

Hospital trainings manual

Surgical ward

For 5th year students

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List of Abbreviations

ABG	Arterial blood gases
ACEI	Angiotensin IIenzyme inhibitor
AS	Aortic stenosis
AF	Atrial fibrillation
ARF	Acute renal failure
BP	(blood pressure)
BUN	Blood urea nitrogen
CABG	coronary artery bypass grafting
CC	Chief complaint
CNS	Central nervous system
CTs	computed tomography
CVA	cerebrovascular accident
CVS	Cardiovascular system
CXR	Chest X-ray
DM	Diabetes mellitus
ECG	electrocardiography
FBC	Full blood count
FBG	Fasting blood glucose
FFP	fresh frozen plasma
GA	general anesthetic
GIS	Gastrointestinal system
GP	General practitioner
GUT	Genitourinary system
HOPI	History Of Present Illness
HRT	Hormonal replacement therapy
IHD	Ischemic heart disease
INR	International ratio
ITU	intensive treatment unit
LFTs	Liver function tests
LIF	Left iliac fossa
LMWH	low molecular weight heparin
LV	Left ventricular
MI	Myocardial infarction
MRCP	Magnetic resonance chollingeo pancreatic
NCEPOD	The national confidential enquiry into patient outcome and death
NPO	Nothing by oral
BD	Twice daily
OCPs	Oral contraceptives
OD	Once daily

PMH	Past Medical History
PONV	Post-operative nausea vomiting
PPI	Proton pump inhibitors
RIF	Right iliac fossa
RS	Respiratory system
RUQ	right upper quadrant
TFT_s	Thyroid function tests
TPN	Total parenteral nutrition
U&E	Urea and electrolytes
USS	ultrasound scan
VRII	variable rate insulin infusion



Patient Data sheet

Patient Name

Age:

Sex:

Date of admission:

Weight:

Height:

BMI:

Chief Compliant (CC):

- History Of Present Illness (HOPI):
- Past Medical History(PMH):
- Past Surgical History(PSH):
- Medication history:
- Drug Allergy:
- Review of other system
 - CNS
 - CVS
 - GIS
 - RS
 - GUT
- **Vital Signs:** BP PR(pulse rate)
RR (respiratory rate) Temp. (Temperature)
- Investigation:
- Lab. Data :
- U/S(ultrasound):

- Morning Tour Night Tour Treatment

- Morning Tour Night Tour Treatment

Problem identification
Medical /pharmaceutical problem

Problem 1:

Problem 2:

Problem 3:

Problem 4:

Problem	pharmaceutical care plan
No.1:	clinical outcomes & objectives

Pharmacist interventions

No.2:	clinical outcomes & objectives
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Pharmacist interventions

No.3:	clinical outcomes & objectives
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Pharmacist interventions

Pre-operative care

1. Pre-operative assessment in the elective surgical patient

- ✚ The aim of pre-operative assessment is to maximize patient safety and minimize the complications of surgery by identifying potential problems and optimizing patients before surgery.
- ✚ The extent of pre-operative preparation depends on several factors, mainly:
 - ✓ The timing of surgery (i.e. how urgent it is);
 - ✓ The nature of surgery (i.e. minor or major procedure);
 - ✓ The patient's past medical history and current status.

History and examination

- ✚ All patients should have a thorough history taken. Many units now have specially designed forms to ensure all areas are adequately explored. It is important to remember that the junior doctor who undertakes the pre-operative assessment may be the only person to take a detailed history and will often uncover important factors regarding the patient's peri-operative care.
- ✚ It is important that if such a critical factor is identified or an abnormal result is found, it is communicated to the consultants' team before surgery, as this may avoid preventable complications or cancellation of the operation.
 - ✓ For example, identification that a patient is on clopidogrel, which must be stopped before elective surgery to reduce the risk of hemorrhage, or identification of undiagnosed aortic stenosis.
- ✚ Pre-operative assessment is therefore a very responsible role with detailed, systematic history covering all areas is required.

Past medical history

- ✚ In particular focusing on cardiovascular disease (including hypertension), respiratory disease, renal disease, diabetes and significant obesity.
- ✚ If a disease is identified, it should be optimally managed pre-operatively, so it is not enough to simply document 'hypertension'.
- ✚ The level of control should be checked and if sub-optimal, steps taken to improve it. This may necessitate liaison with the GP or hospital specialist responsible for the care of this illness and delaying the planned surgery by contacting the consultant's team.

Drug history

- ✚ Particular care is needed to ensure this is accurate with correct drug names, doses and administration times. If necessary, contact the GP for verification.
- ✚ Certain drugs should be flagged up to the consultant's team, as they may have a significant impact on the surgery. These include:
 - ✓ anticoagulants (see Section 1.3.6);
 - ✓ steroids (see Section 1.3.9);
 - ✓ anti-diabetic medication (see Section 1.3.10);
 - ✓ chemotherapeutic agents: blood counts need to be carefully monitored;
 - ✓ ACE-inhibitors (e.g. ramipril) and angiotensin II receptor antagonists (e.g. losartan), which should be omitted on the morning of surgery.
- ✚ The following medications should **not** be stopped prior to surgery:
 - ✓ antihypertensive, especially beta-blockers (e.g. atenolol);

- ✓ other cardiac medications (e.g. digoxin);
- ✓ inhalers, especially steroid inhalers (e.g. beclomethasone);
- ✓ analgesics;
- ✓ Proton pump inhibitors (PPIs) (e.g. omeprazole).

Allergies

- ✚ These should be documented and described in terms of severity.
- ✚ In latex allergy the surgical team should be informed, as these patients should be first on the operating list and may require special equipment.

Social history

- ✚ Important issues include:
 - ✓ Jehovah's Witnesses who refuse blood products;
 - ✓ patients who have inadequate support on discharge where timely input from social services may ensure smooth discharge planning;
 - ✓ Smoking and illicit substance abuse.

Family history

- ✚ A family history of cardiovascular disease or hyperlipidemia may trigger more detailed assessments.

Previous anesthetic problems

- ✚ Previous reactions, airway problems or post-operative nausea or vomiting should be noted.

Examination

- ✚ A full physical examination is required for all patients.

2. Pre-operative assessment in the emergency surgical patient

- ✚ In the emergency setting, preparation for theatre is about optimization to give the patient the best possible chance of a good surgical outcome.
- ✚ In patients with severe comorbidity, NCEPOD recommends:
 - ✓ Communication between surgeon and anesthetist pre-operatively;
 - ✓ Adequate pre-operative investigation;
 - ✓ Appropriate grade of surgeon (minimizing operating time and blood loss);
 - ✓ Adequate resuscitation;
 - ✓ Availability of critical care bed.
- ✚ It should not be forgotten that emergency admissions may require urgent surgery, and so a thorough history and examination should be performed by the admitting doctor.

Pre-operative investigations in emergency patients

- ✚ All emergency general surgical patients should have an appropriate set of blood test results available prior to surgery, including FBC, U&E, LFTs, glucose, clotting screen, group and save or cross-match depending on the type of surgery, their comorbid conditions and the underlying acute illness.
- ✚ All sexually active women of child-bearing age should have a pregnancy test.
- ✚ Patients admitted with an acute abdomen, where perforation of a viscus is part of the differential diagnosis, should have an erect chest x-ray.
- ✚ Any patients with tachycardia and all adults aged 40 over years should have a baseline ECG, as should anyone with a cardiac, hypertensive or respiratory history.
- ✚ All patients should have a simple urinalysis.

- ✚ An ABG may be relevant in patients with significant respiratory disease and those with acute sepsis, pancreatitis or suspected intestinal ischaemia where a metabolic acidosis may develop.

How to manage conditions of special relevance:

Recent myocardial infarction (MI)

- ✚ Avoid elective surgery [unless urgent or coronary artery bypass grafting (CABG)] for >6 months as risk of re-infarction is elevated.
- ✚ Specific relevant investigations:
 - ✓ Up-to-date ECG and echocardiogram.
 - ✓ Continue all normal cardiovascular medications.
 - ✓ Consider stopping anti-platelets 10 days prior to surgery.

Aortic stenosis (AS)

- ✚ Patients with severe AS should not undergo elective surgery without prior consideration of valvular replacement.
- ✚ Specific relevant investigations:
 - ✓ Echo to assess the pressure gradient across the valve and its area—the normal gradient is only a few mmHg.
- ✚ Prophylactic antibiotics may be required, although the indications for this have recently been substantially reduced.
- ✚ Patients may be on anticoagulants.
- ✚ Avoid tachycardia and hypotension as patients are at risk of MI.
- ✚ Diastolic filling is reduced due to left ventricular hypertrophy.

Atrial fibrillation (AF)

- ✚ Can precipitate cardiac failure if inadequately rate-controlled peri-operatively.
- ✚ May be on warfarin.
- ✚ Specific relevant investigations:
 - ✓ ECG to assess rate and rhythm (if paroxysmal);
 - ✓ INR if on warfarin.
 - ✓

Cardiac failure

- ✚ Postpone surgery until stable and optimized on medication.
- ✚ Important to carefully control fluid balance to avoid overload.
- ✚ Specific relevant investigations:
 - ✚ U&Es as diuretics and ACE-inhibitors can cause renal impairment.
 - ✚ CXR to assess for disease severity and fluid overload.
 - ✚ ECG and echocardiogram to assess LV function.

Patients taking anticoagulants

- ✚ If on warfarin, it is important to weigh the risk of stopping the medication against the risk of bleeding intra-operatively, and this risk varies with the indication for therapy.
 - ✓ Patients on warfarin for AF, for example, are not at great risk if they stop taking it and so may simply omit the drug for 5-7 days pre-operatively.
 - ✓ Patients on warfarin for metallic heart valves, however, are at high risk of thromboembolism if the INR is normalized and so should have cover with low molecular weight heparin (LMWH) or IV heparin.

- ✚ In the emergency setting, reversal of warfarin may be required quickly and so drugs such as vitamin K, fresh frozen plasma (FFP) or prothrombin complex concentrate may be used.
- ✚ Anti-platelet medications should be omitted pre-operatively.
 - ✓ Stop clopidogrel 7-10 days pre-operatively.
 - ✓ Stop dipyridamole 7 days pre-operatively.
 - ✓ Omit aspirin on the day of surgery.

Recent cerebrovascular accident (CVA)

- ✚ Avoid elective surgery (unless urgent or carotid endarterectomy) for >6 weeks.
 - ✓ This is because auto regulation of cerebral blood pressure is disrupted following a CVA, and normal ability to cope with fluctuations of cerebral blood pressure caused by anesthetic agents is impaired. This increases the risk of a further peri-operative CVA.

Hyperthyroidism

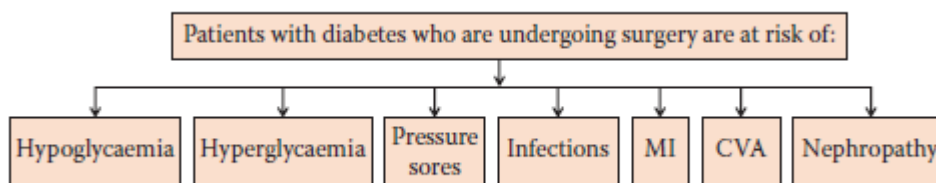
- ✚ Patients with hyperthyroidism are at risk of thyrotoxic crisis, AF and bleeding.
- ✚ Efforts should be made to make the patient euthyroid prior to surgery.
- ✚ Specific relevant investigations:
 - ✓ Thyroid function tests(TFTs), ECG, CXR.
- ✚ AF may resolve on treating the underlying thyroid disease, but patients may require rate control with beta-blockers, digoxin, verapamil or amiodarone.
- ✚ Anti-thyroid drugs may increase bleeding in thyroid surgery, and are often stopped 10-14 days prior to surgery.

Immunosuppression

- ✚ Surgical patients on long-term steroid therapy are at risk of Addisonian crisis.
- ✚ These should be continued throughout surgery, as their use results in adrenal suppression leading to an impaired stress response.
- ✚ Patients unable to take their oral steroids will require IV hydrocortisone.
- ✚ If patients are taking <10 mg of prednisolone daily (or equivalent dose of other steroid), they need no additional steroid cover.
- ✚ If taking >10 mg daily, patients will need 25 mg IV hydrocortisone at induction of anaesthesia and additional cover post-op dependent on the type of surgery.

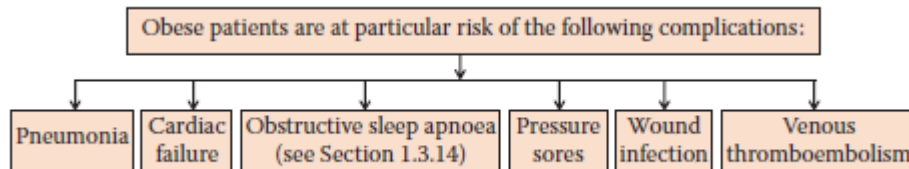
Diabetes mellitus

- ✚ Diabetic patients who undergo surgery are at risk of multiple complications.
- ✚ Ensure the patient is first on the operating list to optimize glycaemic control.
- ✚ Specific relevant investigations:
 - ✓ U&E, blood glucose, ECG.
- ✚ Adjust medication according to the timing and extent of surgery and in consultation with the anesthetist.
- ✚ Patients may require a variable rate insulin infusion (VRII), particularly if starved for a substantial period or if they have decompensated diabetes (e.g. in the presence of sepsis).
- ✚ BMs should be monitored regularly to ensure good control.



Morbid obesity

- ✚ Operations are made more complicated due to:
 - ✓ Harder to manually handle larger patients.
- ✚ Special equipment may be required, such as larger operating tables, beds and special hoists. Not every hospital will have these.
- ✚ Increased prevalence of comorbidities, e.g. IHD, DM, gallstones, etc.
- ✚ Poor airway for anesthetic intubation.
- ✚ Obesity may predispose to multiple complications.
- ✚ Specific relevant investigations:
 - ✓ ECG, blood glucose, respiratory function tests, including spirometry, ABG and CXR.



Renal failure

- ✚ Manage all renal failure patients jointly with a nephrologist.
- ✚ Mild chronic renal failure is common in the elderly surgical patient.
 - ✓ Delay elective surgery to allow optimization and stabilization of the condition.
 - ✓ Avoid nephrotoxic drugs, e.g. gentamicin.
 - ✓ Make sure hydration is maintained pre-operatively.
- ✚ Severe renal failure.
 - ✓ Risk of fluid overload, electrolyte imbalances, metabolic acidosis and anaemia of renal failure.
 - ✓ Uraemia is immunosuppressant so ensure prophylactic antibiotics.
- ✚ Monitor urine output, plasma electrolytes, creatinine, urea and bicarbonate.
 - ✓ Check potassium regularly, as having a general anesthetic (GA) increases the risk of hyperkalaemia.
- ✚ Hemodialysis patients should have dialysis more than 24 hours pre-operatively to allow the effects of heparin to wear off.
- ✚ Patients using peritoneal dialysis having abdominal surgery may need to be converted to hemodialysis pre- and post-operatively.

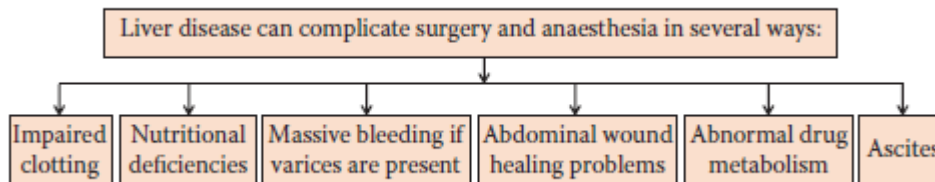
Asthma and COPD

- ✚ Lung function decreases with the use of GA, so alternatives, such as regional anaesthesia, may be considered.
- ✚ Optimize medical therapy with nebulizers, oxygen and steroids as required.
- ✚ Advise smoking cessation 4-8 weeks prior to surgery.
- ✚ Use of chest physiotherapy at least twice a day:
 - ✓ passive, e.g. breathing exercises;
 - ✓ Active, e.g. postural drainage.
- ✚ May need to book a bed on HDU for post-operative non-invasive ventilation.
- ✚ Specific relevant investigations:
 - ✓ Assess lung function using spirometry, ABG and CXR.

Liver disease

Patients with liver disease are at risk of several complications.

- ✚ Patients with liver disease may be classified using the Child-Pugh criteria.



- ✚ Surgical risks vary with the score, and in those with a high score all but the most critical, life-saving surgery are contraindicated.
- ✚ Specific relevant investigations:
 - ✓ FBC, clotting screen, U&E, LFT and bone profile.
- ✚ Patients should have close monitoring of blood sugar, as they have defective glycolysis and gluconeogenesis.
- ✚ Patients with deranged clotting may need vitamin K pre-operatively for several days or require fresh frozen plasma (FFP) peri-operatively.
- ✚ Prophylactic antibiotics are indicated.

Hormonal therapy

- ✚ Patients taking oral contraceptives have an increased risk of postoperative venous thromboembolism because of the combined effects of hormones and the hypercoagulable state, which accompanies surgical stress and postoperative immobility.
The patient should switch to alternative contraceptive methods 4-6 weeks prior to surgery and then restart OCPs after the first menses occurs at least 2 weeks after regaining full mobility following surgery.
Thus the patient is faced with a choice of continuing combined OCPS and receiving thromboprophylaxis in the perioperative period (i.e., SC LMWH and graduated elastic compression stockings) or stopping the pill 4-6 weeks prior to surgery and using another form of contraception (e.g., a progesterone- only pill).
- ✚ Hormone replacement therapy in postmenopausal women may increase the risk of postoperative venous thromboembolism so perioperative thromboprophylaxis is prudent, rather than subjecting the patient to the risk of provoking postmenopausal symptoms by stopping HRT.

Infection

Surgical antibiotic prophylaxis is the appropriate use of preoperative and postoperative antibiotics to decrease the incidence of postoperative wound infections.

Treatment Goals

Surgical Antibiotic Prophylaxis aims to:

- ✚ Decrease the incidence of postoperative wound infections.
- ✚ Decrease the incidence of adverse drug effects in patients undergoing surgery.
- ✚ Decrease the cost of care for surgical patients.
- ✚ Minimize the adverse effects of prophylactic antibiotics on the microflora of the patient and the overall bacterial resistance patterns in a particular institution.

Pathophysiology

Surgical wound infection does not necessarily follow bacterial contamination. The predominant organisms involved are the endogenous microflora at the surgical site. The development of a surgical wound infection is dependent on a complex interaction between the patient's host-defense response, intrinsic bacterial factors, and local tissue factors. Factors which increase the risk of surgical wound infection are as follows:

- 1. Host-Defense Response Factors:** Patients with an underlying host-defense deficit are at increased risk of surgical wound infection (extremes of age, malnutrition, diabetes, corticosteroid therapy, other immunologic deficiency)
- 2. Bacterial Factors:**
 - i. Degree of wound contamination
 - ii. Bacterial virulence
 - iii. Microbial resistance to prophylactic antibiotics
- 3. Local Tissue Factors:**
 - i. Blood supply and tissue hypoxia.
 - ii. Presence of necrotic material.
 - iii. Presence of hematoma.
 - iv. Presence of a foreign body.

Antimicrobial Spectrum

The antimicrobial agent chosen for an individual patient should have activity against the most common pathogens which cause surgical wound infections. The agent does not need to possess antibacterial activity against the entire endogenous microbial flora at the surgical site; the use of agents with an excessively broad spectrum of activity increases the risk of microbial resistance and superinfection without an improvement in effectiveness. Third- generation cephalosporins exemplify this point. Despite their increased antimicrobial activity, these agents have not proven superior to first-generation cephalosporins in any operative procedure.

Timing of Antibiotic Administration

The most common error encountered in surgical prophylaxis is in the timing of antibiotic administration. Antibiotic prophylaxis has contributed to a reduction in superficial wound infection rates. Coverage should be initiated not more than 2 hours (oral route) or 30 min.(IM,IV) before the skin incision is made and, in the absence of gross contamination or overt infection, should not be given beyond 24-48 hrs. after surgery.

Duration of Prophylaxis

Antibiotic administration should be continued for the shortest duration established to decrease the risk of postoperative infections. Antibiotic administration continued beyond 24 hours has not been shown to be superior to shorter duration antibiotic prophylaxis in most surgical procedures. Antibiotic prophylaxis continued beyond 24 hours increases cost, alters the patient's microflora, and adversely affects the bacterial resistance patterns of the institution. Single-dose antibiotic prophylaxis provides the optimal balance of reducing wound infections in most operative procedures while decreasing adverse drug effects.

Postoperative Care

Routine postoperative care include

✚ **Intravenous fluids:** The intravascular volume of surgical patients is depleted by both:

- ✓ insensible fluid losses
- ✓ And redistribution into the third space.

As a general rule, patients should be maintained on IV fluids until they are tolerating oral intake. Extensive abdominal procedures require aggressive fluid resuscitation. Insensible fluid losses associated with an open abdomen can reach 500-1000 ml/hr.

✚ **Medications**

- ✓ Antiemetics
- ✓ Ulcer prophylaxis
- ✓ Pain control
- ✓ Antibiotics
- ✓ anticoagulants

✚ **Laboratory tests should be done after surgery: see appendix 1 table ()**

Postoperative complications

Postoperative complication is classified into:

✚ **Local complications:**

- ✓ Local- involving the operation site itself

✚ **General-affecting any of the other systems of the body:** e.g. respiratory, urological, or cardiovascular complications.

1. Neurologic complications

- A. Perioperative stroke
- B. Seizures
- C. Delirium

2. Cardiovascular complications

- A. Myocardial ischemia and infarction
- B. CHF
- C. Hypertension

3. Pulmonary complications

4. Renal complications: acute renal failure occurs postoperatively can be divided into prerenal, intrinsic renal, and post renal.

- A. Prerenal ARF: results from decreased renal perfusion that might be secondary to hypotension, intravascular volume contraction, or decreased effective renal perfusion.
- B. Renal ARF: the causes include drug-induced acute tubular necrosis, pigment-induced renal injury, radio contrast dye administration, acute interstitial nephritis, and prolonged ischemia from suprarenal aortic cross-clamping.

- C. Post renal ARF: results from obstruction of the ureters or bladder. Operations that involve dissection near the ureters, such as colostomy closure, or total abdominal hysterectomy, have a higher incidence of ureteral injuries. In addition, obstruction of the bladder from an enlarged prostate, postoperative pain and medication administration, or obstructed urinary catheter can occur.

5. Infectious complications

- ✓ Evaluation of fever should take into context the time after operation in which the fever occurs.
 - A. Intra-operative fever: may be secondary to malignant hyperthermia, a transfusion reaction, or a preexisting infection.
 - B. High fever (>39 C) in the first 24 hrs. is commonly the result of a streptococcal or clostridial wound infection, aspiration pneumonitis, or a preexisting infection. Streptococcal wound infections present with severe local erythema and incisional pain. Penicillin G (2 million units IV every 6 hours) or ampicillin (1-2 g IV every 6 hours) is effective therapy. Patients with a severe necrotizing clostridial infection present with systemic toxemia, pain, and crepitus near the incision. Treatment includes emergent operative debridement and metronidazole (500 mg IV every 6 hours) or clindamycin (600-900mg IV every 8 hours).
 - C. Fever that occurs more than 72 hours after surgery has a broad differential diagnosis, including pneumonia, UTI, thrombophlebitis, wound infection, intra-abdominal abscess, and drug allergy.

6. DVT and PE

7. **DM complications:** as DKA and nonketotic hyperosmolar syndrome.

Complications of general anesthesia

1. *Malignant hyperthermia:* is a hyper metabolic disorder of skeletal muscle that is characterized by intracellular by hypercalcemia and rapid adenosine triphosphate consumption. This condition is initiated by exposure to one or more anesthetic-triggering agents, including halothane, and isoflurane.
 - ✓ Signs and symptoms may occur in the operating room or more than 24 hours postoperatively and include tachycardia, tachypnea, hypertension, hypercapnia, hyperthermia, acidosis, and skeletal muscle rigidity.
 - ✓ Treatment involves immediate administration of dantrolene (1mg/kg IV up to a cumulative dose of 10 mg/kg). This attenuates the rise in intracellular calcium. Repeat doses are given as needed if symptoms persist with care monitoring for 24-72 hours.
2. *Laryngospasm:* during emergence from anesthesia, noxious stimulation of the vocal cords can occur at light phases of anesthesia. In addition, blood or other oral secretions can irritate the larynx.
 - ✓ As a result, the vocal cords may be brought into forceful apposition, and the flow of gas through the larynx may then be restricted or prevented

completely. This alone may cause airway compromise or may lead to negative-pressure pulmonary edema.

- ✓ Treatment involves the use of positive-pressure ventilation by mask to break the spasm. Such therapy usually is sufficient. Succinylcholine may be required in refractory cases to allow successful ventilation.

3. *Nausea and vomiting*

4. *Urinary retention*: although very common with spinal anesthesia, occurs after pelvic operations and in conjunction with benign prostatic hypertrophy.

- ✓ Treatment ranges from conservative (early ambulation, having patient sit or stand while attempting to micturate) to aggressive (bladder catheterization).

5. *Hypothermia*: general anesthesia induction causes peripheral vasodilatation, which leads to internal redistribution of heat, resulting in an increase in peripheral temp. at the expense of the core temp. The core temp. then decreases in a linear manner until a plateau is reached. Such hypothermia is more pronounced in the elderly. Hypothermia may provoke cardiac arrhythmia.

- ✓ Treatment includes passive warming during an operation by insulation of all exposed surfaces. In addition, active warming with forced-air convective warmers is effective, but care should be taken in using warmers with patients with vascular insufficiency.

6. *Nerve injury*: can occur secondary to improper positioning of the patient on the operating table or insufficient padding of dependent regions. Such palsies can be long lasting and debilitating.

Parenteral fluid therapy

A. Crystalloids: are solutions that contain sodium as the major osmotically active particle. Crystalloids are relatively inexpensive and are useful for volume expansion, maintenance infusion, and correction of electrolyte disturbances.

1. Isotonic crystalloids: as lactated Ringers solution, 0.9% NaCl distribute uniformly throughout the extracellular fluid compartment so that after 1 hr. only 25% of the total volume infused remains in the intravascular space. Lactated Ringers solution is designed to mimic extracellular fluid and is considered a balanced salt solution. This solution provides a HCO₃ precursor and is useful for replacing GI losses and extracellular fluid volume deficits. In general, lactated Ringers solution and 0.9% NaCl solution can be used interchangeably. However, the last is preferred in the presence of hyperkalemia, hypercalcemia, hyponatremia, hypochloremia, or metabolic alkalosis.

2. Hypertonic saline solutions: alone and in combination with colloids, such as dextran, have generated interest as a resuscitation fluid for patients with shock or burns. These fluids are appealing because, relative to isotonic crystalloids, smaller quantities are required initially for resuscitation. This immunomodulatory effect of hypertonic saline plus dextran may help to prevent widespread tissue damage and multiorgan dysfunction seen after traumatic injury. The possible side effects of hypertonic solutions include hypernatremia, hyperosmolarity, hyperchloremia, hypokalemia.

3. Hypotonic solutions: as D5W, 0.45% NaCl distribute throughout the total body water compartment, expanding the intravascular compartment by as little as 10% volume expansion. They are used to replace free water deficits.

B. Colloid solutions: contain high-molecular weight substances that remain in the intravascular space. Early use of colloids in the resuscitation regimen may result in more prompt restoration of tissue perfusion and may lessen the total volume of fluid required for resuscitation. The use of colloids is indicated when crystalloids fail to sustain plasma volume because of low colloid osmotic pressure(as increased protein loss from the vascular space, as in burns and peritonitis).

1. Albumin preparations: ultimately distribute throughout the extracellular space, although the initial location of distribution is the vascular compartment. Preparations of 25% albumin and 5% albumin expand the intravascular volume by an equivalent amount. Albumin 25% is indicated in the edematous patient to mobilize interstitial fluid into the intravascular space.

2. Dextran: is a synthetic glucose polymer that undergoes predominantly renal elimination. In addition to its indication for volume expansion, it is also used for thromboembolism prophylaxis and promotion of peripheral perfusion. Dextran solutions expand the intravascular volume by an amount equal to the volume infused.

Principles of fluid management

A normal individual consumes an average of 2000-2500 ml of water daily. Daily water losses include approximately 1000-1500 ml in urine and 250 ml in stool. The minimum amount of urinary output that is required to excrete the catabolic end products of metabolism is approximately 800ml. An additional 750 ml of insensible water loss occurs daily via the skin and respiratory tract. Insensible losses increase with hyper metabolism, fever and hyperventilation. Contraindications to fluid management include:

- volume overload
- pulmonary edema
- acid-base disturbances
- fever if pyrogenic
- And electrolyte disturbances.

1. Maintenance: maintenance fluids should be administered at a rate that is sufficient to maintain a urine output of 0.5-1 ml/kg/hr. Maintenance fluid requirements can be approximated on the basis of body weight as follows:

100 mL/kg/day for the first 10 kg.

50 mL/kg/day for the second 10 kg.

20 mL/kg/day for each subsequent 10 kg.

Maintenance fluids should contain Na^+ (1-2 mmol/kg/day) and K^+ (0.51 mmol/kg/day) (e.g., D5/0.45% NaCl+20-30 mmolK⁺/L).

2. Intraoperative fluid management: requires replacement of preoperative deficit as well as ongoing losses. Intraoperative losses include maintenance fluids for the duration of the case, hemorrhage, and third-space losses. The maintenance fluid requirements are calculated as detailed above. Acute blood loss can be replaced with a volume of crystalloid that is 3-4 times the blood loss or an equal volume of colloid or blood. Intraoperative insensible and third-space fluid losses depend on the size of the incision and the extent of tissue trauma and dissection and can be replaced with an appropriate volume of lactated Ringers solution.
3. Postoperative fluid management: sequestration of extracellular fluid into the sites of operative trauma can continue for 12 or more hours after operation. It is released in response to surgery, conserving water. Hypervolemia will cause aldosterone secretion and salt retention by the kidney. Potassium is released by damaged tissues, and the potassium level may be further increased by blood transfusion, each unit containing in excess of 20 mmol/L. If renal perfusion is poor, and urine output sparse, this potassium will not be excreted and accumulates, causing life-threatening arrhythmias. This is the basis of the recommendation that supplementary potassium may not be necessary in the first 48 hours following surgery or trauma. Urine output should be monitored closely and intravascular volume repleted to maintain a urine output of 0.5-1mL/kg/hr.

Postoperative nausea and vomiting

Postoperative nausea and vomiting (PONV) is one of the most common side effects associated with surgical procedures. It can be very distressing for patients, can lead to medical complication and imposes an economic burden. The medical complications of PONV include possible wound disruption, esophageal tears, gastric herniation, muscular fatigue, dehydration and electrolyte imbalance. There is also an increased risk of pulmonary aspiration of vomitus. Aside from the medical complications, PONV can have psychological effects that may result in patients experiencing anxiety about further surgery.

Risk factors associated with PONV

Patient risk factors

1. Gender: the prevalence of PONV is 3 times higher in women than in men. This gender difference is not evident in pre-pubertal children or in the elderly, which indicates that there may be hormonal involvement.
2. Age: children are 2 times more likely to develop PONV than adults .
3. Obesity: fat-soluble anesthetics may accumulate in adipose tissue and continue to be released for an extended period resulting in prolonged side effects, including PONV.
4. Migraine: patients with a history of migraine are more likely to experience PONV.
5. Pre-operative eating patterns: adequate preoperative fasting reduces the risk of PONV, whereas excessive starvation appears to increase the risk. In emergency surgery where there has not been an adequate fast the risk is increased.
6. History of PONV or motion sickness: such patients may have a lower threshold to nausea and vomiting than the rest of the population. Anxiety, due to a previous experience of PONV may lead to the risk.
7. Gastro paresis: patients with delayed gastric emptying secondary to an underlying disease may be at increased risk of PONV.

Procedural risk factors

The type and duration of surgery is a major factor in PONV. Extended surgical procedures are more likely to lead to PONV than shorter operations, and Gynecological, abdominal especially gastrointestinal, laproscopic, ear-nose and throat, ophthalmic surgical procedures predispose to a higher incidence of PONV

Anesthetic risk factors

Certain anesthetic agents have been associated with a higher incidence of PONV than others. Use of opioid analgesics, use of nitrous oxide, use of some inhalation agents and longer procedures and greater depth of anesthesia.

Postoperative risk factors

1. Pain: relief of pain is associated with the relief of nausea, though the use of opioid analgesics may exacerbate the risk because of their known emetic potential.
2. Dizziness; PONV is increased in patients who experience dizziness.
3. Early ambulation: early or sudden movement can increase the risk of PONV, especially if the patients have received opioids.
4. Hypotension: postoperative hypotension is common and can trigger PONV.
5. Premature oral intake: it is generally considered wise to restrict oral intake, and then to recommended small sips of water to minimize the risk of PONV.

Management of PONV

- ✚ Benzamide (metoclopramide): 0.1-0.2 mg/kg IV over 1-2 min.
- ✚ Phenothiazines(prochlorperazine): 12.5 mg IM.
- ✚ Antiserotonine (ondasterone) :4 mg by slow 1v infusion and can be repeated every 6-8 hours, or tropisterone 2 mg by slow IV infusion.

Postoperative analgesia

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. It is a complex process influenced by both physiological and psychological factors.

Effects of postoperative pain

Postoperative pain can affect all organ systems and includes

- ✚ Respiratory: reduced cough, atelectasis, sputum retention and hypoxemia.
- ✚ Cardiovascular: increased myocardial oxygen consumption and ischemia.
- ✚ Gastrointestinal: decreased gastric emptying reduced gut motility and constipation.
- ✚ Genitourinary: urinary retention.
- ✚ Neuroendocrine: hyperglycemia, protein catabolism and sodium retention.
- ✚ Musculoskeletal: reduced mobility, pressure sores and increased risk of DVT.
- ✚ Psychological: anxiety and fatigue.

Analgesic drugs

Many patients are unable to tolerate oral medications immediately after postoperative period.

1. Narcotics: the intermittent administration of IV or IM narcotics has the disadvantage that the narcotics may be given too infrequently, too late, and in insufficient amounts to provide adequate pain control. This may be the only choice in patients who are functionally unable to operate a patient-controlled analgesia device.
 - Morphine, 2-4 mg IV every 30-60 min., or meperidine 50 -100 mg IV every 30-60 min.
 - Tramadol, 100 mg then 50 mg every 10-20 min. during the first hour to total max.250 mg then 50-100mg every 4-6 hours; max.600mg daily.
2. NSAIDs: as diclofenac sodium.25-50 mg after surgery; further doses given after 4-6 hrs. if necessary;max.150 mg in 24 hrs. for 2 days.

Wound management and diabetic foot

✚ **Wound abscess:** - A wound abscess presents all the Celsian clinical features of acute inflammation:

- calor (heat),
- rubor (redness),
- dolor (pain)
- And tumour (swelling).

All to which can be added *functio laesa* (loss of function) and it damages the infected part. Pyogenic organisms, predominantly *Staphylococcus aureus*, cause tissue necrosis and suppuration.

Pus is also composed of dead and dying white blood cells which release damaging cytokines, oxygen-free radicals and other molecules. An abscess is surrounded by an acute inflammatory response, and a pyogenic membrane composed of fibrous exudate and edema, and the cells of acute inflammation. The Granulation tissue (macrophages, angiogenesis and fibroblasts) forms later around the suppuration and this may lead to collagen deposition. If excessive or partly sterilized by antibiotics (antibioma), a chronic abscess may result. Wound abscesses may spontaneously discharge through a surgical incision but most take 7-9 days to form after surgery. Many infections may present after the patient has left hospital. Abscesses may need debridement and curettage with an exploration to break down all loculi before resolution can occur.

Persistent chronic abscesses may lead to sinus or fistula formation. In a chronic abscess, lymphocytes and plasma cells are seen with sequestration and later calcification. Certain organisms are related to chronicity, sinus and fistula formation, e.g. mycobacteria.

Plain or contrast radiographs may not be helpful, whereas CT scan, MRI and isotope scans are usually accurate and may allow guided aspiration without the need for surgical intervention.

✚ **Surgical site infections:**

Surgical wounds are classified into three strata according to the potential risk of microbial contamination.

Class I or clean wounds are those in which only skin microfloras are likely to contaminate the operative field; because no hollow viscous that possesses endogenous microflora is entered, the risk of infection is low (1–4%). A subset of class I wounds are those in which prosthetic material such as mesh, a cardiac valve; although the consequences of wound infection can be dire, often defeating the purpose of the procedure.

Class II clean contaminated wounds are those in which a hollow viscous likely to harbor microbes is entered (gut, biliary tract), such that both skin microflora plus resident microbes may be present in the wound, producing a slightly higher risk of infection (3–6%).

Class III wounds are those in which substantial microbial contamination (e.g., fecal soilage; traumatic heavily contaminated wound) may be present, and the overall wound infection risk is substantial (4–20%), particularly if the skin edges of the superficial wound are opposed.

Wound classification systems are used by the surgeon to decide whether to administer prophylactic antibiotics, whether to select agents with activity against pathogens that are likely to contaminate the wound and cause infection, and whether to close the skin edges of the wound per primum. A series of maneuvers appear, in composite, to reduce the rate of infection of either the superficial or deep portions of the wound or both sites. These include:

- (1) Preoperative patient skin preparation in the form of scrubbing the prospective wound area and showering using a topical microbicide;
- (2) Clipping of hair, but avoidance of shaving the skin of the prospective wound site, particularly in advance of the procedure because microbial proliferation can occur in areas of epidermal damage;
- (3) Surgeon hand scrubbing and patient skin preparation using a topical microbicide immediately before the procedure;
- (4) Instrument sterilization and avoidance of breaks in aseptic technique;
- (5) Use of mechanical preparation, intraluminal antibiotics, or antiseptics for selected procedures to reduce the microbial inoculum within a hollow viscous that will be entered as part of the procedure; and
- (6) Administration of prophylactic antibiotics that possess activity against numerically common, albeit not all, potential pathogens that may contaminate the wound during the operation.

The role of antibiotics in the treatment of wound abscesses:

It is controversial unless there are signs of spreading infection (cellulitis or lymphangitis). Surgical decompression and curettage must be adequate and may allow re-suture without antibiotics but this is also controversial. Delayed primary or secondary suture is safer.

Treatment

Following the trend to discharge patients earlier, many wound infections may be missed by surgeons unless they undertake a prolonged and carefully audited follow up with family doctors. Supportive wound infections take 7-10 days to develop, whereas cellulitis around wounds caused by invasive organisms (such as the beta-hemolytic streptococcus) appears in 3-4 days. Major wound infections with systemic signs or evidence of cellulitis justify the use of appropriate antibiotics.

In severely contaminated wounds, e.g. incisions made for drainage of an abscess, it is logical to leave the skin layer open. Delayed primary or secondary suture is undertaken when the wound is clean and granulating.

Who to close the suitable antibiotic? The choice may be empirical or based on culture and sensitivities of isolates harvested at surgery. Although the identification of organisms in wound 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 but if clinical response is poor by the time sensitivities are known then antibiotics can be changed. This is unusual if the empirical choice of antibiotics is sensible change of antibiotics promotes resistance and risks complications, such as *Clostridium difficile* enteritis.

When taking pus from infected wounds, specimens should be sent fresh for microbiological culture. Swabs should be placed in transport medium but as large a volume of pus as possible is likely to yield more accurate results. If bacteremia is suspected, repeat specimens may be needed to exclude negative results.

Many dressings are now available for use in wound care to help debride open infected wounds, others to absorb excessive exudate or to encourage epithelialization and formation of granulation tissue. Polymeric films are used as incise drapes and also to cover sutured wounds but are not indicated for use in wound infections.

Antibiotics used in treatment and prophylaxis of wound infection

1. Penicillin: - Benzylpenicillin, has proved most effective against Gram-positive pathogens including most streptococci, the clostridia and some of the staphylococci. It is still effective against actinomycosis, which rarely is a cause of wound infection, and may be used to treat spreading streptococcal infections specifically, even if other antibiotics are required as part of therapy of a mixed infection. All serious infections, e.g. gas gangrene, require high-dose intra-venous benzylpenicillin, e.g. 1.2 g 4-hourly.

Ampicillin and amoxycillin. These 3-bactam penicillins are absorbed orally or may be given parenterally. Pharmacodynamically, amoxycillin is superior. Both are effective against enterobacteriaceae, against *E. faecalis* and the majority of group D streptococci, but not species of *Klebsiella* or *Pseudomonas*.

Clavulanic acid is available combined with amoxycillin for oral treatment. This anti beta lactamase protects the amoxycillin from inactivation by beta-lactamase-producing bacteria. It is of considerable value for treating *Klebsiella* strains and beta-lactamase-producing *E. coli* infections, but of no value against *Pseudomonas* strains. Sometimes it is used for localized cellulitis or superficial staphylococcal infection and should be used for infected human and animal bites. It is available for oral or intravenous therapy.

2. Flucloxacillin and methicillin:- These are beta-lactamase-resistant penicillins and are therefore of use in treating staphylococcal 3-bactamase-producing organisms. This is the only reason for using them; and flucloxacillin has poor activity against other pathogens.

3. Cephalosporins: - There are many beta-lactamase-susceptible and beta lactamase-stabbe cephalosporins available. There are three that find a place in surgical practice: cefuroxime, cefotaxime and ceftazidime. The first two are most effective in intra-abdominab skin and soft tissue infections, being active against *S. aureus*, and most entero-bacteria. As a group, the enterococci (*S. faecalis*) are not sensitive to any of the cephalosporins. Ceftazidime, although being active against the Gram-negative organisms and, to a lesser extent, *S. aureus*, is most effective against *P aeruginosa*. These cephalosporins may be combined with an aminoglycoside, such as gentamicin, or an imidazole, such as metronidazole, if guaranteed anaerobic cover is needed.

4. Aminoglycosides: - Gentamicin and tobramycin have similar activity and are particularly effective against the Gram-negative enterobacteriaceae. Gentamicin is effective against many strains of *Pseudomonas*, although resistance develops rapidly, but all aminoglycosides are inactive against anaerobes and streptococci. Serum levels immediately before and 1 hour after intramuscular injection must be taken 48 hours after the start of therapy, and dosage should be modified such that the trough level remains at or below 2.5 mg/L and the peak level should not rise above 10 mg/L. Ototoxicity and nephrotoxicity may follow sustained high toxic levels. They have a marked post antibiotic effect and single large doses are effective and may be safer. Vancomycin is most active against Gram-positive bacteria. It is ototoxic and nephrotoxic. Serum bevels should be monitored but this antibiotic has proved most effective against multi-resistant *staphylococcal* infection and, when given oral-by, it is effective against *C. difficile* in cases of pseudomem-branous colitis.

5. Metronidazole: - is the most widely used member of the imidazobe group and is active against all anaerobic bacteria. It is particularly safe and may be administered orally (up to 600 mg 8-hourly), rectally (up to 1 g suppository 8-hourly) or intravenously (500 mg 8-hourly). Infections with anaerobic cocci and strains of *Bacteroides* and *Clostridia* are effectively treated or prevented by its use. Metronidazole is responsible for the reduction of anaerobic infections after abdominal and pelvic surgery.

6. Meropenem together with Imipenem: - is a member of the carbopenems, which are stable to beta-lactamase. They have useful broad-spectrum anaerobic as Webb as Gram-positive activity but are expensive.

7. The quinolones: - are potent microbicidal agents with action against *Pseudomonas* spp., e.g. ciprofloxacin. Their clinical robe in managing wound infection has not been defined.

Diabetic foot: - Diabetic foot is a common chronic foot problems cause great disability within the diabetic patients. 10-15% of diabetic patients develop foot ulcers and foot related problems are responsible for up to 50% of diabetes related hospital admission.

Precipitating factors of foot ulceration

1. Friction in ill-fitting or new shoes.
2. Ulcerated callus.
3. Self-treated callus.
4. Foot injuries (unnoticed trauma in shoes).
5. Burns.
6. Corn plaster.
7. Nail infection.
8. Foot deformities.

Causes of foot ulceration

1. Neuropathy:
 - A. Peripheral neuropathy: is believed to be the most significant contributor to the development of lower extremity ulcers in diabetic patients through impaired detection of injury from poorly fitting shoes or trauma. Diabetic motor neuropathy is also associated with abnormal weight bearing. The motor neuropathy results in abnormalities such as hammer toes which shifts weight bearing more proximally than normal on the metatarsal heads. Additionally, the dorsum of the toes at the posterior interphalangeal joints is often traumatized by ill-fitting shoes in patients with hammer toes.
 - B. Autonomic neuropathy: leads to failure of sweating and inadequate lubrication of the skin. Dry skin leads to mechanical breakdown that initiates ulcer formation. Autonomic neuropathy also contributes to failure of auto regulation in the microcirculation; therefore, arterial blood will shunt past capillaries into the venous blood flow. This reduces the nutritive blood flow to the skin and predispose to ulcer formation.
2. Ischemia: the microvascular disease seen in diabetic patients also contributes to the development and progression of lower-extremity. These patients should be evaluated for proximal atherosclerotic disease, which may be amenable to intervention, thus improving the chances of healing of the ulcer or healing of an amputation.

Types of foot ulceration

1. **Neuropathic** : clinical features are
 - ✚ Warm with intact pulses
 - ✚ Diminished sensation
 - ✚ Ulceration (usually on tips of toes and plantar surfaces under metatarsal heads).
 - ✚ Sepsis.
 - ✚ Local necrosis
 - ✚ Edema
2. **Ischemic(neuroischemic)**: clinical features:
 - ✚ Pulses less, not warm.
 - ✚ Usually diminished sensation.
 - ✚ Ulceration (often on margins of foot, tips of toes, heels).
 - ✚ Sepsis.
 - ✚ Necrosis or gangrene.
 - ✚ Critical ischemia (urgent attention foot pink, painful, pulse less, and often cold).

- Despite preventive measures, foot ulceration and infection are common and represent a potentially serious problem and ulcers can be secondarily infected by staphylococci, streptococci, Gram-ve bacteria and anaerobic bacteria; infection can quickly lead to cellulitis, abscess formation, and osteomyelitis.
- The reasons for this include incompletely defined abnormalities in cell-mediated immunity and phagocyte function associated with hyperglycemia, as well as diminished vascularization. Hyperglycemia aids the colonization and growth of a variety of organism. Sepsis complicating toe ulcers can lead to in situ thrombosis of the digital arteries, resulting in gangrene of the toe.

Treatment

1. Clean wounds: are treated with conservative debridement and dressing changes, with careful trimming of the calluses and nails.
2. Infected wounds: are diagnosed clinically; excess keratin should be pared away to expose the floor of the ulcer and allow efficient drainage of the lesion. A bacterial swab should be taken from the floor of the ulcer after the callus has been removed. Plain X-rays may show osteomyelitis or gas in the soft tissues when lesions fail to heal or continue to recur. The patient should be instructed to dress the ulcer daily. A simple non-adherent dressing should be applied after cleaning the ulcer with normal saline solution. Patients with superficial ulcers can be treated as outpatients and prescribed appropriate oral antibiotics until the ulcer has healed.
3. Urgent treatment: patients with the danger signs listed below:
 - ✓ Redness and swelling of a foot; this often indicates a developing abscess.
 - ✓ Cellulites, discoloration, and crepitus.

Those patients need to be admitted to the hospital immediately for urgent therapy. They should have bed rest and be started on IV antibiotics. An IV insulin pump may be needed to control blood glucose level. In the 1st 24 hours before bacteriological cultures available, a wide spectrum of antibiotics cover is needed.

Therapy consisting of antibiotics covering aerobic and anaerobic organisms according to culture and sensitivity test may be necessary. This treatment may be adapted when the results of bacteriological culture are available.

Prevention

Prevention remains one of the most important elements in the management of the diabetic foot.

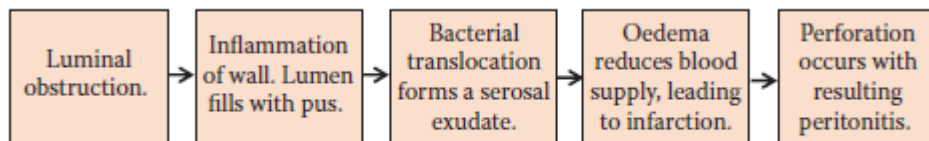
1. Careful selection of footwear.
2. Daily inspection of the feet to detect early signs of poor-fitting footwear or minor trauma.
3. Daily foot hygiene to keep the skin clean and moist.
4. Avoidance of self-treatment of foot abnormalities and high-risk behavior.
5. Promote consultation with a health care provider if an abnormality arises.

Appendicitis

Epidemiology

- ✚ Commonest cause of an acute abdomen and surgical admission in the UK.
- ✚ Approximately one in seven people will have an appendicectomy.
- ✚ It most commonly occurs between 10 and 20 years; it is rare under 3 years of age.

Pathophysiology



Pathological process of acute appendicitis.

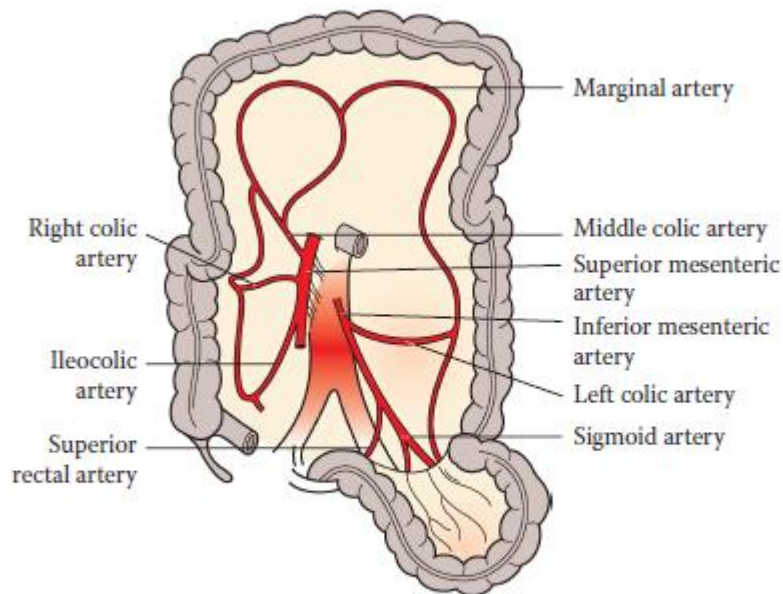
Clinical features

Abdominal pain

- ✚ Initially vague, colicky central abdominal pain.
 - ✓ Visceral pain caused by luminal obstruction of the appendix and stretch of the visceral peritoneum.
- ✚ Localising to the right iliac fossa and becoming constant.
 - ✓ The pain changes as the parietal peritoneum becomes involved.
- ✚ Usually accompanied by a low-grade fever, nausea, vomiting and anorexia.
- ✚ The appendix position varies and can result in different symptoms; for example a pelvic appendix may cause urinary symptoms or diarrhoea.
- ✚ On examination there may be general signs of sepsis:
 - ✓ Usually a low-grade pyrexia initially, which may spike up to 38-39° C in the presence of perforation or abscess formation.
 - ✓ There may be tachycardia, flushing and evidence of dehydration.

Abdominal examination

- ✚ Tenderness over McBurney's point is the usual feature.
 - ✓ There may also be signs of peritoneal inflammation, including:
 - Guarding, tenderness on percussion, pain on coughing or other movement.
 - ✓ Signs of generalised peritonitis may develop as the illness progresses with abdominal rigidity.
- ✚ Rovsing's sign: Pain is felt in the RIF when pressure is applied to the LIF.
 - ✓ There must also be RIF tenderness for this sign to be positive.
- ✚ Psoas sign: The patient keeps his or her hip in flexion to relieve his or her pain.
 - ✓ The appendix is anatomically adjacent to the psoas muscle, which is involved in hip flexion.
- ✚ PR examination may reveal tenderness anterolaterally on the right.



Superior and inferior mesenteric arterial supply of the large intestine.

Diagnosis

- ✚ The diagnosis of appendicitis is a clinical one; however there are some tests that may be useful, particularly where the diagnosis is not clear-cut. These include:
 - ✓ The performance of a full blood count (FBC) can be useful to determine whether or not the patient has a leucocytosis.
 - ✓ A urinalysis to exclude urinary tract infection. Although appendicitis may cause a hematuria or pyuria with associated urinary symptoms.
 - ✓ A pregnancy test in women of child-bearing age is mandatory to rule out an ectopic pregnancy.
 - ✓ An ultrasound scan (USS) in women to exclude tubo-ovarian pathology as the cause of RIF pain
 - ✓ A computed tomography (CT) scan can be useful especially in the elderly where a caecal tumour may be causative, or in the obese where examination is difficult.
 - ✓ Diagnostic laparoscopy allows immediate treatment if appendicitis is confirmed.
 - ✓ Urea and electrolytes (U&E) should also be performed to assess hydration status.

Management

- ✚ Patients are often dehydrated at presentation and so require fluid resuscitation. IV fluids should be continued whilst the patient remains starved for theatre.

Open appendicectomy

- ✚ Usually performed in children.
- ✚ A Lanz incision is used for the best cosmetic result.
- ✚ If the appendix is found to be perforated or gangrenous, then peritoneal lavage is performed to remove any pus or contamination.

- ✚ Most patients can be discharged on the second or third day post-operatively.

Laparoscopic appendicectomy

- ✚ Improves diagnostic accuracy and minimises negative appendicectomy rates.
 - ✓ It is indicated in patients who are unwell but there is question as to the diagnosis, and is particularly indicated in young women.
 - ✓ It is useful in the obese where wound infections are more common and laparoscopic procedures have lower wound infection rates.
- ✚ There is now evidence to suggest that laparoscopic appendicectomy should be performed where expertise is available for this to be done.
- ✚ Laparoscopy has decreased length of stay in hospital, faster return to normal diet and activities and better post-operative pain.
- ✚ If the patient presents late (usually after several days of symptoms) with a palpable appendix mass, he or she requires CT scanning to determine whether there is an associated appendix abscess or a perforated caecal tumour.
 - ✓ The initial management of an appendix abscess is conservative with IV fluids, antibiotics and observation. They may require radiological drainage.
- ✚ If there is deterioration, or frank perforation, surgery may still be required.

Complications

- ✚ Abscess formation; peri-appendicular, pelvic or sub-hepatic.
- ✚ Post-operative collection or abscess.
- ✚ Wound problems, including infection or haematoma.
- ✚ Intestinal obstruction due to adhesion formation within the abdomen.

Thrombosis disorders and Deep Venous Thrombosis

Thrombosis is the development of a 'thrombus' consisting of platelets, fibrin, red cells and white cells in the arterial or venous circulation. If part of this thrombus in the venous circulation breaks off and enters the right heart, it may be lodged in the pulmonary arterial circulation, causing pulmonary embolism (PE). In the left-sided circulation, an embolus may result in peripheral arterial occlusion, either in the lower limbs or in the cerebral circulation (where it may cause thromboembolic stroke).

A. Pulmonary embolism

Pulmonary embolism is a potentially fatal complication of lower limb deep vein thrombosis. A clot from the lower limb veins becomes detached from its site of formation and passes via the inferior vena cava and right heart to the pulmonary arteries. Here it may totally occlude the perfusion to part or all of one or both lungs. This leads to collapse or sudden death in some patients and is a medical emergency. Treatment includes full, immediate anticoagulation with intravenous heparin combined with standard methods of resuscitation. Large emboli may be treated by infusion of fibrinolytic drugs into the pulmonary arteries via catheters inserted via arm or leg veins.

Prevention of pulmonary embolism

In some patients the risk of pulmonary embolism is very great, e.g. a large venous thrombosis in the lower limb where anticoagulation is contraindicated (e.g. following a hemorrhagic stroke). In others, pulmonary embolism occurs despite full anticoagulation with warfarin. Pulmonary embolism may be prevented by the insertion of an inferior vena cava filter which traps large thrombi in its wires and prevents them from occluding the pulmonary arteries. These filters are usually placed by a radiologist via the femoral or jugular vein under X-ray control without the need for an open surgical procedure

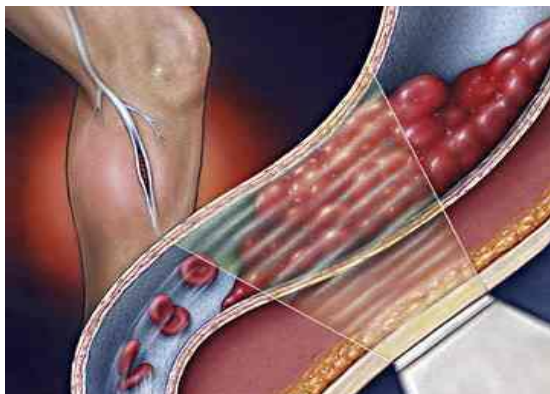
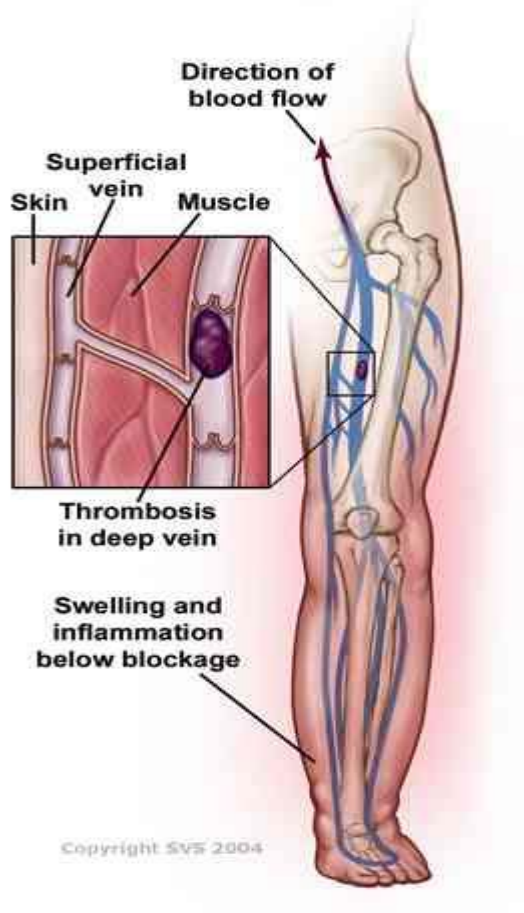
B. Venous thrombosis

Venous thrombosis of the deep veins is a serious life-threatening condition which may lead to sudden death in the short term or to long-term morbidity due to the development of a post-thrombotic limb and venous ulceration. All patients admitted to hospital or being treated for a serious illness should be assessed for the risk of deep vein thrombosis. These patients may be considered at low risk, moderate risk or high risk:

- low risk / young patients, minor illnesses, operations lasting for less than 30 minutes with no additional risk factors;
- moderate risk / patients over the age of 40 years with debilitating illnesses, undergoing major surgery but no additional risk factors;
- high risk / patients over the age of 40 years with serious medical conditions, such as stroke and myocardial infarction, and undergoing major surgery with

additional risk factor, such as a past history of venous thromboembolism, extensive malignant disease or obesity.

Deep venous thrombosis (DVT) is a common cause of death. The true incidence of DVT difficult to determine because its clinical diagnosis can be inaccurate and often occurring in the setting of other critical illness. Approximately 20% of cases of calf DVT propagate to the thigh, and 50% of cases of thigh or proximal DVT embolize. In modern medical practice the most common cause of lower limb venous thrombosis is following hospital admission for treatment of medical or surgical conditions.



Risk factors for DVT

- ✚ Endothelial injury by malignancy:
 - ✓ Adhesion of tumor cells to endothelium .
Chemotherapeutic drugs, such as bleomycin, carmustine, vincristine and doxorubicin can also cause vascular endothelial cell damage.
- ✚ Venous stasis:
 - ✓ This is caused by immobility, venous obstruction, increased venous pressure, and increased blood viscosity.
 - ✓ Surgery and critical illness. E.g.: -Major chest surgery. Similarly, a prolonged non-ambulatory state, such as fracture of the hip requiring bed rest can increase DVT risk.
- ✚ Drugs: Oral contraceptives (OCPs) and estrogen hormone replacement therapy: These have been linked to increased risk of venous thrombus formation.
- ✚ Hypercoagulable states:

Clinical features

DVT can be silent but typically symptoms and signs occur during the second postoperative week, although they may come earlier or later. The patient complains of pain in the calf, and on examination there is tenderness of the calf and swelling of the foot, often with edema, raised skin temp., and dilation of the superficial veins of the leg. This is accompanied by a mild pyrexia. If the pelvic veins or the femoral vein are affected, there is massive swelling of the whole lower limb.



Investigations

- ✚ Venography: it is an invasive procedure.
- ✚ Duplex scanning: it can detect thrombi in all major veins at and above the knee. It is simple and noninvasive.
- ✚ Compression ultrasonography: is highly sensitive in detecting thrombosis of the proximal veins but less sensitive in detecting calf vein thrombosis.

Management

A. Prophylaxis

- ✚ Treat avoidable risk factors.
- ✚ Active mobilization: stimulation of blood flow by encouraging early mobilization reduces the risks.
- ✚ Intermittent calf compression: using inflatable cushions wrapped around the lower legs may be used intra-operatively to reduce the incidence of thrombosis.
- ✚ Mechanical methods are effective (Graduated compression stockings TED) have been shown in clinical trials to reduce the incidence of deep vein thrombosis. Also elevation of the legs to increase venous return are simple and effective.
- ✚ Low-dose unfractionated heparin: this is given SC at 5000 units 2 hrs. before surgery every 8 or 12 hrs. Postoperatively. It should not be used for patients undergoing cerebral, ocular or spinal surgery.
- ✚ Low- molecular weight heparins: such as enoxaparin.
- ✚ Newer medications: such as the direct thrombin inhibitors represent a possible alternative to the unfractionated and LMWHs in the prevention of thromboembolic disease.

B. Treatment

The aim is to minimize the risk of pulmonary embolism and encourage the thrombus to resolve. Pharmacological methods evaluated include: low-dose heparin, low-molecular-weight heparin, dextran and adjusted dose warfarin. In treating an established deep vein thrombosis it is important to make the correct diagnosis. Twenty percent of patients with clinical signs and symptoms of a deep vein thrombosis have normal deep veins. When the diagnosis of DVT is made postoperatively, begin full-dose heparinization (bolus of 5000-10000IU, followed by continuous infusion of 1000-1500IU/hr) with the dose adjusted according to the weight of the patient and controlled by the activated partial thromboplastin time (APTT), if surgical hemostasis is achieved, also most studies suggest that the use of 5000 units of heparin, given subcutaneously two or three times a day, is effective. This should be continued for at least 5 days. Once on therapeutic heparin (aPPT of 1.5-2), warfarin should be initiated and the dose adjusted to maintain an appropriate INR (ie, The INR should be prolonged to between 2.5 and 3.5 times the control value.). Heparin and a therapeutic level of warfarin should overlap for at least 48 hrs. before discontinuing heparin. The aim here is to reduce the risk of a further recurrence of venous thrombosis. Warfarin does not remove the clot from blocked veins and the duration of treatment (usually 3-6 months) is selected to prevent further episodes of venous thrombosis. Patients with recurrent venous thromboembolic problems should be anticoagulated for life.

If edema is present, the patient should remain on bed rest with the affected limb elevated above the level of the heart for several days. The patient should remain on bed rest for 2-3 days even if no pain or edema is present and even if the aPPT is at a therapeutic range to allow fixation of the clot to the vessel wall. Administer 3-6 months of therapy in the case of proximal DVT, assuming that surgery was the only predisposing risk factor.

- ✚ Daltapain sodium is administered at 200 IU/kg/day SC. With a single dose not to exceed 18000 IU.
- ✚ The use of subcutaneous injections of low-molecular-weight heparin for the treatment of deep vein thrombosis is an alternative method of anticoagulation. The dose is based on the patient's weight and treatment given without blood tests to control the dose. This has been found to produce reliable anticoagulation without the risk of hemorrhage. This may in future become a very convenient way to manage patients with acute deep vein thrombosis, either in hospital or at home. Warfarin treatment is commenced at the same time and controlled using the INR in the same way as for the intravenous heparin regime. Enoxaparin sodium is administered at 1mg/kg q12 hrs. SC. Or at 1.5mg/kg/day SC. The single daily dose should not exceed 150 mg. The advantages of low-molecular-weight heparin over standard heparin include once a day administration and a lower risk of bleeding complications, making it more suitable for out of hospital use.

Venous thrombectomy:

Occasionally if massive venous thrombosis in the lower limb can lead to severe impairment in the blood supply to the limb, leading to ischemia and, eventually, gangrene. This is a surgical emergency and requires rapid relief of the venous obstruction. This can be achieved surgically by opening the femoral vein via an incision in the groin and removing all clots from the deep veins of the leg and pelvis. This operation used to be more widely performed on the assumption that it would reduce the severity of post-thrombotic vein damage following a deep vein thrombosis. However, very few surgeons now perform this operation. The more modern treatment of thrombolysis, achieved by passing a catheter into the affected vein and infusing a fibrinolytic drug such as streptokinase or tissue plasminogen activator (TPA), is reducing the need for this operation.

Cholelithiasis

Epidemiology

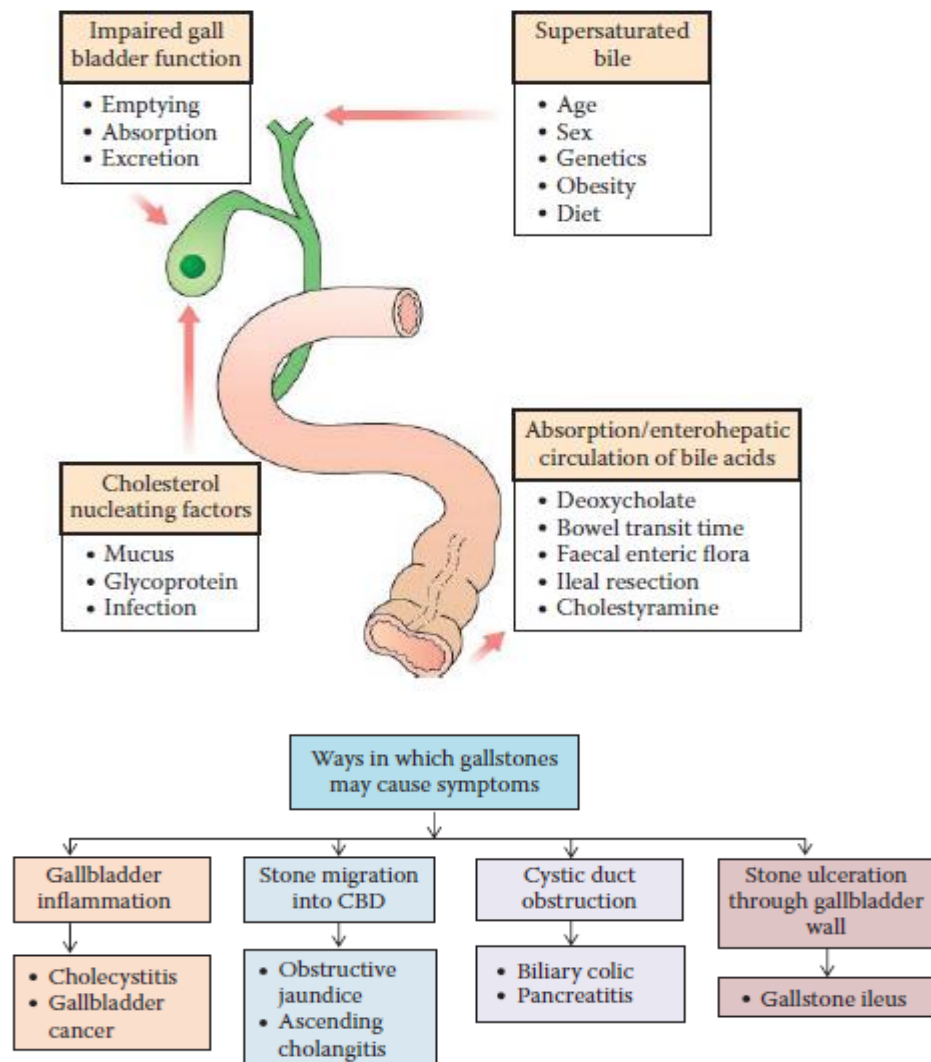
- ✚ Gallstones are common:
 - ✓ 10% of people over 50 years have gallstones.
 - ✓ Incidence increases with age.
- ✚ Affects more women than men 2F:1M.

Aetiology

- ✚ Three types of gallstones:
 - ✓ Cholesterol stones (20%).
 - ✓ Bile pigment stones (5%).
 - ✓ Mixed stones (75%).

Clinical features

- ✚ 80% are asymptomatic.
- ✚ There are several clinical presentations associated with gallstones (see Figure)



Causes of symptomatic gallstones.

Biliary colic

- ✚ Pain occurring when the gallbladder contracts against an obstruction (e.g. a stone in Hartmann's pouch or the cystic duct).
- ✚ Severe right upper quadrant (RUQ)/epigastric pain, lasting for a few hours.
- ✚ Usually precipitated by eating (often fatty foods).
- ✚ May be associated with nausea &/ or vomiting.
- ✚ Patient is usually systemically well (in contrast to acute cholecystitis)

Acute cholecystitis

- ✚ Prolonged gallbladder outlet obstruction, resulting in inflammation due to concentrated bile, initially resulting in chemical cholecystitis.
- ✚ May subsequently be complicated by infection, pus (empyema) or mucus (mucocele).
- ✚ Often a history of previous biliary colic.
- ✚ RUQ/epigastric pain that becomes more severe, constant and localised after a day or two.
- ✚ Associated fever, ↑ WCC, may be rigors and other features of sepsis.
- ✚ On examination there will be tenderness and guarding in the RUQ.
- ✚ Murphy's sign positive.

Chronic cholecystitis

- ✚ Repeated episodes of inflammation resulting in chronic fibrosis and thickening of the entire gallbladder wall.
- ✚ Recurrent episodes of pain with or without fever.

Diagnosis

- ✚ Inflammatory markers (WCC, CRP) will usually be elevated in acute cholecystitis, cholangitis and pancreatitis.
- ✚ LFTs may show an obstructed picture. Serial measurements should be taken if obstructive jaundice is present to ensure its resolution or prompt further treatment if it remains elevated.
- ✚ Ultrasound scan (USS) is used to visualise the gallbladder and biliary tree, allowing diagnosis of stones, inflammation and duct dilatation.
- ✚ Plain abdominal x-ray is useful in gallstone ileus, as there will be evidence of small bowel obstruction, often with pneumobilia.
- ✚ MRCP allows better visualisation of the biliary tree and will demonstrate any gallstones within the CBD that may be causing obstruction (Figure 5.7), which will require removal (e.g. with ERCP or at surgery).
- ✚ ERCP is diagnostic for biliary tree dilatation and CBD stones, and is used therapeutically to remove obstructing CBD stones, insert stents and perform sphincterotomy (sphincter of Oddi).

Treatment:

Supportive measures:-

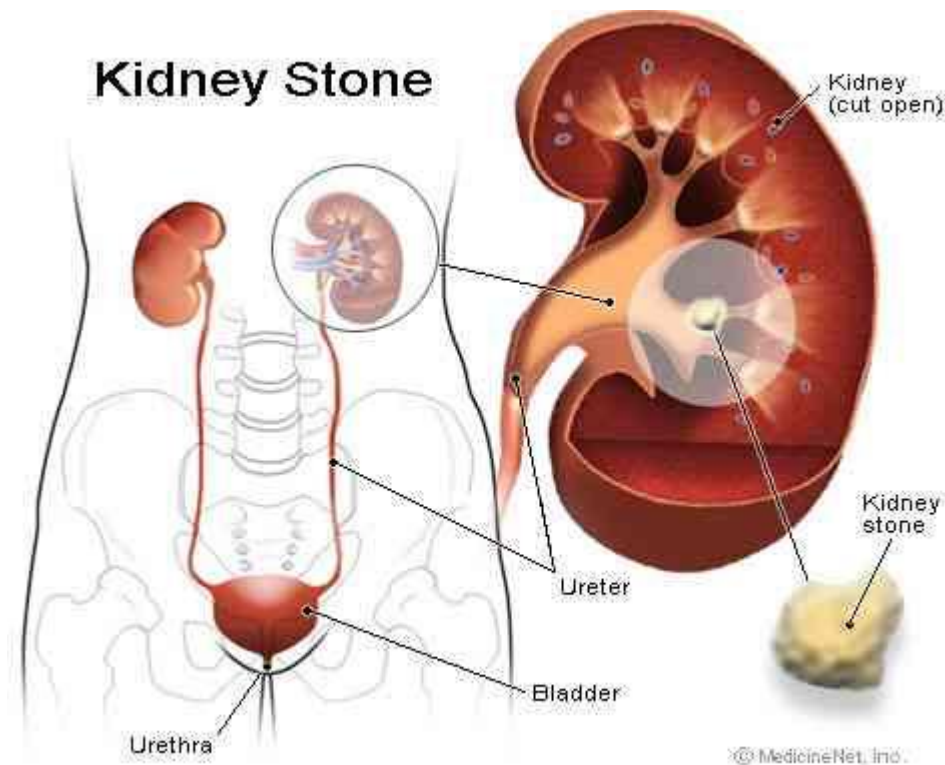
- ✚ Intravenous fluids and analgesia.
- ✚ Antibiotics are required in patients with acute cholecystitis, cholangitis and acute severe pancreatitis.
- ✚ Vitamin K is used to correct coagulopathy caused by obstructive jaundice. ERCP may be used therapeutically in the presence of CBD obstruction
- ✚ Trawling of the duct to remove stones.
- ✚ Sphincterotomy to prevent further obstructive episodes.
- ✚ Insertion of stents to allow bile drainage in difficult cases.
- ✚ Percutaneous transhepatic cholangiography (PTC) is used in patients with severe biliary obstruction and sepsis who are unsuitable for ERCP or where it has been unsuccessful.
- ✚ Insertion of a percutaneous stent may relieve obstruction until sepsis subsides and the patient is well enough for alternative management.
- ✚ Complications include:
 - Bleeding, infection (cholangitis), pancreatitis, perforation.

Cholecystectomy:-

- ✚ Indications:
 - Acute or chronic cholecystitis, recurrent biliary colic, gallstone-induced pancreatitis, biliary peritonitis due to perforation of the gallbladder or previous CBD obstruction.
- ✚ Usually performed laparoscopically.
- ✚ Conversion to open procedure is rare and should occur in <5% of elective cases and <10% of emergency cases.
- ✚ May be a day-case procedure in simple elective cases.
- ✚ There is evidence to suggest that index admission laparoscopic cholecystectomy (i.e. on the patient's first admission with symptoms) is safe, prevents readmission and shortens overall hospital stay.
- ✚ On-table cholangiogram and duct exploration may be performed during laparoscopic cholecystectomy to identify and remove any stones.

Nephrolithiasis

Urinary stones belong to the group of biochemicals different inorganic and organic substances with a crystalline or amorphous structure are the major constituents of the stones. Urinary stones occur in all parts of the renal collecting system. The peak incidence of urinary calculi is in the third to fifth decades. Stones are more prevalent in men than in women and incidence is increased during the late summer months.

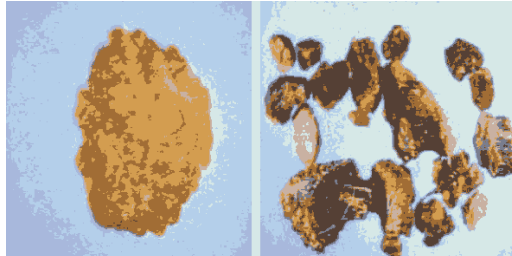


Types of renal stones

1. Calcium stones: are the most common (60%). These stones are composed of mixtures of calcium oxalate (CaOx) and calcium phosphate (CaP), only CaOx or rarely only CaP and higher rates of recurrence in stones with CaP occur. Approximately 50% of people who form a single calcium stone eventually form another within the next 10 years. Calcium stone disease is frequently familial.



3. **Uric acid stones:** are radiolucent and are also more common in men. Half of patients with uric acid stones have gout; uric acid lithiasis is usually familial whether or not gout is present.



3. **Struvite stones:** are composed of a mixture of calcium, ammonium, and magnesium phosphate (triple phosphate stone) are common and potentially dangerous. These stones occur mainly in women or patients who require chronic bladder catheterization and result from urinary tract infection with urease-producing bacteria, usually *Proteus* species. The stones can grow to a large size and fill the renal pelvis and calyces to produce a “staghorn” appearance. They are radiopaque and have a variable internal density.



4. **Cysteine stones:** are uncommon; their radioopacity is due to the sulfur content. They appear in the urine as flat, hexagonal plates.



Clinical features

- Pain is the presenting feature of the greatest majority of kidney stones, but if the calculus is embedded within the solid substance of the kidney it may be entirely symptom free. Impaction of the stone at the peliv-ureteric junction, or migration down the urter itself, produces the dreadful agony of uretric colic; the pain radiates from loin to groin, is of great severity and is accompanied by

typical restlessness of the patient associated with nausea and vomiting, who is quite unable to lie still in bed.

- Haematuria, which may be microscopic or macroscopic, is frequently present.

Investigation

Clinical investigations:

1. *Urine*: is tested for the presence of blood.
2. *Plain abdominal X-ray*: specifically looking at kidneys, urters and bladder will show the presence of stone in 90% of cases.
3. *CT scan*: is the investigation of choice to confirm the diagnosis of renal colic since it is rapid, is more sensitive at detecting a stone, and can diagnose alternative pathologies if present (e.g. torted ovarian cyst, ruptured aortic aneurysom).

Investigation of the underlying cause:

1. *Urine microscopy and culture*: the urine is cultured for bacteria and examined microscopically for the presence of cystine crystals.
2. Analysis of the stone, whether passed spontaneously or removed surgically, should be performed.
3. *Uric acid estimation*: the serum uric acid is raised in gout with its associated uric acid stones.
4. *Serum calcium*: hypercalcaemia(a value above 2.75mmol/L) is associated suspicious of the presence of a parathyroid tumor, although the incidence of stones due to this cause is low.

Complications:

1. *Hydronephrosis*: is a dilatation of the renal pelvis and calyces. It may be secondary to obstruction within the lumen as ureteric calculus; or in the wall as transitional cell tumor or outside the wall as retroperitoneal fibrosis. Hydronephrosis may be without obstruction. It may be unilateral(e.g. calculus stuck in one ureter) or bilateral(e.g. prostatic hypertrophy) with resultant bilateral hydronephrosis.
2. *Infection* : pyelonephritis, pyonephrosis.
3. *Urinary retention*: due to either impaction of calculi in the ureter on each side, or blockage of the ureter in a remaining solitary kidney

Therapeutic strategies

1. Calicum stones

A. Idiopathic Hypercalciuria: This condition appears to be hereditary. In some patients, primary intestinal hyperabsorption of calcium causes transient postprandial hypercalcemia that suppresses secretion of parathyroid hormone, leads to urinary

losses of calcium and renal synthesis of 1,25-dihydroxyvitamin D is increased, enhancing intestinal absorption of calcium. Vitamin D overactivity, either through high calcitriol levels or excess vitamin D receptor, is a likely explanation for the hypercalciuria in many of these patients. Hypercalciuria contributes to stone formation by raising urine saturation with respect to calcium oxalate and calcium phosphate.

Treatment

- For many years the standard therapy for hypercalciuria was dietary calcium restriction. However, recent studies have shown that low-calcium diets increase the risk of incident stone formation and also likely contribute to the low bone mineral density with increased risk of fracture. Low-sodium and low-protein diets are a superior option in stone formers.
- Thiazide diuretics may be used to lower urine calcium and are effective in preventing the formation of stones. . Thiazide-induced hypokalemia should be aggressively treated since hypokalemia will reduce urine citrate, increasing urine calcium ion levels.

B. Hyperuricosuria: About 20% of calcium oxalate stone formers are hyperuricosuric, primarily because of an excessive intake of purine from meat, fish, and poultry. The mechanism of stone formation is probably due to salting out calcium oxalate by urate. A low-purine diet is desirable but difficult for many patients to achieve. The alternative is allopurinol at a dose of 100 mg bid is usually sufficient.

C. Primary Hyperparathyroidism: The diagnosis of this condition is established by documenting that hypercalcemia that cannot be otherwise explained is accompanied by inappropriately elevated serum concentrations of parathyroid hormone. Hypercalciuria, usually present, raises the urine super-saturation of calcium phosphate and/or calcium oxalate. Prompt diagnosis is important because Para-thyroidectomy should be carried out before renal damage or bone disease occurs.

D. Hyperoxaluria: Urine oxalate comes from diet and endogenous metabolic production, with approximately 40-50% originating from dietary sources. Mild hyperoxaluria (50-80 mg/d) is usually caused by:

- Excessive intake of high-oxalate foods such as spinach, nuts, and chocolate.
- In addition, low-calcium diets may promote hyperoxaluria as there is less calcium binding oxalate in the intestine, increasing the amount of oxalate available for absorption.
- Enteric hyperoxaluria is a consequence of small bowel disease resulting in fat malabsorption e.g. jejunoileal bypass for obesity.
- Hereditary hyperoxaluria states are rare causes of severe hyperoxaluria, often greater than 150 mg per day. Patients usually present with recurrent calcium oxalate stones during childhood. Severe hyperoxaluria from any cause can produce tubule-interstitial nephropathy and lead to stone formation.

Treatment

Patients with mild to moderate hyperoxaluria should be treated with:

- A diet low in oxalate and with a normal intake of calcium and magnesium to reduce oxalate absorption.
- Enteric hyperoxaluria can be treated with the oxalate-binding resin cholestyramine at a dose of 8 to 16 g/d, correction of fat malabsorption, and a low-fat, low-oxalate diet.
- Calcium supplements, given with meals, precipitate oxalate in the gut lumen, providing an additional form of therapy.
- Treatment for hereditary hyperoxaluria includes a high fluid intake, neutral phosphate, and pyridoxine (25 to 200 mg/d). Citrate supplementation may also have some benefit. Even with aggressive therapy, irreversible renal failure secondary to recurrent stone formation often occurs. Segmental liver transplant, to correct the enzyme defect, combined with a kidney transplant has been successfully utilized in patients with hereditary hyperoxaluria.

E. Hypocitraturia: Urine citrate prevents calcium stone formation by creating a soluble complex with calcium, effectively reducing free urine calcium. Hypocitraturia is found in 15-60% of stone formers, either as a single disorder or in combination with other metabolic abnormalities. It can be secondary to systemic disorders, such as RTA, chronic diarrheal illness, or hypokalemia, or it may be a primary disorder, in which case it is called *idiopathic hypocitraturia*.

Treatment

Treatment is with alkali, which increases urine citrate excretion; generally bicarbonate or citrate salts are used. Potassium salts are preferred as sodium loading increases urinary excretion of calcium, reducing the effectiveness of treatment. Two randomized, placebo-controlled trials have demonstrated the effectiveness of citrate supplements in calcium oxalate stone formers.

2. Uric acid stones

These stones form because the urine becomes supersaturated with un dissociated uric acid. In gout, idiopathic uric acid lithiasis, and dehydration, the average pH is often below 5.0. Un dissociated uric acid therefore predominates and is soluble in urine only in concentrations of 100 mg/L. Concentrations above this level represent super-saturation that causes crystals and stones to form. Hyperuricosuria, when present, increases super-saturation, but urine of low pH can be supersaturated with un dissociated uric acid even though the daily excretion rate is normal. Plugging of the renal collecting tubules by uric acid crystals can cause acute renal failure.

Treatment

The two goals of treatment are to raise urine pH and to lower excessive urine uric acid excretion to less than 1 g/d.

- Supplemental alkali, 1 to 3 mmol/kg of body weight per day, should be given in three or four evenly spaced, divided doses, one of which should be given at bedtime. The form of the alkali may be important. Potassium citrate may reduce the risk of calcium salts crystallizing when urine pH is increased, whereas sodium citrate or sodium bicarbonate may increase the risk. If the overnight urine pH is below 5.5, the evening dose of alkali may be raised or 250 mg acetazolamide added at bedtime.
- A low-purine diet should be instituted in those uric acid stone formers with hyperuricosuria.
- Allopurinol may be added to patient regimen.
- If hypercalciuria is also present, it should be specifically treated, as alkali alone could lead to calcium phosphate stone formation.

3. Cystinuria and cystine stones

In this autosomal recessive disorder, proximal tubular and jejunal transport of the dibasic amino acids cystine, lysine, arginine, and ornithine are defective, and excessive amounts are lost in the urine. Clinical disease is due solely to the insolubility of cystine, which forms stones.

Treatment

- High fluid intake, even at night, is the cornerstone of therapy. Daily urine volume should exceed 3 L.
- Raising urine pH with alkali is helpful, provided the urine pH exceeds 7.5.
- A low-salt diet (100 mmol/d) can reduce cystine excretion up to 40%.
- Because side effects are frequent, drugs such as penicillamine and tiopronin, which form the soluble disulfide cysteine-drug complexes, should be used only when fluid loading, salt reduction, and alkali therapy are ineffective. Captopril, which has a free sulfhydryl group to bind cysteine, has been used in a limited number of patients with some success.
- Low-methionine diets have not proved to be practical for clinical use, but patients should avoid protein gluttony.

4. Struvite stones

These stones are a result of urinary infection with bacteria, usually *Proteus* species, which possess urease, an enzyme that degrades urea to NH₃ and CO₂. Those in the end will result in the formation of (struvite). Struvite does not form in urine in the absence of infection, because NH₄ concentration is low in urine that is alkaline in response to physiologic stimuli. Chronic *Proteus* infection can occur because of impaired urinary drainage, urologic instrumentation or surgery, and especially with chronic antibiotic treatment, which can favor the dominance of *Proteus* in the urinary tract.

Treatment

- Complete removal of the stone with subsequent sterilization of the urinary tract is the treatment of choice for patients who can tolerate the procedures.
- Irrigation of the renal pelvis and calyces with hemiacidrin, a solution that dissolves struvite, can reduce recurrence after surgery.
- Newer procedures such as lithotripsy and percutaneous nephrolithotomy, alone or in combination, have largely replaced open surgery.
- Antimicrobial treatment is best reserved for dealing with acute infection and for maintenance of sterile urine after surgery.
- For patients who are not candidates for surgical removal of stone, acetohydroxamic acid, an inhibitor of urease, can be used. But acetohydroxamic acid has many side effects, such as headache, tremor, and thrombophlebitis that limit its use.

Hyperthyroidism

Synonymous with 'overactive thyroid' and 'thyrotoxicosis'.

Epidemiology

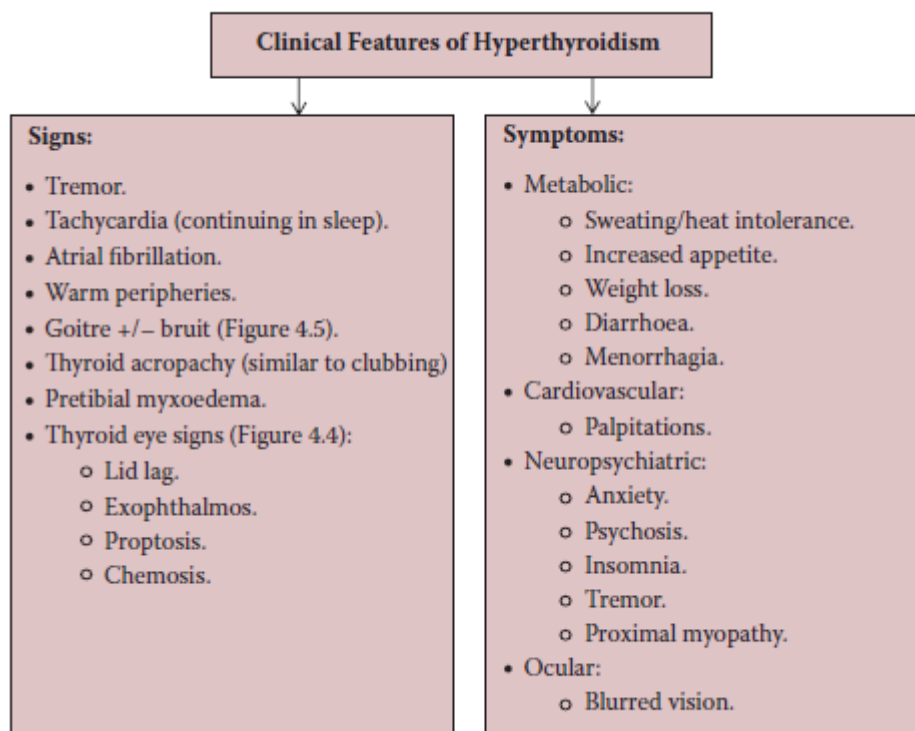
Incidence of 0.2–0.3% of men and 2–3% women.

Aetiology

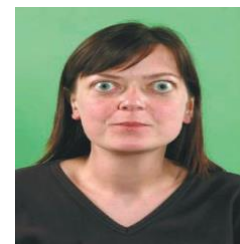
Graves' disease (autoimmune)

Occurs in females, aged 20–40.

Clinical Features of Hyperthyroidism



- ✚ The body produces antibodies that are structurally similar to the binding site of TSH, causing excessive release of T3 and T4 by the thyroid.
- ✚ There is an association with other autoimmune conditions (e.g., type I diabetes mellitus, vitiligo, Addison's disease). And may occur following infection with *Yersinia* or *E. coli*.
- ✚ Eye signs (exophthalmos, lid lag) are more common in Graves' disease.
- ✚ May have a smooth, enlarged thyroid gland.
- ✚ Hyperthyroidism is usually due to an inherent thyroid abnormality; pituitary causes are rare.



Diagnosis

- ✚ Hyperthyroidism is confirmed by thyroid function tests (TFTs):
 - ↓TSH (<0.5 mU/L) , ↑T3 and T4 (except in rare pituitary or gonadal causes).
- ✚ In Graves' disease, thyroid peroxidase (TPO) and thyroglobulin antibodies can be measured.

Imaging

- ✚ Thyroid scintigram uses radioactive iodine to identify overactive areas within the gland ('hot nodules').
- ✚ Ultrasound scans (USS) and computed tomography (CT) allow detail of the gland and identification of compression of adjacent structures.

Biopsy

- ✚ Allows histological analysis.

Treatment

Medication

- ✚ Anti-thyroid drugs can be used definitively or in preparation for surgery.
 - ✓ Carbimazole (first line) or propylthiouracil
 - ✓ Acts by interfering with hormone synthesis.
- ✚ 'Block and replace' (high-dose anti-thyroid drugs to completely suppress hormone production alongside replacement thyroxine) or suppression with lower doses, aiming for lower hormone levels.
- ✚ Control of symptoms, such as palpitations and tremor, may be provided by beta-blockade with propranolol.

Radio-iodine

- ✚ As the thyroid is the only organ to take up iodine, radioactive isotopes (I^{131}) can be given to destroy the thyroid gland.

Surgery

- ✚ May be partial or total excision depending on the cause.
 - ✓ In a subtotal thyroidectomy the posterior rim is left so to avoid damage to the parathyroid glands.
- ✚ Four main indications for surgery over medical therapy:
 - ✓ when a quick, effective treatment is desired (e.g. in young women with Graves' disease);
 - ✓ when anti-thyroid drugs have proved ineffective;
 - ✓ toxic multi-nodular goitre (better medical and cosmetic outcome);
 - ✓ toxic solitary nodule (often resistant to medical treatment).
- ✚ Pre-operative considerations:
 - ✓ Thyroid function should be normalised as much as possible to avoid the dangers of thyrotoxicosis and thyroid storm.
 - ✓ Anti-thyroid drugs are often stopped 10-14 days pre-operatively as they increase vascularity to the gland.

Complications:

- ✓ Hypothyroidism and Hyperthyroidism—Late recurrence due to inadequate excision.
- ✓ Thyroid crisis (rare).
- ✓ There is hyperpyrexia, tachycardia and mania.
- ✓ Recurrent laryngeal nerve injury .May cause slight 'hoarseness' of voice to complete loss of vocal function and critical airway narrowing.
- ✓ Tracheal damage and Hemorrhage.

Burns

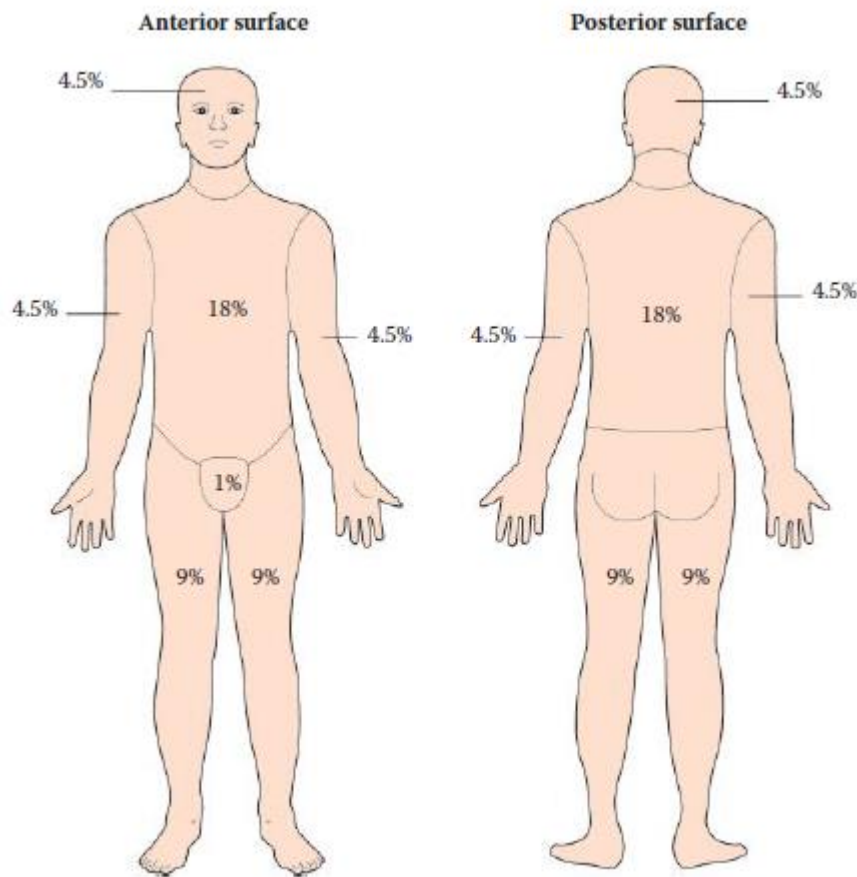
Definition

- ✚ Damage to skin and subcutaneous tissue in response to thermal, electrical, chemical, frictional, cold or radiation injury.
- ✚ By far the commonest type in the UK is a thermal burn, caused mostly by dry burn, e.g. flame, but can be from a wet burn, e.g. scalding.

Assessment of extent of burn

Size of burn

- ✚ The patient's palm is ~1% of his or her total body surface area (TBSA) and can be useful to determine extent of small or very large burns.
- ✚ Wallace's rule of 9's is useful in initial assessment.
- ✚ It is important to use the appropriate chart for the patient's age.

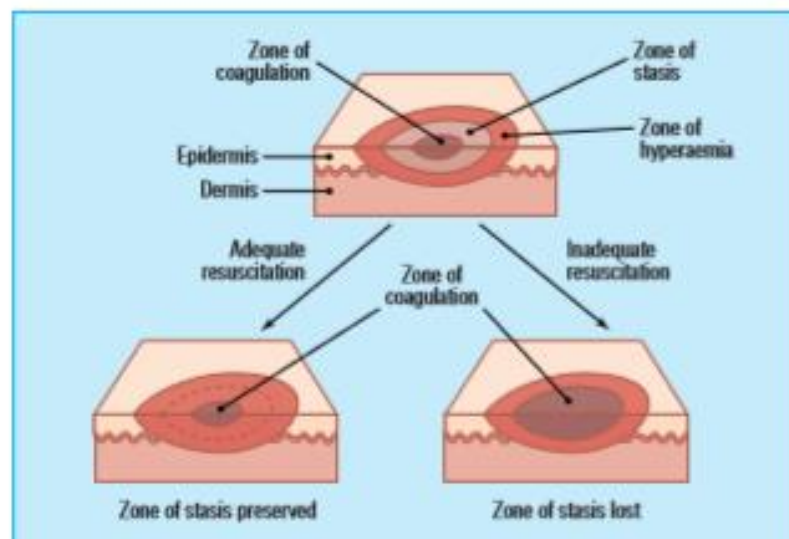


The 'rule of 9's' method for calculating the proportion of the total body Surface area that has been burnt.

Depth of burn

- ✚ Difficult to assess accurately since burns tend to be dynamic and evolve over time.
- ✚ Figures show the characteristics of different burn depths.

	SUPERFICIAL PARTIAL THICKNESS (FIGURE 10.3)	DEEP PARTIAL THICKNESS (FIGURE 10.4)	FULL THICKNESS
Layers involved	Epidermis and upper layers of dermis.	Epidermis and most of dermis.	Epidermis and dermis.
Colour	Pink.	Pink/red.	White/charred.
Blisters	Yes.	Yes.	No.
Capillary refill	Blanches and refills.	Non-blanching.	Non-blanching.
Pain	Yes.	Yes.	No.
Sensation	Normal.	Reduced.	Insensate.
Outcome	Heals well with no scar.	Heals slowly with scarring.	Unlikely to heal if large or heals slowly with severe scarring.



Jackson's burns zones and the effects of adequate and inadequate resuscitation

Pathophysiology:

Burns is now considered one of the most devastating forms of trauma that afflict humans because they induce: local and systemic damage that seriously alter homeostasis. Understanding the pathophysiology of a burn injury is important for effective management. The body's response to a burn injuries result in both local and systemic responses.

✚ **Local response** /The three zones of a burn were described by Jackson in 1947.

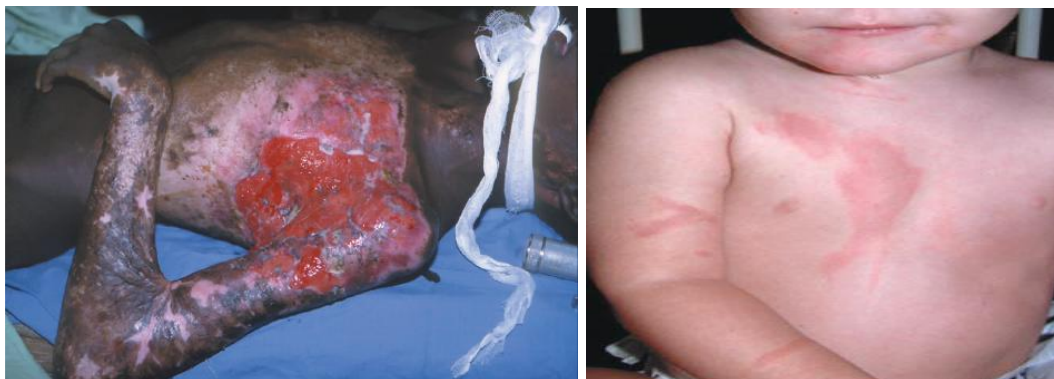
1. **Zone of coagulation:** This occurs at the point of maximum damage. In this zone there is irreversible tissue loss due to coagulation of the constituent proteins.
2. **Zone of stasis:** The surrounding zone of stasis is characterized by decreased tissue perfusion. The tissue in this zone is potentially salvageable. The main aim of burns resuscitation is to increase tissue perfusion here and prevent any

damage becoming irreversible. Additional insults such as prolonged hypotension, infection, or edema can convert this zone into an area of complete tissue loss.

3. **Zone of hyperaemia:** In this outermost zone tissue perfusion is increased. The tissue here will invariably recover unless there is severe sepsis or prolonged hypoperfusion. These three zones of a burn are three dimensional, and loss of tissue in the zone of stasis will lead to the wound deepening as well as widening.

✚ **Systemic response** /The release of cytokines and other inflammatory mediators at the site of injury has a systemic effect once the burn reaches 30% of total body surface area.

1. **Cardiovascular changes:** **Capillary permeability** is increased, leading to loss of intravascular proteins and fluids into the interstitial compartment. Peripheral and splanchnic vasoconstriction occurs. Myocardial contractility is decreased, possibly due to release of tumor necrosis factor. These changes, coupled with fluid loss from the burn wound, result in systemic hypotension and end organ hypoperfusion.
2. **Respiratory changes:** **Inflammatory** mediators cause bronchoconstriction, and in severe burns adult respiratory distress syndrome can occur.
3. **Metabolic changes:** The basal metabolic rate increases up to three times its original rate. This, coupled with splanchnic hypoperfusion, necessitates early and aggressive enteral feeding to decrease catabolism and maintain gut integrity.
4. **Immunological changes:** Non-specific down regulation of the immune response occurs, affecting both cell mediated and humeral pathways.



A mixture of partial-thickness and full-thickness burns.

Initial management of acute burn

✚ Like any emergency, management should take the ABC approach.

MICRO-facts

Signs of potential inhalation injury:

- ✚ Burns on neck, around mouth, on palate or nasal passages.
- ✚ Soot in oropharynx or nostrils.
- ✚ Hoarse voice.
- ✚ Hypoxia.

- ✚ Carboxyhaemoglobin.
- ✚ Stridor, tachypnoea and dyspnoea are late signs.

Airway and breathing

- ✚ If suspected smoke inhalation, early intubation is indicated to prevent airway obstruction and death.
- ✚ If severe airway obstruction, a tracheostomy may be needed.
- ✚ High-flow oxygen through non-rebreathing mask.
- ✚ Perform ABG and check carbon monoxide levels.

Fluid resuscitation

- ✚ Burn injuries lead to large inflammatory exudation of proteins and solutes into the extravascular space.
- ✚ This occurs within the first 6-12 hours post-injury (not post-arrival into the emergency department!) and starts to slow by 36 hours
- ✚ Fluid loss is dependent on size of burn:
 - ✓ $\geq 15\%$ total body surface area (TBSA) in adults and $\geq 10\%$ in children will lead to circulatory shock.
- ✚ Insert a large-bore cannula and catheterize (to monitor output; keep urine output 0.5-1 mL/hour in adults, 1-2 mL/hour in children).
- ✚ Use the Parkland formula to calculate the fluid requirements (usually Hartmann's solution).
 - ✓ Give half in the first 8 hours (from the time of injury), and half in the next 16 hours.

Analgesia

- ✚ Cool wound with running water for 10 minutes.
- ✚ Temporary use of cling-film as dressing reduces evaporation and pain.
- ✚ If small burn, simple oral analgesia may be sufficient.
- ✚ If large burn, IV opiate analgesia will be needed (do not use IM route).

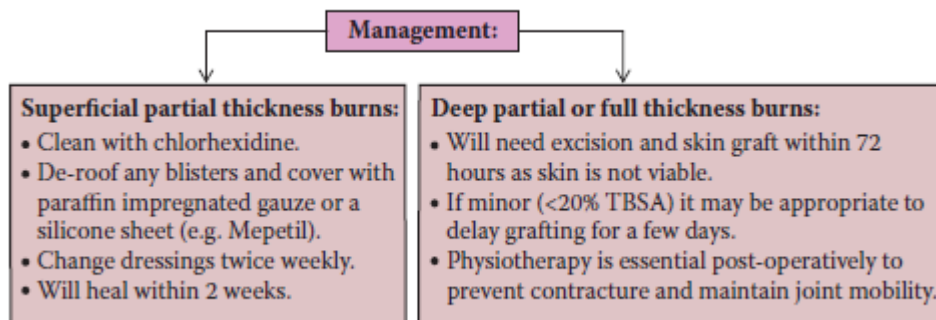
Nutrition

- ✚ Burn injuries lead to a highly catabolic state.
- ✚ If burns $>20\%$ TBSA, insert a nasogastric (NG) tube within 6 hours to prevent stress ulceration of the stomach (Curling's ulcers).

Management of the burn wound

- ✚ Depends on burn depth.
- ✚ Assess if an escharotomy is needed:
 - ✓ An escharotomy is an incision along the full length of the burn to relieve compression due to oedema in circumferential full-thickness burns.
 - ✓ The incision is in the mid-axial line and extends down to the fascia.
 - ✓ Necessary if blood flow to a limb is occluded or respiratory movement restricted.
 - ✓ Escharotomies should be performed in an operating theatre by a burns specialist if at all possible.
- ✚ If assessment indicates that escharotomy may be required, discuss **urgently** with the local burns unit and arrange transfer.

- After escharotomy the principles of management are the same regardless of the size of the burn.



Hernia

Definition

A hernia is the protrusion of an organ or part of an organ through a defect in the wall of the cavity containing it, into an abnormal position. The term is usually used with reference to the abdomen.

Abdominal wall hernias

Most hernias occur as a diverticulum of the peritoneal cavity and therefore have a sac of parietal peritoneum. The common varieties of hernias through the abdominal wall in order of frequency are as follows:

- ✓ inguinal (indirect or direct)
- ✓ femoral
- ✓ umbilical and para-umbilical
- ✓ Incisional (Any abdominal wall incision) .
- ✓ Lumbar, Spigelian (lateral ventral), obturator, perineal or gluteal.

If you or the patient can easily return the contents of the hernia to the abdomen, it is reducible, and you can arrange repair at the patient's convenience. A reducible hernia expands on coughing; any bowel in it may gurgle as you reduce it, and if it contains omentum, it feels doughy.

A hernia may be congenital (existing at birth) or acquired (through increased intra-abdominal pressure from pregnancy, ascites or massive tumour, heavy lifting, coughing, straining to pass urine, or constipation).

There may be several consequences:

- (1) **Irreducibility.** Coughing or straining may push omentum, or a loop of bowel, through the neck of the sac, after which edema may prevent spontaneous reduction. This is more likely the smaller the hernia defect.
- (2) **Obstruction.** A hernia is one of the commonest causes of intestinal obstruction .Again this is more likely the smaller the hernia orifice is. Bowel outside the hernia can rarely also twist and obstruct.
- (3) **Strangulation.** Blood may be able to enter but not leave the organs in a hernia, so that they swell. This is more likely to happen in a hernia with a narrow neck, i.e. femoral, or inguinal. If the swelling persists for >6hrs, the arterial blood supply is cut off and the organs in a hernial sac become ischemic (strangulated)

Obstruction is ultimately as dangerous as strangulation, because, if you leave it, strangulation usually follows. So, be safe, and treat all painful, tense hernias as if they were strangulated .

Aetiology

Hernias occur at sites of weakness in the abdominal wall. This weakness may be congenital as persistence of the processus vaginalis of testicular descent giving rise to congenital inguinal hernia, or failure of complete closure of the umbilical scar. It

may occur at the site of penetration of structures through the abdominal wall as the femoral canal, or the layers of the abdominal wall may be weakened following a surgical incision (incisional hernia), either by poor healing as a result of infection, haematoma formation or poor technique, or by damage to nerves that results in paralysis of the abdominal muscles.

Hernias should also be thought of as portents of other diseases or conditions as they are often associated with pathological increases in intra-abdominal pressure by conditions such as the following:

- ✓ Chronic cough, secondary to chronic bronchitis.
- ✓ Constipation: perhaps due to colonic carcinoma.
- ✓ Urinary obstruction due to prostatic disease.
- ✓ Pregnancy.
- ✓ Abdominal distention with ascites.
- ✓ Weak abdominal muscles as in gross obesity or muscle wasting in cachexia.

Diagnosis:-

The gold standard for hernia diagnosis is a history and physical exam. Patients usually complain of a persistent or intermittent bulge at the site of hernia associated with some degree of discomfort, aggravated by physical exertion. If the hernia is reducible, the pain may wax and wane. A more persistent pain is typical of an incarcerated hernia. If fever, tachycardia, exquisite tenderness on palpation, erythema of the overlying skin, leukocytosis, and obstructive symptoms are present, an irreducible hernia is likely strangulated and warrants immediate operative intervention.

Appendix

Table (I): Perioperative drug management for patients with hypertension

Drug	Day before surgery	Day of surgery	During surgery	After procedure
<i>B-blockers</i>	Usual dose	Usual dose on morning of surgery with sip of water	IV bolus or infusion	Continue IV dose until medication can be taken PO
<i>Calcium channel blockers</i>	Usual dose	Usual dose on morning of surgery with sip of water	IV bolus or infusion	Continue IV dose until medication can be taken PO
<i>ACE-inhibitors</i>	Usual dose	Usual dose on morning of surgery with sip of water	IV formulations	Continue IV dose until medication can be taken PO
<i>Diuretics</i>	Stop day before		IV B-blockers/IV Calcium channel blockers	Restart when patient on oral liquids
<i>Potassium supplements</i>	Stop day before; consider checking potassium level			Restart when patient on oral liquids
<i>Central-acting sympatholytics</i>	Usual dose	Usual dose on morning of surgery with sip of water	Transdermal clonidine/IV methyl dopa	Restart when patient on oral liquids
<i>Peripheral sympatholytics</i>	Usual dose	Usual dose on morning of surgery with sip of water	Any IV formulation	Restart when patient on oral liquids
<i>A-blockers</i>	Usual dose	Usual dose on morning of surgery with sip of water	Any IV formulation	Restart when patient on oral liquids
<i>vasodilators</i>	Usual dose	Usual dose on morning of surgery with sip of water	Any IV formulation	Continue IV dose until medication can be taken PO

Table (II): Perioperative drug management for patients with CAD

Drug	Day before surgery	After procedure	During surgery	After procedure
<i>Nitroglycerin</i>	Usual dose	Usual dose	IV infusion if frank ischemia	Continue IV dose if needed or until medication can be taken PO
<i>Beta-blockers</i>	Usual dose	Usual dose + B-blockers	Usual dose + B-blockers	Usual dose + B-blockers
<i>Calcium channel blockers</i>	Usual dose	Usual dose morning of surgery	Usual dose morning of surgery	Continue IV dose if needed or until medication can be taken PO
<i>Aspirin</i>	Discontinue 1 week before surgery			Restart postoperatively at discretion of surgeon
<i>Ticlopidine</i>	Discontinue 1 week before surgery			Restart postoperatively at discretion of surgeon

Table (III): Perioperative drug management for patients with epilepsy.

Drug	Day before surgery	Day of surgery	During surgery	After surgery	Substitute drug if needed
<i>Phenytoin</i>	Usual dose	Usual dose on morning of surgery with sip of water	IV phenytoin	Continue IV dose until medication can be taken PO	
<i>Phenobarbital</i>	Usual dose	Usual dose on morning of surgery with sip of water	IV phenobarbital	Continue IV dose until medication can be taken PO	
<i>Carbamazepine</i>	PO loading dose of phenytoin or phenobarbital	PO phenytoin or phenobarbital	IV phenytoin or IV phenobarbital	Continue IV dose until medication can be taken PO	phenytoin or phenobarbital
<i>Valproic acid</i>	PO loading dose of phenytoin or phenobarbital	PO phenytoin or phenobarbital	PO phenytoin or phenobarbital	Continue IV dose until medication can be taken P	phenytoin or phenobarbital

Table (IV): Perioperative drug management for patients on NSAIDs

Drug	Day before surgery	Day of surgery	During surgery	After procedure	Substitute drug if needed
NSAIDs with long half-life	Discontinue 1 week before surgery			IM prepration until patient is on oral liquids	Low dose steroids
NSAIDs with short half-life	Discontinue 2-3 days before surgery			IM prepration until patient is on oral liquids	Low dose steroids

Table (V): Perioperative drug management for patients with DM

Drug	Patient for a.m. surgery	Patient for p.m. surgery
Insulin OD	No change to dose.	No change to dose.
Insulin BD	½ a.m. dose, normal p.m. dose.	½ a.m. dose, normal p.m. dose.
Insulin BD separate short and intermediate agents	½total a.m. dose as intermediate-acting insulin, normal p.m. dose.	½total a.m. dose as intermediate-acting insulin, normal p.m. dose.
Insulin 3, 4 or 5 injections daily	Basal bolus regimens: Omit a.m. and lunchtime short-acting insulin keeping basal dose normal. Premixed a.m. regimens: ½ a.m. dose and omit lunchtime dose.	Normal a.m. dose, omit lunchtime dose.
Acarbose or meglitinide (e.g. repalinide)	Omit a.m. dose if NBM.	Give a.m. dose if eating.
Sulphonylurea (e.g. gliclazide)	Omit a.m. dose.	Omit a.m. dose if OD, omit a.m. and p.m. dose if BD.
Pioglitazone or metformin	Normal dosing.	Normal dosing.
DPP IV inhibitor (e.g. sitagliptin) or GLP-analogue (e.g. exenatide)	Omit on day of surgery.	Omit on day of surgery.

Table (vi): postoperative laboratory test.

Tests	Indication for use in patients
CBC	It should be obtained in the postoperative days in any procedure in which significant blood loss occurred.
Serum electrolytes, BUN, creatinine	They are important postoperatively in patients on NPO status or who are receiving large volumes of IV fluids, TPN, or transfusions.
Coagulation studies	They are important in patients who have had insults to the liver or large transfusion requirements.
ECG on daily basis and a series of three troponin I levels 12 hrs. apart	They are appropriate ways to monitor for MI in patients with significant cardiac risk factors.
Chest X-rays	Is necessity after any procedure in which the thoracic cavity is entered or when central venous access is attempted. CXRs on subsequent postoperative days should be considered on an individual basis if significant pulmonary or CV disease is present.

Table (vii): Scores for assessment of liver failure cases.

SCORE	1 POINT	2 POINTS	3 POINTS
Bilirubin	<34 µmol/L	34–50 µmol/L	>50 µmol/L
Albumin	>35 g/L	28–35 g/L	<28 g/L
INR	<1.7	1.7–2.3	>2.3
Ascites	None	Mild	Moderate–severe
Encephalopathy	None	Grade I or II	Grade III or IV

Table viii: Classification of surgical wounds

Wound class	Definition	Example of typical procedures	Wound infection rate(%)	Usual organism
<i>Clean</i>	Nontraumatic, elective surgery; no entry of GI, biliary, tracheobronchial, respiratory, or GU tracts	Wide local excision of breast mass	2	S. aureus
<i>Clean-contaminated</i>	Respiratory, genitourinary, GI tract entered but	Gastroctomy, hysterectomy	<10	Related to the viscus entered

	minimal contamination			
<i>Contaminated</i>	Open, fresh, traumatic wounds; uncontrolled spillage from an unprepared hollow viscus; minor break in sterile technique	Ruptured appendix; resection of unprepared bowel	20	Depends on underlying disease
<i>Dirty</i>	Open, traumatic, dirty wounds; traumatic perforated viscus; pus in the operative field	Intestinal fistula resection	28-70	Depends on underlying disease

Table ix: Recommendations for antibiotic prophylaxis

Nature of operation	Likely pathogens	Recommended antibiotics	Adult dose before surgery
<i>Cardiac; prosthetic valve and other procedures</i>	Staphylococci, conynbacteria, enteric Gram-ve bacilli	Cefazolin Vancomycin cefuroxime	1-2 g IV 1 g IV 1.5 g IV
<i>Vascular: peripheral bypass or aortic surgery with prosthetic graft</i>	Staphylococci, streptococci, enteric Gram-ve bacilli, clostridia	Cefazolin Vancomycin Cefoxitin	1-2 g IV 1-2 g IV 1-2 g IV
<i>Orthopedic: total joint replacement or internal fixation of fractures</i>	Staphylococci	Cefazolin Vancomycin	1-2 g IV 1-2 g IV
<i>Head and neck, entering oral cavity or pharynx</i>	Oral anaerobes, enteric Gram-ve bacilli, staphylococci	Cefazolin Amipcillin-sulbactam Clindamycin Gentamycin	1-2 g IV 1.5-3 g IV 600-900 mg/IV 1.5mg/kg IV
<i>Gasrtoduodenal(high-risk patient)</i>	Enteric Gram-ve bacilli, Gram+ve cocci	Cefazolin	1-2 g IV
<i>Biliary</i>	Enteric Gram-ve bacilli, enterococci, clostridia	Cefazolin Cefoxitin Cefotatan	1-2 g IV 1-2 g IV 1-2 g IV
<i>Colorectal</i>	Enteric Gram-ve bacilli,, anaerobes, enterococci	Oral: neomycin+erythromycin base	1 g of each at 1 PM, 2PM, AND 11 PM the day before

			an 8AM operation
Appendectomy (no perforation)	Enteric Gram-ve bacilli,, anaerobes, enterococci	Cefoxitin Cefotatan	1-2 g IV 1-2 g IV
Vaginal or abdominal hysterectomy	Enteric Gram-ve bacilli ,group B streptocooci, anaerobes, enterococci	Cefazolin Cefoxitin Cefotatan	1-2 g IV 1-2 g IV 1-2 g IV
Cesarean section(high risk patient)	Same as for hysterectomy	Cefazolin	1g IV after cord clamped
Traumatic wound	Staphylococci, group A streptococci, clostridia	Cefazolin or Amipcillin-sulbactam	1g IV q8h 1.5-3 g IV q 6h

Table x: Recommendations for preoperative and postoperative anticoagulation in patients taking oral anticoagulants

Indications	Preoperative	Postoperative
Acute venous thromboembolism Within 1 mo of surgery Within 3 mo of surgery	IV heparin No therapy	
Recurrent venous thromboembolism	No therapy	
Acute arterial embolism (within 30 days)	IV heparin	
Mechanical heart valve	No therapy	
Nonvalvular atrial fibrillation	No therapy	

Table XI: Prophylaxis for DVT and pulmonary embolism

Patient group	Surgery type	Prophylaxis
Low risk	Minor	None
Low or moderate risk	Major	GCS, SC every -12, IPC
High risk	Major	SC every-7 o LMWH
Highest risk	Major	SCevery-8/12 or LMWH +IPC

GCS: graded compression stockings.

IPC: intermittent pneumatic compression.

Low risk: age less than 40 years and no risk factors.

Moderate risk: major surgery and age less than 40 years minor procedure with risk factors or between 40 and 60 years of age.

High risk: major procedure over 40 years, multiple risk factors present, major procedure.

Table IX: Estimation of intraoperative fluid loss and guide for replacement

<i>Preoperative deficit</i>	Maintenance IVF× hr NPO + preexisting deficit related to disease state
<i>Maintenance fluids</i>	Maintenance IVF× duration of case
<i>Third space and insensible losses</i>	1-3 mL/kg/hr for minor procedure(small incision) 3-7 mL/kg/hr for moderate procedure(medium incision) 9-11mL/kg/hr for mextensive procedure(large incision)
<i>Blood loss</i>	1 ml blood or colloid per 1mlblood loss, or 3 ml crystalloid per 1ml blood loss.

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{Remember though the famous wise words of the London surgeon, Sir Astley Cooper: “A surgeon should have an eagle’s eye, a lady’s hands and a lion’s heart”}.