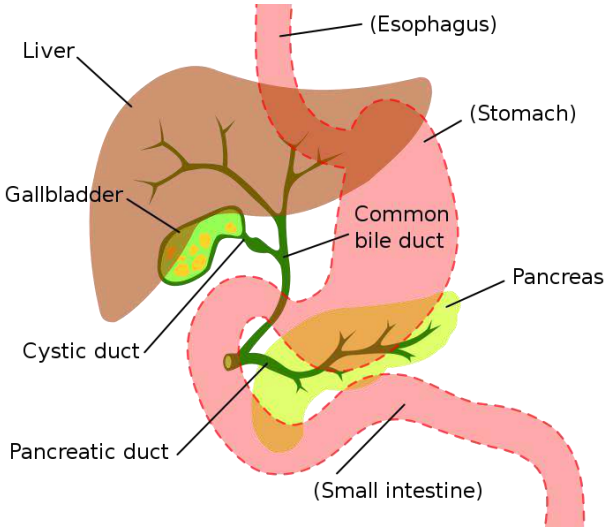
• Ideally a diabetic patient should be scheduled as the first case in the morning to minimize metabolic complications.

• *Type I diabetics:* Half the usual dose of short acting insulin should be administered on the evening before surgery. Withhold insulin on the morning of surgery; blood sugar levels, serum electrolytes as well as urine assay for sugar and ketones is carried out on the morning of surgery.

• *Type II diabetics:* Patients with blood sugars controlled on diet and oral hypoglycemic drugs should withold the oral hypoglycemic agent with the exception of acarbose on the morning of surgery.

• *Type II diabetics* undergoing major abdominal surgery will required to be admitted at least 48 hours before surgery, started on neutralizing drip with insulin or on separate insulin and glucose infusions as described for type I diabetics.

Preparation for patient with obstructive jaundice



Any condition that obstructs this bile flow causes "Obstructive Jaundice"

Preoperative management of obstructive jaundice

Preparation entails the correction of metabolic abnormalities, improvement of the general condition, and institution of specific measures designed to minimize the incidence of complications associated with prolonged or severe cholestasis such as: infections (cholangitis, septicemia, wound infections), disorders of the clotting mechanism; renal failure; liver failure; fluid and electrolyte abnormalities.

Preparation of patient with obstructive jaundice

The preparation of the jaundiced patient includes all of the precautionary and rehabilitative measures which should precede every major operation.

In addition it should include measures directed toward a reduction of the hemocoagulation time and the bleeding time.

1. The diet should be rich in carbohydrates and poor in proteins.
2. The water balance should be regulated. In chronic cases blood chemistry studies should be made where there is indication of disturbance of renal function.
3. Glucose should be administrated in adequate amounts. An isotonic, properly buffered solution in distilled water may be used intravenously or subcutaneously.
4. Normal salt solution should be given in sufficient amounts to overcome the loss of salts by vomiting or diarrhea.
5. Blood transfusions are of definite value.
6. The use of calcium salts intravenously is indicated.
7. Vitamin deficiencies should be corrected by the administration of the fat soluble vitamins A and D and the water soluble vitamin C.

8. Orally administered bile preparations are used to facilitate the absorption of the fat soluble vitamins.

The specific measures required in all patients are:

 Clotting studies should be checked prior to surgery

 Parenteral **administration of vitamin K** analogues (10mg IM or IV) – to normalize prothrombin.

 INR ratio (International Normalized Ratio) (It is also known as prothrombin time) should be less than 1.5 to avoid excessive operation bleeding and if urgent surgery is required, FFP (Fresh frozen plasma) should be administered immediately before and during the procedure.

 Intravenous hydration (Mostly glucose water) and catheterization of the urinary bladder.

 Patients with cardiac disease or sepsis should undergo **central venous monitoring** and in some cases dopamine infusions may be required.

 Forced natriuresis by mannitol with induction of anesthesia (If mannitol not effective give furosemide IV).

 Antibiotic prophylaxis against gram negative aerobes – using a three dose regimen against gram negative, positive and anaerobes.

 Pruritus usually subsides with correction of the underlying disorder or with 2 to 8 gm. orally administered **cholestyramine** bid.

 Unless severe hepatocellular damage is present, hypoprothrombinemia usually subsides after use of (**vitamin K1**) 5 to 10 mg SC once/day for 2 to 3 days.

 Ca and vitamin D supplements, with or without a bisphosphonate, slow the progression of osteoporosis only slightly in long-standing irreversible cholestasis.

 Vitamin A supplements prevent deficiency and severe steatorrhea can be minimized by replacing some dietary fat with medium-chain triglycerides.

Preparation for patient with portal hypertension due to cirrhosis

The preoperative evaluation of a patient with liver disease has long been considered more art than science. Preparation should include correction of coagulopathy, treating preexisting encephalopathy, controlling ascites, preventing sepsis, and optimizing renal function.

Scoring System and the Preoperative Risk Evaluation

The 2 most widely used scoring systems to help predict the morbidity and mortality of patients with cirrhosis undergoing various types of surgeries are Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) score.

CTP classification for severity of cirrhosis

	Points		
Parameter	**1**	**2**	**3**
Ascites	None	Mild to moderate	Severe
Hepatic Encephalopathy	None	Mild to moderate (grade 1–2)	Severe (grade 3–4)
Total bilirubin (mg/dL)	<2	2–3	>3
Serum albumin (g/dL)	>3.5	2.8–3.5	<2.8
International Normalized Ratio	<1.7	1.7–2.3	>2.3

CTP score is obtained by adding the score for each parameter. CTP class A, 5–6 points; class B, 7–9 points; class C, 10–15 points.

In general, elective surgery is well tolerated in patients with Child class A, permitted with careful preoperative preparation in patients with Child class B, and contraindicated in patients with Child class C.

The MELD score uses the patient's bilirubin, serum creatinine, and international normalized ratio (INR). Any value less than 1 is given a value of 1, and if the dialysis occurred twice in the last 1 week, the factor for serum creatinine value is 4.0: MELD = 3.78 × ln[serum bilirubin (mg/dL)] + 11.2 × ln[INR] + 9.57 × ln[serum creatinine (mg/dL)] + 6.43

Preoperative Strategies

Preoperative checklist in patients with cirrhosis or portal hypertension

In an attempt to simplify the process of assessing surgical risk in patients with liver disease, we propose the use of a preoperative liver assessment (POLA) checklist that could not only be privy to consultants but could be embedded into increasingly prevalent electronic medical records in appropriate patient situations, allowing access by other providers as well. Similar preoperative checklists have been shown to be valuable and can reduce morbidity and mortality. The POLA checklist is meant to be filled out in chronologic order, and is shown in Fig.1. Further investigation is required to determine whether implementation of the POLA checklist has any effects on patient and provider decision making, patient care, and outcomes.

The preoperative evaluation concludes with a review of all pertinent studies and information obtained from investigative tests.

Informed consent after discussion with the patient and family members regarding the indication for the anticipated surgical procedure, as well as its risks and proposed benefits

Review the need for β-blockade, DVT prophylaxis

Antibiotic prophylaxis: The appropriate antibiotic is chosen before surgery and administered before the skin incision is made

Preoperative mechanical bowel cleansing, whenever indicated

Revision of medications

- Careful review of the patient's medications is important.
- The aim is to judiciously give medications that control the patient's illnesses and at the same time minimizing the risk associated with anesthetic and other drugs interactions.
- In general, patients taking cardiac drugs, pulmonary drugs or anticonvulsants, antihypertensives, or psychiatric drugs are advised to take their medications with sips of water on the morning of operation.
- Parenteral medications are used if the patient remains NPO for any significant period postoperatively.
- It is important to restitute patients to their usual medications as soon as possible.
- Drugs affecting platelet function are withheld for variable time: aspirin and clopidogrel (Plavix) are withheld for 7-10 days, while NSAIDs are withheld depending on the drug's half-life between 1 day (ibuprofen and indomethacin) and 3 days (naproxen and sulindac).

Preoperative fasting

❒ **Emergent or elective**

- If surgery is potentially life-saving, proceed with surgery with adequate informed consent, but also consider nonsurgical alternatives like such as ongoing medical therapy or interventional radiologic procedures or palliative care as appropriate.

❒ **Characterize liver disease**

- Determine cause and chronicity of liver disease.
  - If acute viral or alcoholic hepatitis or severe drug-induced injury, postpone surgery for at least 3 months
  - If chronic but mild liver disease, proceed with surgery
  - If there is evidence of cirrhosis or noncirrhotic portal hypertension, continue with liver assessment

❒ **Identify significant comorbid conditions**

- Focus on presence of diabetes, chronic kidney disease, and cardiovascular disease
- If moderate or severe nutrition is present, optimize nutrition by oral, enteral, or even parenteral means before surgery

❒ **Perform liver imaging**

- MRI or CT are preferred to evaluate for liver appearance, vessel patency, hepatocellular carcinoma, and evidence of portal hypertension (eg, intra-abdominal varices, spleen size)
- Ultrasound with Doppler is sufficient if there is contraindication to CT or MRI such as acute kidney injury

❒ **Obtain history of prior hepatic decompensation**

- Ascites: if yes, consider future impact on wound healing with postoperative recurrence
- Encephalopathy: if yes, adjust planned sedation and analgesia, and monitor for regular bowel movements.

  Do not restrict dietary protein (give 1.2–1.5 g/kg protein daily)
- Variceal bleeding: if yes, perform upper endoscopy and initiate variceal hemorrhage prophylaxis

❒ **Evaluate for current hepatic decompensation**

- Ascites: if yes, perform diagnostic paracentesis to evaluate for SBP
  If moderate or severe, perform LVP before surgery
  Consider preoperative TIPS if diuretic resistant and MELD <15, but not typically for emergent cases
  Give 2 g sodium diet, 35–45 kcal/g daily
- Encephalopathy: if yes, optimize lactulose to achieve 2–4 bowel movements/day (even by NGT) and give rifaximin
  Do not restrict dietary protein (give 1.2–1.5 g/kg protein daily)
  Order aspiration precautions
- Variceal bleeding: if yes, perform upper endoscopy and initiate variceal hemorrhage prophylaxis
- Hypoxemia or CHF: if yes, consider hepatopulmonary syndrome or portopulmonary hypertension
  Perform ABG, contrast-enhanced echocardiography

❒ **Estimate liver function and likelihood of portal hypertension**

- Check serum total bilirubin, albumin, INR, creatinine, platelets, hepatic venous pressure gradient, if available

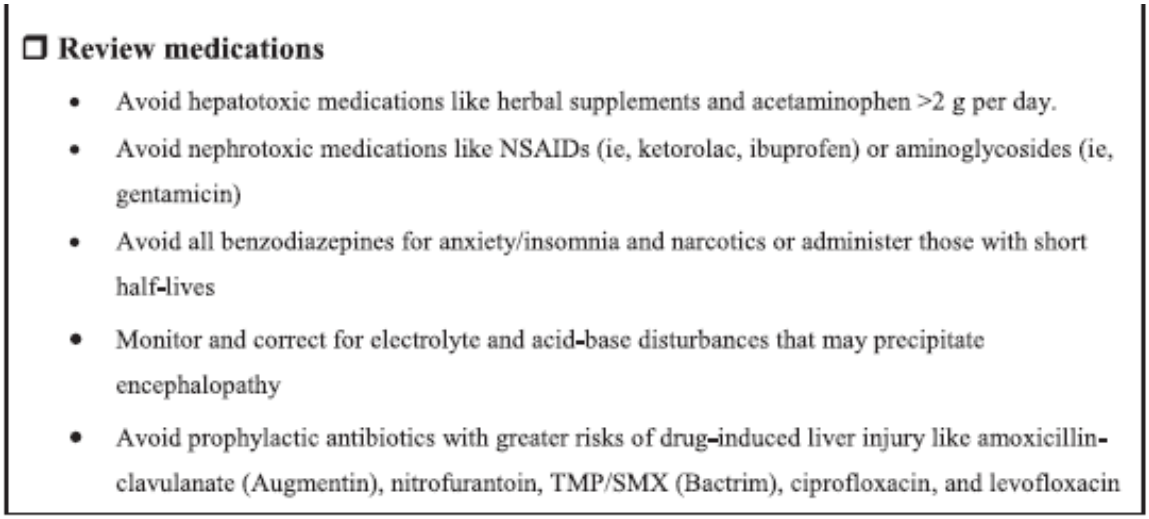
❒ **Calculate CTP, MELD, and modified MELD for surgery at several time points**

(postoperative mortality risk in patients with:
(cirrhosis calculator found at http://www.mayoclinic.org/meld/mayomodel9.html); note: all cirrhotics are ≥ASA class III)

- If Child C or MELD >12 or high risk, consider alternatives to surgery or transfer to liver transplant center
- If Child C or MELD >12 or high risk, consider completing liver transplant evaluation before surgery

❒ **Evaluate coagulopathy and anemia**

- Give subcutaneous vitamin K supplementation leading up to surgery
- Give DDAVP/desmopressin if renal insufficiency present
- Consider use of recombinant factor VIIa for refractory hemorrhage
- In the absence of hemorrhage, do not transfuse platelets if count >50 × 10^3/μL or cryoprecipitate if fibrinogen >50 mg/dL
- Avoid overtransfusion to correct anemia (use hemoglobin goal of 7 g/dL) to avoid increasing portal pressures



❒ **Review medications**

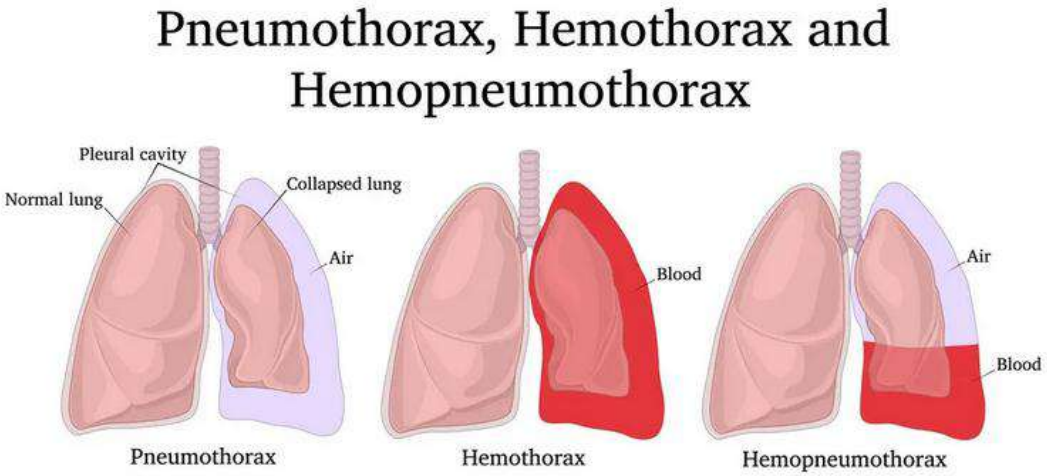
- Avoid hepatotoxic medications like herbal supplements and acetaminophen >2 g per day.
- Avoid nephrotoxic medications like NSAIDs (ie, ketorolac, ibuprofen) or aminoglycosides (ie, gentamicin)
- Avoid all benzodiazepines for anxiety/insomnia and narcotics or administer those with short half-lives
- Monitor and correct for electrolyte and acid-base disturbances that may precipitate encephalopathy
- Avoid prophylactic antibiotics with greater risks of drug-induced liver injury like amoxicillin-clavulanate (Augmentin), nitrofurantoin, TMP/SMX (Bactrim), ciprofloxacin, and levofloxacin

Fig. 1. Preoperative Liver Assessment (POLA) checklist. ABG, arterial blood gas; CHF, congestive heart failure; CT, computed tomography; ddAVP, desamino-D-arginine vasopressin; INR, International Normalized Ratio; LVP, large volume paracentesis; MRI, magnetic resonance imaging; NGT, nasogastric tube; NSAIDs, nonsteroidal antiinflammatory drugs; SBP, spontaneous bacterial peritonitis; TMP/SMX, trimethoprim/sulfamethoxazole.

Management of haemopneumothorax, flail chest

DEFINITION

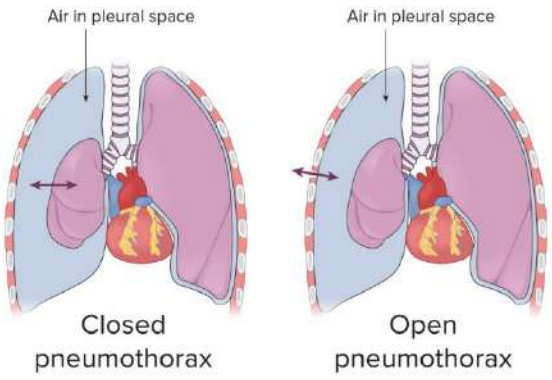
Pneumothorax is the presence of air or gas in the pleural space. Hemothorax is the presence of blood in the pleural space.



ETIOLOGY AND TREATMENT

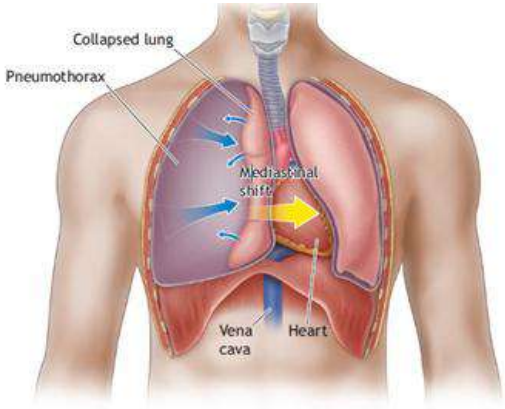
Pneumothorax

Pneumothorax can be subdivided into three categories, depending on whether air has direct access to the pleural cavity. In simple pneumothorax, no communication exists with the atmosphere. Additionally, no shift of the mediastinum or hemidiaphragm results from the accumulation of air in the intrapleural space. The severity of pneumothoraces is graded on the basis of the degree of collapse: collapse of 15% or less is small; collapse of 15% to 60% is moderate; and collapse of greater than 60% is large. Treatment of simple pneumothorax is determined by the size and cause of injury and may include catheter aspiration or tube thoracostomy; close observation of the patient with simple pneumothorax is essential.



In communicating pneumothorax, air in the pleural cavity exchanges with atmospheric air through a defect in the chest wall. Because the exchange of air through the site of injury may often be heard, this entity is commonly known as a "sucking chest wound." **Treatment** measures include administration of supplemental oxygen, tube thoracostomy, and intubation; mechanical ventilation may be indicated.

Tension pneumothorax develops when air progressively accumulates under pressure within the pleural cavity. If the pressure becomes too great, the mediastinum shifts to the opposite hemithorax, and this causes compression of the contralateral lung and great vessels. Tension pneumothorax is potentially lethal; therefore, immediate treatment is essential. Decompression of the chest can be performed with the insertion of a 16- or 18-gauge angiocatheter into the second or third interspace anteriorly or the fourth or fifth interspace laterally.



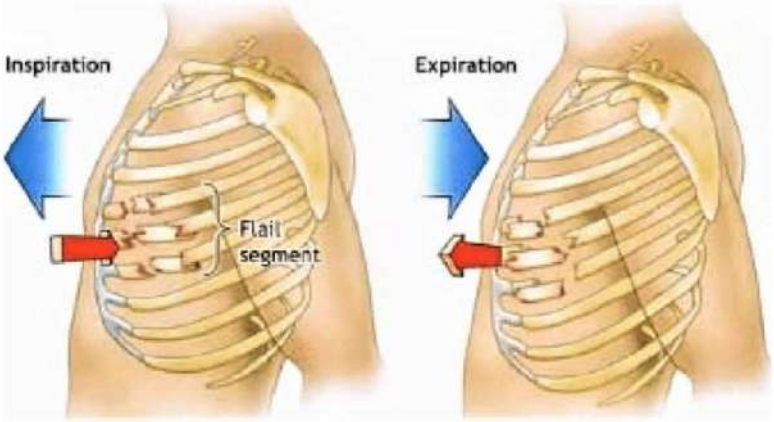
Hemothorax

Hemothorax is the accumulation of blood in the pleural cavity. It usually is a result of trauma, but other causes include the rupture of small blood vessels in the presence of inflammation, pneumonia, tuberculosis, or erosion by tumors. The treatment of hemothorax consists of airway management as necessary, restoration of circulating blood volume, and evacuation of the accumulated blood. Thoracostomy may be indicated if the initial bleeding rate is greater than 20 mL/kg/hr. If bleeding subsides but its rate remains greater than 7 mL/kg/hr, if chest radiographic findings worsen, or if hypotension persists after initial blood replacement and decompression, thoracostomy is indicated.

Ref: *Anesth. John J. Nagelhout, Karen Plaus - Handbook of Nurse Anesthesia (2009, Saunders) - libgen.lc*

A FLAIL CHEST

results from fractures of more than two sites of at least three adjacent ribs or rib fractures with associated costochondral separation or sternal fracture.



- It often develops over a 3- to 6-hour period, causing gradual deterioration of the chest radiograph and arterial blood gases (ABGs).
- Effective pain relief by itself can improve respiratory function and often avoid the need for mechanical ventilation (continuous epidural analgesia).
- Systemic air embolism occurs mainly after penetrating lung trauma and blast injuries or less frequently after blunt thoracic trauma that produces lacerations of both distal air passages and pulmonary veins.

  a. Respiratory maneuvers that minimize or prevent air entry into the systemic circulation include isolating and collapsing the lacerated lung by means of a double-lumen tube or ventilation with the lowest possible tidal volumes via a single-lumen tube.

  b. Transesophageal echocardiography (TEE) of the left side of the heart may permit visualization of air bubbles and their disappearance with therapeutic maneuvers.

Ref: *Paul Barash_ Bruce F. Cullen_ Robert K. Stoelting_ Michael Cahalan_ M. Christine Stock_ Rafael Ortega - Handbook of Clinical Anesthesia-Lippincott Williams & Wilkins (2013)*

Management of respiratory failure, ARDS

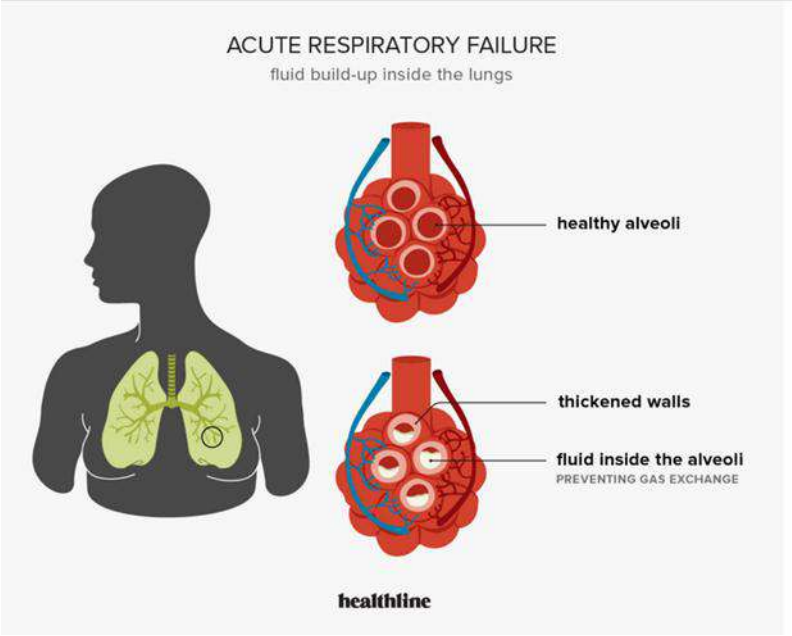
Definition

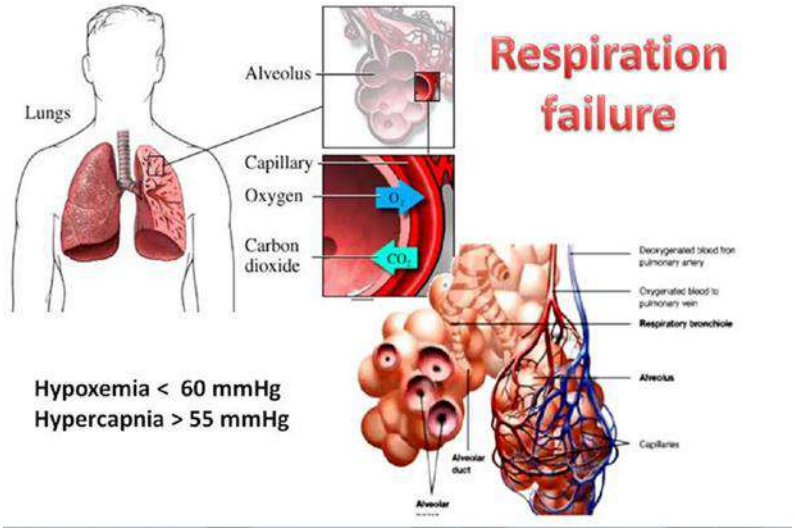
The term *acute respiratory failure* is often used synonymously with *acute* (formerly *adult*) *respiratory distress syndrome* (ARDS). Respiratory failure occurs when pulmonary gas exchange is sufficiently impaired to cause hypoxaemia with or without hypercapnia. In practical terms, respiratory failure is present when the PaO2 is<8 kPa (60 mmHg) or the PaCO2 is >7 kPa (55 mmHg).

It can be divided into two types:

• Type 1 respiratory failure in which the PaO2 is low and the PaCO2 is normal or low. It is most commonly caused by diseases that damage lung tissue.

• Type 2 respiratory failure in which the PaO2 is low and the PaCO2 is high is caused by alveolar hypoventilation.





The clinical presentation includes patients who are dyspneic, hypoxic, and hypovolemic and require intubation and mechanical ventilation. Recovery of lung function is unpredictable. Milder cases resolve quickly, whereas others progress to fibrosis and death.

ETIOLOGY

Events and risk factors associated with the development of ARDS include the following: (1) shock (septic, cardiogenic, or hypovolemic); (2) trauma; (3) pulmonary infection (e.g., with *Pneumocystis carinii (jiroveci)* or *Escherichia coli*); (4) disease states that result in the release of inflammatory mediators (e.g., extrapulmonary infections, disseminated intravascular coagulation, anaphylaxis, coronary bypass grafting, and transfusion reactions); (5) exposure to various agents (e.g., narcotics, barbiturates, and O2); (6) diseases of the central nervous system; (7) aspiration (e.g., of gastric contents or as in drowning); and (8) metabolic events (e.g., pancreatitis and uremia).

Table 1. Types Of Respiratory Failure And Their Management Approaches

Type of Respiratory Failure	Examples	Management Approach
Hypoxic (inadequate oxygenation)	• Pneumonia • Congestive heart failure • Interstitial respiratory disease	• Increase fraction of inspired oxygen • Increase mean airway pressure • Increase peak end-expiratory pressure
Hypercarbic (inadequate ventilation)	• Chronic obstructive pulmonary disease	• Increase respiratory rate • Increase tidal volume

Management

Because lung infections (e.g., *P. carinii [jiroveci]* pneumonia) mimic acute respiratory distress syndrome (ARDS), antibiotic therapy often is initiated before the cause of respiratory failure is known. Maintenance of tissue oxygenation and replacement of lost intravascular fluids are the main goals of therapy. Preservation of end-organ perfusion is of utmost importance.

This includes the *administration of supplemental oxygen, control of secretions, treatment of pulmonary infection, control of airway obstruction and limiting pulmonary oedema.* Correction of abnormalities which may lead to respiratory muscle weakness, e.g. hypokalaemia, hypophosphataemia and undernutrition, is also necessary. Oxygen is delivered by a face mask or by nasal cannulae. With these devices, inspired oxygen concentration varies from 35 to 55%, with flow rates between 6 and 10 L.

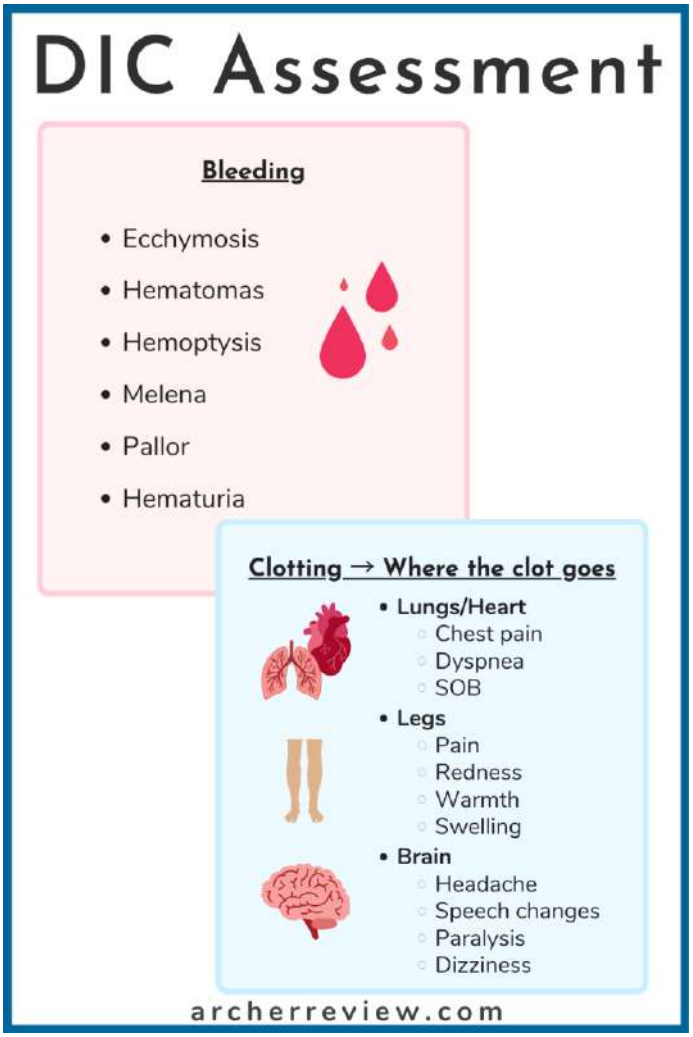
Management of coagulopathy, DIC

Disseminated intravascular coagulation (DIC) is a result of intravascular coagulation activation with microvascular thrombi formation, which causes thrombocytopenia and clotting factors depletion, leading to bleeding and end-organ complications.

Diagnosis

A diagnosis of DIC is made by considering the patient's clinical picture in conjunction with laboratory tests (platelet count, aPTT, PT, fibrin-related markers such as fibrin degradation products (FDP), d-dimer fibrinogen, and antithrombin).

Overt (acute) DIC is characterized by ecchymosis, petechiae, mucosal bleeding, depletion of platelets, clotting factors, and bleeding at puncture sites.



Diagnostic test	Score
Platelet count	>100,000/mm3 = 0 <100,000/mm3 = 1 <50,000/mm3 = 2
Prothrombin time	<3 sec = 0 >3 sec but <6 sec = 1 >6 sec = 2
Fibrin degradation products	No increase = 0 Moderate increase = 2 Strong increase = 3
Fibrinogen level	>1 g/L = 0 <1 g/L = 1
Score calculation • If ≥5 • If <5	Consistent with overt DIC Not likely overt DIC; repeat tests in 1–2 days

The management and treatment of DIC always depends upon the underlying cause. In obstetrical catastrophes, DIC may resolve as a result of prompt delivery, and treatment of sepsis with antibiotic therapy may halt the progression of DIC. Restoration of physiologic anticoagulant pathways with activated protein C in the treatment of sepsis with overt DIC holds promise.

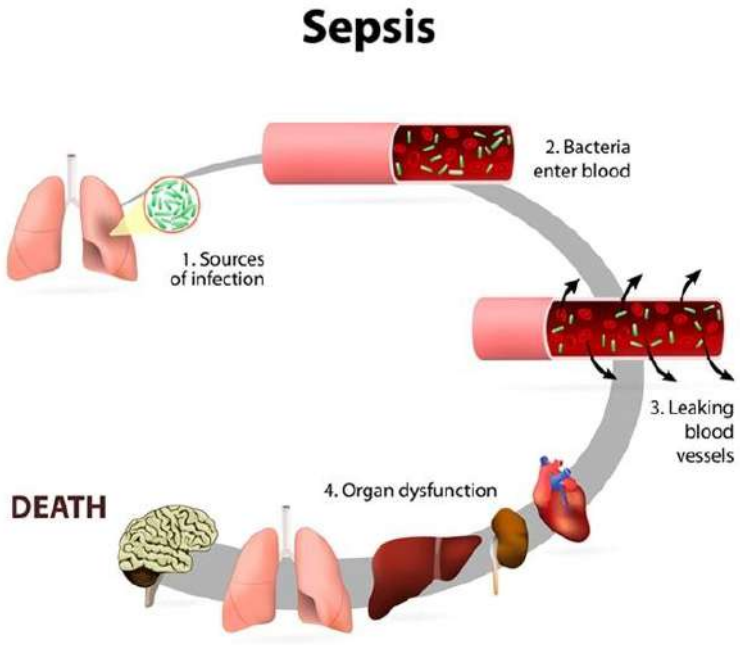
For individuals requiring surgery who are bleeding or at risk for active bleeding, correction of coagulopathy with platelets (<50,000/mm^3), fresh frozen plasma, and cryoprecipitate (fibrinogen <50mg/dL) must be employed. Continued replacement of blood products should be based upon the clinical picture and reassessment of laboratory results.

Management of septicemia

Septicaemia

• Bacteraemia refers to the transient presence of organisms in the blood (generally without causing symptoms) as a result of local infection or penetrating injury.

• Septicaemia is reserved for the clinical picture that results from the systemic inflammatory response to infection. Sepsis is a reaction by the body to severe infection. You may also hear severe infection being referred to as septicaemia. Strictly speaking, septicaemia is an infection of the blood, whereas sepsis refers to the whole body. Septicaemia has a high mortality without treatment, and demands immediate attention.



Clinical features

Fever, rigors and hypotension are the cardinal features of severe septicaemia. Lethargy, headache and a minor change in conscious level may be preceding features.

Investigations

In addition to blood count, serum electrolytes, liver biochemistry and lactate measurement:

• Blood cultures

• Cultures from possible source: urine, abscess aspirate, sputum

• Chest radiography and if necessary abdominal ultrasonography and CT.

Management

The Surviving Sepsis Campaign advocates the use of the care bundle 'Sepsis six', which is associated with decreased mortality, decreased length of stay in hospital and fewer intensive care bed days. The following three diagnostic and three therapeutic steps in the bundle should be instituted within 1 hour of the initial recognition of sepsis.

• Deliver high-flow oxygen

• Take blood cultures

• Administer empiric intravenous antibiotics

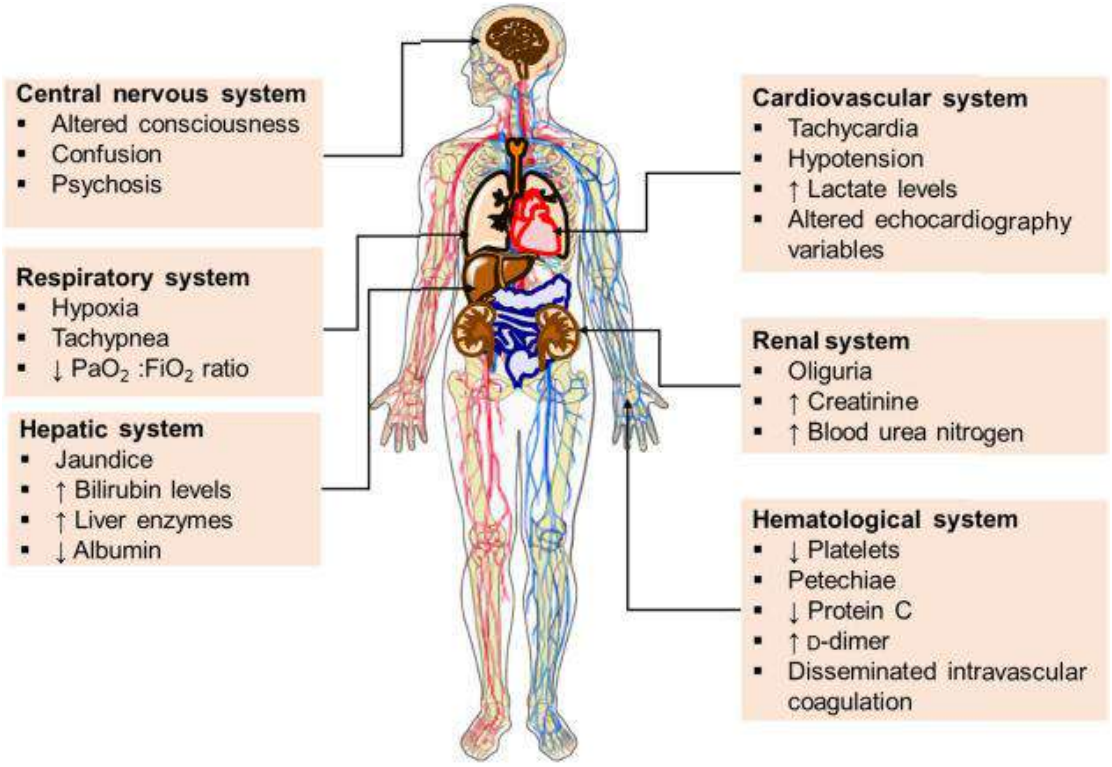
• Measure serum lactate and send full blood count

• Start intravenous fluids

• Commence accurate urine output measurement.

Antibiotic therapy should be appropriate for the probable site of origin of sepsis in accordance with local antibiotic policies. In some cases the source of sepsis will not be immediately apparent and empirical broad spectrum antibiotic cover will be required. Therapy should subsequently be rationalized on the basis of culture and sensitivity results. In severe sepsis and septic shock, significant attention should be paid to fluid resuscitation and often the patient will be best managed in a critical care environment.

Multiple organ failure syndrome (MOFS) Critical illness due initially to a primary failure of one organ system may rapidly escalate to fulminant *multiple organ failure syndrome* (MOFS), particularly when resuscitative management is delayed, inadequate or inappropriate. Mortality correlates with the number of organ system failures and their duration.



The mortality in **multiple organ dysfunction syndrome (MODS)** is high and treatment is supportive, i.e. safeguarding hemodynamics, and respiration. Maintaining adequate tissue oxygenation is a principal target. Starting enteral nutrition within 36 hours of admission to an intensive care unit has reduced infectious complications