UNIVERSITY OF HONG KONG

Department of Surgery

LECTURES FOR MEDICAL STUDENTS

VOLUME 1

CONTENTS

PRINCIPLES OF SURGERY AND HISTORY TAKING

WHAT IS SURGERY?

"That branch of medicine which treats diseases wholly or in part, by manual and operative procedures"
(Dorland's Illustrated Medical Dictionary)

THE PRINCIPLES OF SURGERY

To use manual and operative procedures when indicated, to palliate or correct congenital anomalies or acquired diseases.

THE GENERAL INDICATIONS FOR SURGERY

1. When surgery is the only effective treatment.
2. When surgery is the treatment of choice.
3. When medical treatment fails or is doomed to fail and surgery gives satisfactory results.

MANUAL PROCEDURES

1. Closed reduction of dislocations and fractures.
2. Manipulation - correction of subluxation.

OPERATIVE PROCEDURES

1. Incise - otomy, craniotomy
   thoracotomy
   laparotomy
2. Excise - ectomy, gastrectomy
3. Divide
4. Anastomose
5. Repair suture, - plasty
6. Anatomical/physiological correction
7. Replacement - parts/organs

THE SCOPE OF SURGERY

1. Treatment of injuries.

2. Removal of foreign bodies, diseased tissue and organ.

3. Anatomical or functional correction.

HISTORY TAKING

1. Aim

To get from the patient concerned an accurate account of his/her complaint and to see this against the findings at physical examination so that a clinical diagnosis can be established.

2. Principles
   a. Allow patient to tell his/her story ("listen to the patient telling one the diagnosis")
   b. Ask direct questions when indicated.
   c. Inspire confidence and express concern (to establish doctor-patient relationship).
   d. Detect non-verbal forms of communication.

3. Format
   a. Presenting complaint.
   b. History of present illness.
   c. Past health including history of previous illness and treatment.
   d. Menstrual history.
   e. Family history
   f. Social and occupational history.

a. Presenting Complaint

Define the main complaint and its duration.

b. History of the Present Illness

i. Ask the patient to tell the story of his/her illness from the beginning, avoid the use of medical terms, describe what they actually feel, enlarge on "important points", clear up any doubt regarding onset and duration, then

ii. Take each symptom in turn and examine it in detail, e.g., pain: site, radiation, severity, timing, character, occurrence, aggravation and relief.

c. Past Health

i. All important previous illnesses should be noted.

ii. Previous drug treatment, surgery, radiotherapy and psychotherapy.

iii. Adverse reactions to drugs - hypersensitivity.

d. Menstrual History

i. Menarche $\frac{\text{Duration}}{\text{Cycle}}$ Menopause

ii. Premenstrual tension, pain during the period, amount of flow

iii. Oral contraceptive

e. Family History

i. Patient's position in the family

ii. Ages of the children

iii. State of health, important illness, cause of death

f. Social and Occupational History

i. Exact nature of his/her occupation

ii. His/her home surroundings

iii. His/her diet, use of alcohol and tobacco

iv. Whether or not he/she has lived abroad

WOUND HEALING

The phenomenon of wound healing is fundamental to all surgery. The characteristic chain of events is found to some degree whenever there is tissue damage. The same general process is found in the organising blood clot, the bed of a chronic ulcer, or the healing of a myocardial infarct.

TYPES OF HEALING

1. Healing by first intention - an incision is closed with sutures and heals without complications.

2. Healing by second intention - an open wound is allowed to close naturally by a combination of wound contraction, connective tissue formation and epithelialisation.

3. Delayed primary closure (secondary suture, healing by third intention) - leaving a contaminated or crushed wound open for a short period of a few days before suture.

PHASES OF HEALING

1. Substrate phase (lag phase) (3-4 days) - a period of intense biological and chemical activity, early inflammation, wound edge hyperaemia and leucocytic infiltration.

2. Proliferative phase (fibroplasia) (2-3 weeks) - dominated by growth and activity of the fibroblast-capillary system, deposition of collagen and ground substance.

3. Maturation phase - fibroblasts and macrophages disappear and vascularity decreases, collagen remodels.

ELEMENTS OF HEALING AND REPAIR

1. Epithelialisation - process in which surface covering of a wound is restored by a process of cell multiplication and migration.

2. Contraction - a natural process by which the edges of an open wound gradually close together. Distinguish from contracture.

3. Connective tissue formation - process by which main body of the wound is united. Strength of the wound is dependent on this. The role of the fibroblast-capillary system. Key organ of repair. This new vascular connective tissue is called granulation tissue.

4. Reformation of tissue - only epithelium, endothelial tissue and bone can regenerate. Another example, reformation of blood after loss or hypertrophy of kidney after removal on one side.

INCISED WOUND

1. Mechanical aspects
   a. breaking strength
   b. bursting strength
   c. load-extension curves for wounds

2. Histological aspects
   a. vascular events
   b. rbc, neutrophils, macrophages
   c. epidermal migration
   d. fibroblasts, collagen

3. Collagen chemistry
   a. configuration
   b. formation
   c. metabolism

4. Ground substance (mucopolysaccharides)
   a. chemistry
   b. synthesis

THE OPEN WOUND

1. Epithelialisation
2. Contraction

LOCAL AND SYSTEMIC FACTORS AFFECTING WOUND HEALING

1. Local

   a. surgical technique
      - gentle handling of tissues
      - meticulous haemostasis-diathermy, ligatures
      - prevention of dead space
        layered closure
        external pressure
        drainage
      - avoid tissue necrosis
   b. blood supply
   c. mechanical stress
   d. suture materials
   e. suture techniques
   f. radiation
   g. infection

2. Systemic

   a. age
   b. malnutrition
   c. vitamin deficiency
   d. zinc deficiency
   e. trauma, hypovolaemia, hypoxia
   f. anaemia
   g. uraemia
   h. malignant disease
   i. jaundice
   j. corticosteroids
   k. cytotoxics, antimetabolites

REPAIR OF SPECIAL TISSUES

1. Bowel

2. Urinary tract

3. Blood vessels

PROBLEMS OF SKIN WOUND HEALING

1. Hypertrophic scars contractures

2. Keloids

3. Infection

CONTROL OF HEALING

1. Collagen synthesis

2. Collagenolysis

3. Intermediary metabolism of collagen

FLUID, ELECTROLYTES AND NUTRITION

FLUID AND ELECTROLYTES

1. Homoeostasis in Health + Disease

a. Fluid
b. Electrolytes
c. Acid Base
d. Energy Substrate Utilisation

What is in balance should be kept in balance.
What is not in balance should be brought into balance.
What is lost should be replaced, and often more.

2. Body Fluid and Compartments

Water - 60-65% body weight (60 kg man)

1.	Intracellular Fluid Space (ICF)	40%	24 litres
2.	Extracellular Fluid Space (ECF)	20%	12 litres
	Interstitial Fluid Space	16%	9.6 litres
	Intravascular Fluid Space (Plasma)	4%	2.4 litres

[Blood with a haematocrit of 40 would give a volume of 4 litres]

Intravascular space, although small, is the most important and must be maintained at all costs.

Dynamic state of the compartments.

c. Third space is a collection of ECF that is not functionally available to normal mechanisms maintaining fluid + electrolyte balance e.g., intestinal content in bowel obstruction, ascites.

3. Electrolyte Distribution

	ICF Concentration	ECF Concentration	ICF	ECF	Other	Total
Na	10 mmol/L	140 mmol/L	10%	50%	40%	4000 mmol
K	150 mmol/L	4 mmol/L	98%	2%		3000 mmol
Cl	-	105 mmol/L	1%	99%		2000 mmol
PO	60 mmol		99%	1%		1500 mmol

SODIUM is the most important ECF ion and provides most of the ECF osmotic pressure.
POTASSIUM is the most important ICF ion and provides most of the ICF osmotic pressure.
Cell membrane is permeable to all ions.
Gradients exist because of energy requiring active transport.

4. Osmotic Pressure

Water freely distributes through all compartments subject to osmotic pressure.

Measurement of osmolality of one compartment reflects the osmolality of all compartments.

Osmotic effect depends on the number of particles and not on charge, valence or molecular size.

$$\text{Estimate of serum osmolality} = [Na] \times 2 + \frac{\text{Urea N}}{2.8} + \frac{\text{Glucose}}{18}$$

Normal osmolality 280 - 295

Starling's Law

Protein is not freely diffusable between capillaries and interstitial space. The osmotic pressure exerted is balanced by hydrostatic pressure.

Permeability of capillaries can vary under circumstances. Lymphatics play a major role in returning proteins into the circulation.

5. Regulation of Volume and Sodium

ADH regulates both ECF volume and osmolality by excreting free water.

Aldosterone regulates Na excretion and is increased by
a. ↑ serum K
b. ↓ B.P.
c. ↑ ACTH
d. ↑ renin, due to ↓ renal blood flow

The kidney is the ultimate regulator.

6. Disorders of Volume, Sodium and Potassium Homoeostasis

a. Monitor of intravascular volume
- central venous pressure
- pulmonary artery wedge pressure
- left atrial pressure
- end diastolic left ventricular volume

Depletion of intravascular volume
- haemorrhage
- loss to third space
- dehydration etc.

Expansion of intravascular volume
- congestive heart failure
- overtransfusion etc.

b. Monitor of total body fluid
- body Weight
- relates to intravascular space and ECF

Clinical signs of volume depletion

minor (1.5 litres or less) thirst

moderate (1.5 litres - 4 litres) marked thirst, dry mouth, absence of sweat, furred tongue, urine specific gravity increased, orthostatic hypotension, collapsed neck veins, decreased turgor

marked (4 litres or more) pre-renal oliguria, hypotension, tachycardia, increased haematocrit

Clinical indications of volume excess
i. dilated neck veins
ii. pulmonary oedema + effusion
iii. peripheral oedema
iv. tachycardia + gallops
v. hepatomegaly

c. Monitor of sodium
serum sodium - does not reflect total body sodium
- only sodium relative to H_2O in the vascular space

	↑ Volume	↓ Volume
↑ Na Hypernatraemia	e.g. hyperaldosteronism	e.g. osmotic diuresis, excess sweating, G.I. loss
↓ Na Hyponatraemia	e.g. - congestive heart failure - inappropriate ADH	e.g. excess diuretics, third space loss

d. Monitor of potassium
99% K in ICF
Potassium in serum only 1/30 potassium in cell
Serum potassium again does not reflect total body potassium

- Na + volume has higher priority in renal regulation
Aldosterone ↑ Na retention
↑ K excretion

- at normal pH, a drop in serum K indicates a significant deficit of total body K

- H^+ balance significantly affects K distribution
  acidosis → K shift out of cells
  alkalosis → K shift into cells

- insulin/glucose promotes K shift into cells

- anabolism → ↑ requirement of K
  catabolism → ↓ total body K
  tissue trauma → ↑ serum K

Hyperkalaemia can cause - atrial standstill, ventricular fibrillation
- paralysis

Common causes - renal failure, excess therapy, tissue trauma, haemolysis, acidosis

Hypokalaemia can cause - muscle weakness
- ileus
- polyuria
- ventricular arrhythmia, especially with digitalis

Common causes - G.I. loss, diuretics, alkalosis

7. Calcium, Phosphate, Magnesium

Serum Ca^{++} and ionic Ca^{++}
↑ H^+ → ↑ ionic Ca^{++}
↓ H^+ → ↓ ionic Ca^{++}
↑ albumin → ↑ total Ca^{++}
↓ albumin → ↓ total Ca^{++}

8. Principles of Management

a. Deficit - nature, amount
b. Daily requirement - sensible and insensible loss
c. Ongoing loss - nature, content, amount
d. Replace with appropriate solutions

ACID-BASE BALANCE

Homoeostasis - most enzymatic process operate within a narrow pH range.
Normal metabolism produces 15,000 mmol H^+/day

1. 3 mechanisms for regulation

   a. Body buffer system
      i. haemoglobin
      ii. HCO_3^-
      iii. tissue + bone

   b. Renal mechanism - ultimate regulator reabsorb HCO_3^-, excrete H^+

   c. Respiratory mechanism - transient regulator

$$H^+ + HCO_3^- = HCO_3 = H_2O + CO_2$$

2. Body $pH = 6.1 + \log \frac{HCO_3 \text{ (controlled by kidney)}}{pCO_2 \text{ (controlled by lung)}}$

Henderson-Hasselbalch equation

Arterial blood gas			
	pH	7.35 - 7.45	
	pCO_2	4.7 - 6.0	pKa
	pO_2	10 - 13	pKa
	Base excess	0 $\pm$	mmol/L

3. Acidosis vs alkalosis
   The body tolerates acidosis better than alkalosis.
   The oxyhaemoglobin dissociation curve operates more efficiently in acidosis.
   Acidosis is easy to treat, alkalosis almost impossible to treat.

4. a. Metabolic acidosis -

↑ production	e.g. diabetic ketoacidosis
↓ excretion	e.g. renal failure
↑ loss of HCO_3	e.g. diarrhoea, fistula

   b. Metabolic alkalosis -

↑ loss H^+ G.I.	e.g. nasogastric suction
↑ loss H^+ Renal	e.g. hypoparathyroidism
↑ HCO_3	e.g. milk alkali, transfusion contraction of ECF

   c. Respiratory acidosis -

↓ respiratory drive	e.g. opiates
↓ ventilation	e.g. flail chest
↓ gas exchange	e.g. pulmonary oedema

   d. Respiratory alkalosis

↑ respiratory drive	e.g. hypoxia, CNS, psychogenic, sepsis, hypermetabolic state

Acid-base disorders rarely occur in pure state. Compensatory mechanisms always set in.

	pH	pCO_2	HCO_3	Base excess	Immediate compensation	Long term compensation	Treatment
Metabolic acidosis	↓	-	↓		respiratory	renal	HCO_3
Metabolic alkalosis	↑	-	↑		respiratory	renal	-
Respiratory acidosis	↓	↑	-	-	-	renal	ventilate
Respiratory alkalosis	↑	↓	-	-	-	renal	↑ dead space

NUTRITION

1. Basal energy expenditure — 25 cal/kg/day
   a. Activities energy expenditure
   b. Stress energy expenditure
2. Metabolic response to starvation
3. Metabolic response to trauma
4. Components of nutrition
   a. Carbohydrates/glucose
   b. Protein/amino acid
   c. Fat/fatty acid
   d. Fluid and electrolytes
   e. Vitamins and trace elements

MEANS OF NUTRITION

1. Enteral
2. Parenteral

SURGICAL INFECTIONS

DEFINITIONS

1. Infection - Inflammation resulting from infective organisms

2. Acute Inflammation - Reaction of vascular and supporting tissue to insult.

3. Chronic Inflammation - Process of inflammation going on side by side with healing process.

4. Anaerobic Infections - Infection with facultative or obligatory anaerobic organisms.

TYPES OF INFECTIONS

1. Cellulitis - Spreading inflammation along subcutaneous and fascial planes.

2. Abscess - Collection of pus (acute or chronic).

3. Empyema - Collection of pus in a natural cavity.

4. Bacteraemia - Carriage of organisms by blood.

5. Pyaemia - Clumps of organisms and infective material carried by blood.

6. Septicaemia - Multiplication of organisms in blood.

CELLULITIS

Organisms - Strept pyogenes; anaerobes or intestinal organisms.

Entrance - Small wound

Predisposition - Diabetes; alcoholism; renal insufficiency; steroids.

Treatment - Rest; elevation; antibiotics; treatment of predisposing factors; treatment of complications.

Complications - Abscess; gangrene; septicaemia; pyaemia.

Special sites

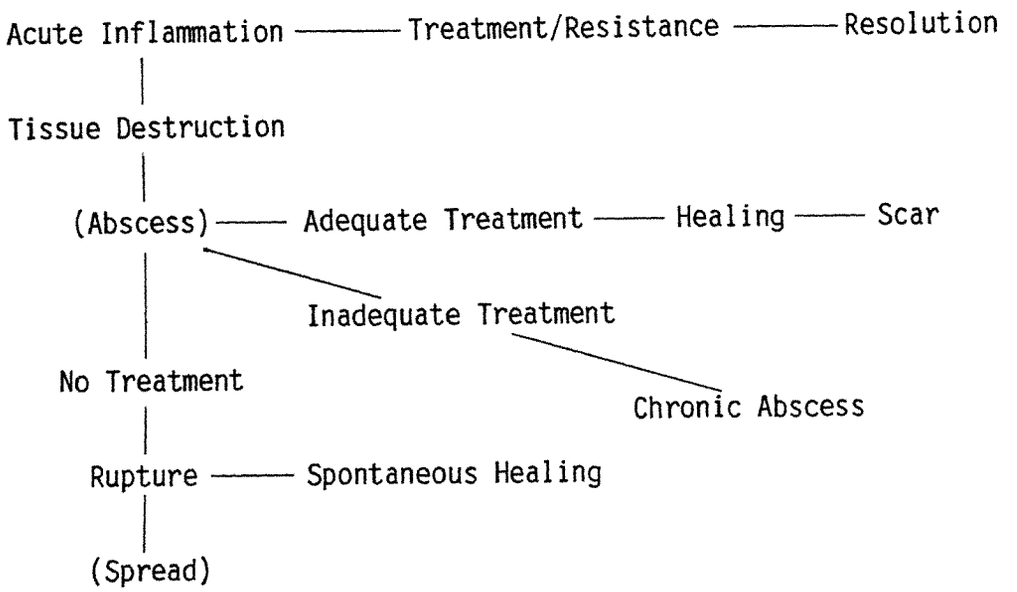
1. Scalp - under aponeurosis
   necrosis of bone
   venous thrombosis - intracranial

2. Orbit - behind eye
   meningitis
   venous thrombosis - intracranial
   panophthalmitis

3. Neck - submandibular region
   oedema of glottis
   mediastinitis

ABSCESS

Pathogenesis



Superficial Abscess

1. Boil, furuncle - hair follicle
   Injury, dirt
   Neck, axilla, perineum

2. Carbuncle - infective gangrene of subcutaneous tissue
   Multiple discharging sinuses from abscesses

3. Suppurative lymphadenitis - source related to site

4. Infected sebaceous cyst

Deep-seated Abscesses

1. Intracranial - extradural
   - subdural
   - intracerebral
2. Neck - parapharyngeal
   - retropharyngeal
3. Thorax - lung abscess
   - empyema thoracis
4. Intraperitoneal - subphrenic
   pelvic
   paracolic
   appendicular
5. Extraperitoneal - perinephric

Treatment

1. Drainage of pus
2. Removal of debris, foreign body
3. Treat predisposing cause
4. Treat source - metastatic
5. Treat complications
6. Antibiotics ?

Chronic Abscess

1. Inadequate treatment of abscess
2. Specific - TB (special features)

NFECTIONS OF THE BLOOD STREAM

Source - acute infection - SBE, pyelonephritis, osteomyelitis, thrombophlebitis, abscesses

Fever - intermittent

Chills and rigor - more so in septicaemia or pyaemia

Shock

Metastatic abscesses - especially in pyaemia

Treatment - eliminate source
- antibiotics - before blood culture
  - according to culture
- complications - shock
  - metastatic abscesses

HOSPITAL INFECTION

Definition - infection arising as a result of staying in hospital.

Main concern - wound

Organism - staph aureus
- gram -ve bacilli
- clostridium

Source - self-infection
- cross-infection

Major transport - direct contact

Timing - at operation, airborne

Prevention - limit source
- limit crossing over
- limit exposure of susceptible sites
- prophylactic antibiotics

Special site of infection - surgical wounds (tracheostomy)
- respiratory tract
- urinary tract
- burns
- drip sites (parenteral nutrition)

Special situations -
Highly susceptible individuals
- incompetent immune mechanism
- immunosuppression
- other diseases e.g., diabetes

Operation on colonised organs
- Upper respiratory tract
- Alimentary tract
- Skin

Prevention of infection in operating theatres
- Clothings, objects and instruments
- Staff and scrubbing
- Patient, preparation outside and inside
- Design - personnel and air traffic
- direction of flow
- turbulence

Operative technique - cleanliness
tidiness

SPECIFIC INFECTIONS

1. Tetanus
2. Gas gangrene
3. Tuberculosis
4. Actinomycosis

TRAUMA, SHOCK AND METABOLIC RESPONSES TO INJURY

SHOCK

DEFINITION

Peripheral circulatory failure such that tissue perfusion is inadequate to meet the nutritional requirements of the cells and remove the waste products of metabolism

Various types of shock result from failure in one or more of the three major components of the circulatory system :

- pump
- peripheral resistance
- blood volume

TYPES OF SHOCK

1. Hypovolaemic shock : Blood loss
   Plasma loss
   Fluid and electrolytes loss

2. Normovolaemic shock : Cardiogenic
   Neurogenic
   Septic
   Others - anaphylaxis, pulmonary embolism, insulin

HYPOVOLAEMIC SHOCK

e.g. haemorrhage, burns, bowel obstruction, etc.

CARDIOGENIC SHOCK

e.g. myocardial infarction, cardiac arrhythmias, congestive heart failure, etc.

NEUROGENIC SHOCK

e.g. quadriplegia, spinal anaesthesia, etc.

SEPTIC SHOCK

e.g. infection, peritonitis, meningitis, etc.

MISCELLANEOUS TYPES OF SHOCK

These include other unclassified types :

- Pulmonary embolism produces right heart failure when the pulmonary vasculature is filled by thrombus, with obstruction to flow.
- Inadequate cardiopulmonary bypass can produce shock.
- Anaphylaxis
- Insulin shock

PATHOPHYSIOLOGY

1. Metabolism

   a. Protein

      Increased metabolism with cell breakdown → ↑ blood urea
      ↑ serum creatinine
      ↑ serum uric acid

   b. Fat

      Catecholamines initiate lipolysis
      Tissue lipids / Serum triglycerides ——→ Free fatty acids (FFA) → ↑ serum FFA

   c. Carbohydrate

      - Catecholamines increase liver glycogenolysis → ↑ blood glucose
      - Aerobic metabolism —↓ Perfusion→ Anaerobic metabolism
        i.e. Glucose pyruvate —↓ Perfusion→ Lactate → ↑ blood lactate
        → ATP (small quantities)

2. Cells

   Breakdown of sodium pump

3. Circulation

   Functional shunting
   Protein extravasation

	Pulmonary Arterial	Systemic Vascular	Cardiac Output	Oxygen Consumption
Hypovolaemic shock	↓	↑	↓	↓
Hyperdynamic septic shock	±	↓	↑	±
Hypodynamic septic shock	↓	↑	↓	↓
Cardiogenic shock	↑	↑	↓	↓
Neurogenic shock	↓	↓	±	↓

↓ = depressed; ↑ = elevated;
± may be depressed, elevated or normal

4. Hormones

↑ catecholamines
↑ mineralocorticoids
↑ glucagon
↑ insulin (but ↑↑ insulin antagonists)
↑ ADH

5. Coagulation

Disseminated intravascular coagulation

6. Immune defects

ORGAN RESPONSES

1. Lung - 'shock lung' (wet and dry types)
2. Heart - reduced coronary perfusion
3. Kidneys - acute renal failure
4. Brain - cerebral hypoxia
5. GI tract - stress ulceration, ischaemic colitis
6. Others - liver, skeletal muscle

'Refractory shock' results from prolonged inadequately treated shock.

CLINICAL FINDINGS IN HAEMORRHAGIC SHOCK

1. Mild shock (up to 20% blood volume loss)

a. Pathophysiology: Decreased perfusion of nonvital organ and tissues (skin, fat, skeletal muscle and bone).
b. Manifestations: Pale, cool skin. Patient complains of feeling cold. Urine is concentrated.

2. Moderate shock (20-40% blood volume loss)

   a. Pathophysiology: Decreased perfusion of vital organs. (liver, gut, kidneys)
   b. Manifestations: Oliguria to anuria and slight to significant drop in blood pressure.

3. Severe shock (40% or more blood volume)

   a. Pathophysiology: Decreased perfusion of heart and brain.
   b. Manifestations: Restlessness, agitation, coma, cardiac irregularities, ECG abnormalities and cardiac arrest.

MONITORING THE SHOCK PATIENT

1. Pulse and blood pressure
2. Peripheral perfusion
3. Urine output
4. Central venous pressure, pulmonary capillary wedge pressure (PCWP)
5. Cardiac output and oxygen transport
6. Arterial blood gases
7. Haemoglobin and packed cell volume

PRINCIPLES OF TREATMENT IN HYPOVOLAEMIC SHOCK

1. Ensure adequacy of airway
   a. Clinical evaluation
   b. Arterial blood gases

2. Restore blood volume

   In general frequent clinical assessment of peripheral perfusion, maintenance of the CVP between 5-15 cm H_2O and a urine output above 0.5 ml/kg/h are indices of adequate[2] treatment.

   a. Crystalloids : e.g. Ringer's lactate and N. Saline
   b. Colloids : e.g. blood, plasma, serum albumin, plasma substitutes such as dextran

   In the initial treatment of haemorrhagic shock, resuscitation with crystalloids is favoured because :

   - the solutions are readily available
   - they effectively restore vascular volume for short periods
   - they lower the blood viscosity and enhance resuscitation of the microcirculation

   Ideally, initial resuscitation with crystalloids should be promptly followed by blood replacement in shock due to blood loss.

Burn shock, which produces a greater deficit of plasma volume than red cell volume, is the principal form of shock that still requires large quantities of colloid for treatment. It is prudent to withhold colloid administration during the first 24 hours of resuscitation in cases of severe burns.

3. Improve cardiac function

   a. Primary cardiac malfunction - e.g. arrythmias, infarcts - appropriate medical treatment.
   b. Blood loss externally and internally must be replaced.
   c. Cardiac tamponade - prompt evacuation needed.
   d. Tension pneumothorax - prompt needle or tube drainage
   e. Myocardial contractility may be improved by dopamine 2-9 µg/kg/min.
   f. Calcium depletion following severe hypovolaemic shock, septic shock, cardiopulmonary bypass, massive blood replacement with citrated blood can interfere with normal excitation - contraction coupling and alter contractility. Treatment : Calcium chloride 10 mls over 1-5 minutes.

4. Improve oxygen transport

   - Keep haemoglobin levels between 12 and 15 G/dl.
   - Ensure effective function of red cells by:
     preventing inorganic phosphate depletion,
     treating hypothermia
     avoiding alkalosis

5. Maintain acid-base balance

   Blood gases monitored regularly. Correct metabolic acidosis since pH levels below 7.2 or 7.3 depress myocardial contractility.

6. Assisted ventilation

   Endotracheal intubation and positive pressure ventilation are of value in profound shock.

7. Modify microcirculation

   a. Low molecular weight dextran 1-2 units (500 ml each) initially. One unit daily later.
   b. Heparin may be used in DIC. Dose 2500-5000 iu. 4-6 hrly iv.

8. Adrenocorticoids

   Some believe that massive doses of steroids may modify some of the adverse effects of gram-negative sepsis.

TRAUMA

1. Accidental trauma
   e.g. traffic accidents, fractures, burns and scalds, etc.

2. Surgical trauma
   Surgical operations constitute a form of trauma or injury e.g. herniorrhaphy, gastrectomy, oesophagectomy, etc. The effect of surgical trauma on a patient depends on both the magnitude of the operation and the general health of the patient.

METABOLIC RESPONSE TO INJURY

After any injury, there is a metabolic response which is, in general, proportional to the degree of trauma. It is characterised by:

1. A very low output of concentrated urine for 24-36 hours.
2. Immediate reduction of sodium excretion which lasts for 3-5 days.
3. A negative nitrogen balance, the so-called catabolic phase.

In addition, injury is associated with a movement of sodium into cells and a movement of potassium out of them. The plasma sodium concentration falls (contributing to the dilution hyponatraemia often observed) and the plasma concentration of potassium may rise. These changes indicate a temporary disturbance of selective membrane permeability.

FACTORS MODIFYING METABOLIC RESPONSE

1. The severity of injury
2. The nature of injury
3. The environmental temperature after injury
4. The nutritional status of the individual
5. Age
6. Sex
7. Therapy

BLOOD AND BLOOD COMPONENTS

BLOOD FOR TRANSFUSION

1. Living donors
2. Cadaveric blood

Synthetic blood has been used in clinical trials and found to be satisfactory, but it is not yet available for commercial use.

COLLECTION, STORAGE AND ADMINISTRATION

Once blood is collected from donors it is stored in plastic bags. Each full sized bag contains approximately 430 ml blood and 60 ml of a mixture of citrate, phosphate and dextrose; half-sized ones contain 300 ml blood and 42 ml of the mixture. Blood is stored at 4-6^{0}C. This is essential to preserve RBC and minimize multiplication of bacterial contaminants. Under these conditions blood can be stored up to 3 weeks.

Donor's blood must be fully cross matched against the recipient's. A full cross match involves

1. ABO and Rhesus compatibility of recipient's blood
2. Three other tests to exclude abnormal antibodies

Blood with positive VDRL test or blood from carriers of Australia antigen is not suitable for transfusion. Testing for the virus of AIDS is also carried out. Blood is usually given by venipuncture. Intra-arterial route is seldom used. It is superior to I.V. transfusion in that

a. Coronary circulation and peripheral pressure improves quickly
b. Rate of transfusion is faster.

Intra-arterial transfusion is occasionally used when massive unexpected haemorrhage occurs.

Important precautions to be taken before giving blood are to read the label on the bag, check and re-check patient's name, hospital number, name of ward, blood group and rhesus group. Blood should not be left outside the refrigerator for more than half an hour. It is important to watch a patient during the first 30 minutes of a transfusion

a. To make sure that proper flow is maintained
b. To see if there is any reaction.

Where there has been severe internal or external blood loss, rapid transfusion (if necessary using a pump) is essential to restore

adequate blood volume as soon as possible. Once a systolic B.P. of 100 mm is reached, then slow down transfusion to maintain B.P. at normal levels. Slow rates (10 - 20 drops per minute) of transfusion are indicated in

a. Chronic anaemias with a Hb < 3.7 g per cent
b. Cachexia
c. Cardiac disease
d. Respiratory disease
e. In toxaemia

It is important to watch state of filling of jugular veins : examine lung bases frequently for pulmonary oedema.

INDICATIONS FOR USE OF WHOLE BLOOD

1. Acute loss of blood - after trauma; in anticipation of loss during major surgical operations.

2. Anaemia - special precautions are necessary in chronic anaemia
   - the red blood cell component may be given separately instead of whole blood. Concentrated blood cells should be used as soon as possible and never more than 12 hrs. after preparation. Anaemia should be corrected well before surgery. Assume one pint will raise Hb by 1.0 g per cent. No major surgical procedure is carried out unless haemoglobin is at least 10.4 g per cent.

3. Thrombocytopenia - Fresh blood is given, as platelets deteriorate in stored blood
   - Platelet rich transfusions may be given separately, instead of fresh blood

PURE FIBRINOGEN

May be given in defibrination syndrome. Each bottle contains 3-4g of fibrinogen and this is reconstituted in 200 ml of sterile water.

PLASMA

1. Burns and crush injuries
2. Hypovolaemic shock until blood is available
3. Defibrination syndrome - Here one may use purified fibrinogen itself instead of plasma
4. To correct AHG and Factor V deficiencies - This requires fresh plasma or plasma stored at -20°C to prevent deterioration of these factors. AHG & Factor V can also be given in a purified form.

COMPLICATIONS OF BLOOD TRANSFUSION

1. Febrile reaction
2. Circulatory overloading and pulmonary oedema
3. Haemolytic reaction
4. Transmission of infection
5. Air embolism
6 Allergic reactions
7. Thrombophlebitis
8. Thrombocytopenia
9. Haemosiderosis

If massive transfusions (large quantities over short periods) are given, then the following may occur:

1. Dilutional thrombocytopenia and dilution of clotting factors
2. Disseminated intravascular coagulation
3. Citrate toxicity
4. Metabolic acidosis

NEOPLASTIC DISEASES

Neoplasia (new growth) is a non-conformist cellular population no longer dedicated to the purpose of the organism as a whole

- does not form organs
- not fixedly related to other cells
- functions physiologically as relatively independent and uncontrolled elements

TYPES OF PROLIFERATIVE GROWTH

1. Hyperplasia
2. Metaplasia
3. Dysplasia
4. Anaplasia
5. Neoplasia
   a. bengin neoplasms
   b. malignant neoplasms

GENERAL DIFFERENCES BETWEEN BENIGN AND MALIGNANT NEOPLASMS

	Benign	Malignant
1.	Frequently encapsulated	Non-encapsulated
2.	Non-invasive	Invasive
3.	Well differentiated	Poorly differentiated
4.	Slow growing	Rapid growing
5.	Low mitotic rate	High mitotic rate
6.	Non-metastasising	Metastasising

AETIOLOGY

1. Genetic

2. Viral

3. Environmental

EVIDENCE FOR "GENETIC" THEORY

1. Association of chromosomal abnormalities and cancers
   a. Philadelphia chromosome - CML
   b. Absence of chromosome 22 - meningioma
   c. Chromosome 14 - lymphoma, ataxia telangiectasia
   d. Trisomy 18 - Wilm's tumour
   e. Down's, Klinefelter's syndromes - leukaemia

2. Association of inherited conditions with cancer
   a. Xeroderma pigmentosum - basal and squamous cell carcinoma
   b. Ataxia telangiectasia - lymphomas
   c. Von Recklinghausen's disease - phaeochromocytoma, medullary carcinoma of the thyroid

3. Study of relatives of cancer patients
   a. one first-degree relative - 8.9%
   b. three first-degree relatives - 27.4%

EVIDENCE FOR "ENVIRONMENTAL" THEORY

1. Chemical agents
   a. scrotal cancer in chimney sweeps
   b. effect of coal tar on rabbit's ears
   c. nitrosamine and hepatoma
   d. smoking and lung cancer
   e. methylcholanthrene and sarcoma in mice

2. Physical agents
   a. Irradiation - carcinoma of the thyroid
      - skin cancer of hands of radiologists
   b. UV - skin cancer in farmers, sailors
   c. Atom bomb - leukaemia

3. Chronic irritation
   a. Ingestion of corrosives - carcinoma of the oesophagus
   b. Burn scars - squamous cell carcinoma of skin
   c. Schistosomal infestation - carcinoma of the bladder
   d. Achalasia - carcinoma of the oesophagus

4. Geographic considerations
   a. Diet
   b. Infections
   c. Local customs

EVIDENCE FOR "VIRAL" AETIOLOGY

1. Animal models
   a. Oncorna virus - avian sarcoma (Rous')
      - murine, feline, avian leukaemia
   b. Herpes virus - lymphoma in chickens
      - renal adenocarcinoma in frogs

2. Human models
   a. Burkitt's lymphoma - Epstein-Barr virus
   b. Nasopharyngeal carcinoma - EBV
   c. Hepatoma - hepatitis B virus

TUMOUR MAY SPREAD BY

1. local invasion

2. direct spread - implantation

3. transport through lymph, blood or spinal fluid

LOCAL INVASION

Physical factors favouring local invasion

1. Increased tumour tissue pressure
2. Amoeboid motility of cancer cells
3. Release of lytic tissue enzymes
4. Diminished cell-to-cell cohesion

DISTANT METASTASIS

Governed by

1. Flow-distribution
2. Susceptibility of the target organ
3. Interaction of the immune surveillance of the host

MANAGEMENT OF CANCER PATIENTS STAGING OF TUMOURS (TNM)

T - primary growth
N - nodal spread
M - distant metastasis

STEPS IN STAGING A PATIENT

1. History and physical examination

2. Laboratory investigations
   general - complete blood picture, renal and liver function tests
   specific - CEA, Bence Jones proteins, alpha-fetoprotein
3. Other investigations
   a. Radioisotope scans
   b. X-ray studies - gastrointestinal series
      tomograms
      angiograms
      ERCP
      CAT scan
   c. Ultrasonography
   d. Investigatory procedures - endoscopy

TREATMENT

1. The role of surgery in cancer

a. Surgery to prevent cancer

Underlying condition	Cancer	Prophylactic Surgery
Cryptorchidism	Testicular	Orchiopexy
Polyposis coli	Colon	Colectomy
Familial colon cancer	Colon	Colectomy
Ulcerative colitis	Colon	Colectomy
Medullary carcinoma of thyroid, familial (MEA II)	Thyroid	Thyroidectomy
Familial breast cancer	Breast	Mastectomy
Familial ovarian cancer	Ovary	Oophorectomy

b. Diagnosis and staging

Biopsy
- Aspiration
- Needle
- Incisional
- Excisional

Staging
- lymphomas
- ovarian cancers

c. Treatment of cancer

i. Definitive surgical treatment for primary cancer, identification of patients treatable by local treatment.
Selection of appropriate local treatment.
Integration of surgery with other adjuvant modalities.

ii. Surgery to reduce the bulk of tumours
- Burkitt's lymphoma
- Ovarian cancer

iii. Surgical resection of metastatic disease with curative intent
- Pulmonary metastasis in sarcomas
- Hepatic metastasis in colorectal cancer

iv. Surgery for the treatment of oncological emergencies
haemorrhage, perforation, drainage of abscess.

v. Surgery for palliation
To improve quality of life
For pain-relief, obstruction, disfigurement

vi. Surgery for reconstruction and rehabilitation
restore appearance - in head and neck cancers
restore function

2. Radiation therapy

a. external
b. internal

3. Chemotherapy

a. drugs
b. hormones

4. Immunotherapy - investigational

a. specific
b. non-specific

5. Treatment with combined modalities

PROGNOSIS

Depends on

1. Location of primary growth
2. Staging at presentation
3. Susceptibility of tumour to chemotherapy or radiotherapy
4. Inherent growth characteristic of the tumour

Half the patients have distant metastases on presentation

ASSESSMENT OF OPERATIVE RISKS, OPERATIVE AND POSTOPERATIVE CARE

PHASES OF MANAGEMENT OF THE SURGICAL PATIENT

1. Preoperative care
   - Diagnostic work-up
   - Preoperative evaluation
   - Preoperative preparation

2. Anaesthesia and operation

3. Postoperative care
   - Post-anaesthetic observation
   - Intensive care
   - Intermediate care
   - Convalescent care

PREOPERATIVE EVALUATION

1. General health assessment

2. Non-surgical disease affecting operative risk
   a. Endocrine disease
   b. Cardiac disease
   c. Respiratory disease
   d. Renal disease
   e. Haematologic disease
   f. Liver disease

3. Special factors affecting operative risk
   a. The paediatric patient
   b. The elderly patient
   c. The obese patient
   d. The pregnant patient
   e. The compromised or altered host
      - Increased susceptibility to infection
      - Delayed wound healing
   f. Drug effects

4. Consultations

5. Preoperative note

PREOPERATIVE PREPARATION

1. Informing the patient

2. Consent

3. Preoperative orders
   a. Skin preparation
   b. Diet
   c. Enemas
   d. Bed time and pre-anaesthetic medication
   e. Special orders
      Blood transfusion
      Nasogastric tube
      Bladder catheter
      Venous or arterial catheter
      Continuing medications
      Prophylactic antibiotics

ASSESSMENT OF OPERATIVE RISKS

1. The patient
2. The disease
3. The operation
4. The facilities

1. The patient
   a. status of homoeostasis
   b. vital organ functions
   c. coexisting diseases

2. The disease
   a. nature
   b. extent
   c. pathophysiological consequences

3. The operation
   a. type and complexity
   b. surgeon's skill
   c. anaesthetist's experience

4. The facilities
   a. nursing staff
   b. paramedical staff
   c. special equipments

OPERATIVE CARE

Aims

1. To provide ideal operative conditions
2. To achieve optimal conditions for healing
3. To prevent postoperative complications

1. Fastidious preoperative preparation

2. Maintenance of homoeostasis by monitoring important parameters
   a. Respiration
   b. Temperature
   c. Blood volume
   d. Fluid and electrolyte balance
   e. Vital organ functions
   f. Tissue protection and preservation
   g. Meticulous surgical technique

3. Close postoperative observation and early detection of complications.

POSTOPERATIVE CARE

Phases	Facilities
Post-anaesthetic observation	Recovery room
Intensive care	Intensive care unit
Intermediate care	General ward
Convalescent care	Convalescent hospital or home

PARAMETERS OF POSTOPERATIVE MONITORING

1. Cardiac system
2. Respiratory system
3. Haemopoietic system
4. Gastrointestinal system
5. Urinary system
6. Central nervous system
7. Metabolism

POSTOPERATIVE COMPLICATIONS

1. Wound complications
   a. Haematoma
   b. Seroma
   c. Wound infection
   d. Wound dehiscence
   e. Systemic risk factors
   f. Local risk factors
      - Adequacy of closure
      - Intra-abdominal pressure
      - Deficient wound healing

2. Respiratory complications
   a. Atelectasis
   b. Pulmonary aspiration
   c. Postoperative pneumonia
   d. Pulmonary oedema
   e. Postoperative pleural effusion and pneumothorax

3. Cardiac complications
   a. Arrhythmia
   b. Postoperative myocardial infarction
   c. Postoperative cardiac failure

4. Fat embolism

5. Urinary complications
   a. Postoperative urinary retention
   b. Urinary tract infection
   c. Postoperative oliguria and renal failure

6. Cerebral complications
   a. Postoperative cerebrovascular accident
   b. Convulsions

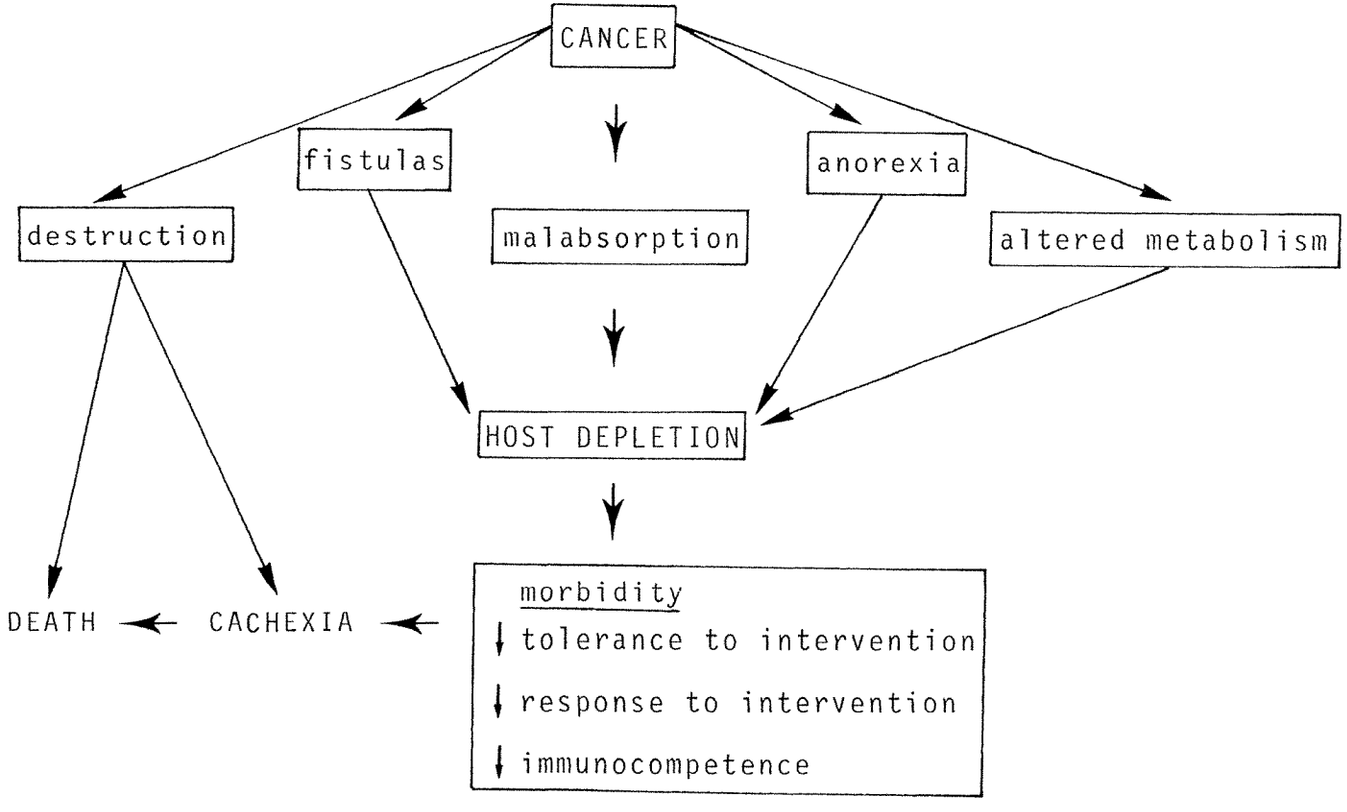
7. Psychiatric complications
   a. The ICU syndrome
   b. Delirium tremens

8. Complications of intravenous therapy and haemodynamic monitoring
   a. Air embolism
   b. Foreign bodies
   c. Phlebitis
   d. Cardiopulmonary complications
   e. Infections
   f. Ischaemic necrosis of the finger

9. Postoperative fever

COMPLICATIONS OF GASTROINTESTINAL SURGERY

1. Vascular complications
   a. Haemorrhage
   b. Gangrene
2. Postoperative ileus
3. Gastric dilatation
4. Mechanical problems
   a. Stoma obstruction
   b. Bowel obstruction
   c. Postoperative faecal impaction
5. Anastomotic leakage and fistula
6. Injury to adjacent structures
7. Complications of drains
8. Postoperative pancreatitis and cholecystitis
9. Postoperative hepatic dysfunction


CANCER
fistulas
anorexia
destruction
malabsorption
altered metabolism
HOST DEPLETION
DEATH
CACHEXIA
morbidity
tolerance to intervention
response to intervention
immunocompetence

CUTANEOUS AND SUBCUTANEOUS LESIONS

1. To make diagnosis

   Layer of tissue of origin
   Physical characteristics of lump
   Lymph node involvement

2. Possible layer of tissue of origin

   Skin
   Subcutaneous tissue
   Muscle
   Bone or joint

3. Physical characteristics of lump

Site	temperature
Shape	colour
Edge	ulceration
Surface	fluctuation
Consistency	transillumination
	pulsatility

4. Epidermal cyst

   Cyst of skin lined with squamous epithelium containing keratin that is arranged in whorls.

5. Sebaceous cyst

   A retention cyst lined by superficial squamous cells
   Contains yellowish white putty material of fat and epithelial cells
   Common site - face and scalp
   Not on palms and soles
   Hemispherical swelling
   Attached to skin
   Punctum (blocked duct)

   Complications : a. infection
   b. ulceration
   c. sebaceous horn
   d. calcification

   Treatment : excision
   incision avulsion

6. Implantation dermoid

Epithelium driven beneath skin by puncture wound
Common site - fingers

7. Sequestration dermoid

Inclusion of epithelium beneath surface where lines of developing skin meet and join

a. external angular dermoid
b. periauricular
c. midline

8. Branchial cyst

Vestigial remnant of second branchial cleft
20-25 years or later
Cystic swelling under anterior border of upper 1/3 of sternomastoid.
May transilluminate.
May be complicated by infection.
Wall lined by squamous epithelium. Surrounded by lymphadenoid tissue.
Fluid contains cholesterol crystals.
Has a tract that passes through carotid bifurcation, to end in lateral pharyngeal wall.

Treatment : Excision of cyst with tract
Drainage in infected stage may lead to a persistent branchial fistula, which is at a higher site than congenital branchial fistula.

9. Branchial fistula

Persistent branchial cleft.
External orifice lies in lower 1/3 of neck near anterior border of sternomastoid.
Tract passes through carotid bifurcation to internal orifice behind tonsils.
Often ends blindly in region of lateral pharyngeal wall.
Congenital type - lined by columnar epithelium
surrounded by muscle
discharges mucus
seat of recurrent inflammation
Fistulogram or sinogram may show up tract.

Treatment - excision

10. Papilloma of skin

Consists of a central axis of connective tissue with blood vessels and lymphatics.
The surface epithelium may be squamous cell or basal cell.

11. Squamous papilloma

Most are viral in origin (infective wart, common wart, verruca vulgaris)

In children
In hands or feet, in crops
Hemispherical with a papilliform surface
Warts on the sole where they are buried in a thick layer of keratin have a smooth surface exquisitely tender

Infectious
Often disappears spontaneously

Treatment : cauterisation
curettage

Other types : of varying sizes, have been variously called cutaneous papilloma, fibroepithelial polyp, soft fibroma, molluscum fibrosum

Soft papilloma around eyelid of elderly people.
Keratin horn - papilloma with excess keratin formation in old people.

12. Basal cell papilloma (seborrhoeic or senile wart, seborrhoeic keratosis)

A flattened papilloma
Chiefly of basal-like cells, relatively little differentiation into prickle cells.
Keratin abundant, characteristically in spherical mass (keratin pearls, horn cysts) within epithelium and sometimes reaching the surfaces.
Melanocytes present, melanin abundant.
Mitosis absent.
Growth slow.

Older people
Forehead, chest and back, arms
Circular, brownish, slightly raised
Sharply circumscribed
'Stuck on' appearance
Friable hyperkeratotic surface

13. Keratoacanthoma (molluscum sebaceum)

Derived from prickle cell layer
Microscopically resembles squamous cell carcinoma
- Strands of cells spreading laterally
- Under thinned out epithelium
- Cell nest present
- ↑ mitosis

Middle age or elderly
Face or hands
Firm hemispherical nodule
Grows rapidly into a rounded, slightly umbilicated mass
- 1-2 cm diameter in course of 1-2/12

Summit ulcerates, forms crust beneath which is on ulcer crater
Crust is shed
Regression occurs
Heals within 6/12 or so, leaving a small pitted scar

14. Dermatofibroma, histiocytoma, nodular subepidermal fibrosis, fibroma simplex, sclerosing haemangioma, fibroma durum

Benign non-encapsulated dermal nodular lesion
- Of proliferating fibroblast
- Irregularly arranged collagen bundles
- Capillary blood vessels
- Histiocytes
- Mononuclear inflammatory cells

In different proportions
Histiocytes may contain haemosiderin or become xanthomatised

Early stage - histiocytoma - histiocytes predominate
Mature stage - dermatofibroma - collagen bundles and fibroblasts
Ageing stage - sclerosing haemangioma - vascular connective tissue
End stage - nodular subepithelial fibrosis - dense fibrous tissue

Adult:
Lower legs female, most common
Also seen on trunks and arms

Firm dermal nodule, rarely exceeds 1-2 cm
Colour range - pale skin hue to red, brown, and yellow

Treatment - excisional biopsy for diagnosis
no treatment
excision for cosmesis and symptoms

15. Granuloma pyogenicum

An acquired capillary haemangioma
Trauma or infection
Face, fingers and toes
Soft or firm, pedunculated nodule
Bleeds easily

Treatment - excision

16. Pilomatrixoma

A benign tumour from basal layer of hair follicle
Consists of 2 types of cells - ghost cell and basaloid cell
Calcification occurs in the stroma

17. Lipoma

Subcutaneous
Soft, fluctuate
Lobulated surface
Definite edge
Truly mobile
Occasionally tender especially if multiple

18. Fibroma

True fibroma rare
Mostly combined with other mesodermal tissue
- Fibrolipoma
- Fibromyoma
- Neurofibroma

Desmoid tumour

19. Neurofibroma

Localised
Generalised

20. Haemangioma

Capillary - salmon patch
portwine stain
strawberry angioma
Venous
Arterial

21. Lymphangioma

    Capillary
    Cavernous
    Cystic hygroma

22. Inflammatory swelling

    Boils
    Carbuncles

MALIGNANT DISEASES OF SKIN

I. BASAL CELL CARCINOMA

Fair skin, sunlight
Age - middle or late

Site - anywhere
90% face above line from ear lobule to corner of mouth
commonest - inner canthus of eye

Types - 1. papulo-pearly
2. cystic
3. sclerosing and morphotic
4. cicatricial
5. pigmented

Slow growing
Locally invasive

Treatment - radiotherapy
surgery

II. SQUAMOUS CELL CARCINOMA

More rapid growth, wider local spread
Lymphatic and blood metastasis
De novo or in damaged skin

Premalignant skin diseases - chronic ulcer
radio dermatitis
senile keratosis
leucoplakia
Bowen's disease
Paget's disease

Sites - anywhere
common on head and neck and upper limbs

Gross - cracking or hardening of skin
mass
ulcer with everted edge

Treatment - local - RT
surgery
- lymph nodes

III. MALIGNANT MELANOMA

Fair skin, sunlight
De novo or from benign pigmented naevus of junctional or compound type

Benign pigmented naevus

1. lentigo
2. junctional
3. dermal
4. compound

Signs of malignant change in benign pigmented naevus

1. ↑ size
2. ↑ or ↓ pigmentation
3. ulcerate, weep or bleed
4. itch or burning sensation
5. spread of pigment
6. L.N.

* suspicion - biopsy

Types of malignant melanoma

1. lentigo malignant melanoma
2. superficial spreading melanoma
3. nodular

Prognostic factors

1. clinical type
2. depth (Clark's level)
3. growth rate
4. size
5. site
6. lymphatic invasion
7. female better

Treatment (malignant melanoma)

1. not radiosensitive
2. surgical excision - primary
   L.N.
3. chemotherapy
4. immunotherapy

MANAGEMENT OF THE BURNED PATIENT

1. Mode of Injury (Types of Burns)

   Scalds - hot liquids, steam
   Dry heat - flash, flame, friction
   Electrical - low/high tension
   Chemical - acid, alkali, others
   Radiation - UV, X-ray, gamma

2. History Relating to Burn Injury

   Time of burn, place - confined place ?
   Were clothes burned ?]
   Were clothes removed ?] Determines depth
   Was part cooled ?]
   Previous treatment - IV fluids, pain, tetanus, local burn applications.

   Past History

   Allergies, diabetes, hypertension, epilepsy, drug addiction, alcoholism.

3. Physical Examination

   a. General examination
   b. Burn - extent, depth
   c. Associated injuries

4. Extent of Burn

   a. 'Rule of Nines'
   b. Palm size = 1%
   c. Lund and Browder method

5. Depth of Burn

Degree	Thickness		Healing		
First (Erythema)	-		-		
Second	Partial	- superficial	7 days		
		- deep dermal	3 weeks	]	Deep Burns
Third	Full	-	> 3 weeks	]	

   For resuscitation, ignore 1st degree (erythema)

   Total of 2^{0} and 3^{0} burns is basis for resuscitation.

6. Criteria for Admission

7. First Aid Treatment

8. Resuscitation in Burns

Adults > 15% surface area
Children > 10% surface area

9. Pathophysiology of Burns Shock

a. Oedema and haemoconcentration
b. Vasoconstriction in skin, kidney, GIT

10. Pattern of Fluid Loss

Period of increased capillary permeability lasts for 36-48 hrs, more fluids are required in first 24 hours.

11. IV Fluids

a. Replacement - many types of fluids and formulae are used. They are based on resuscitation data on large numbers of patients who have been brought through the shock period successfully with widely differing methods. The aim of resuscitation is to bring the patient through the shock period with as wide a margin of safety as possible, in as fit a state as possible to face the difficulties and dangers of the following weeks.

i. Colloids - plasma, PPF, dextran
ii. Crystalloids - saline - normal, hypertonic
Ringer's lactate
iii. Combinations - Parkland Ringer's lactate and plasma

Example - Resuscitation with Plasma (freeze dried)
Muir & Barclay formula (Mount Vernon Hospital, Middlesex)

$$\frac{?\ \% \times ?\ kg}{2} = ?$$ c.c.plasma per period

3 rations in first 12 hours (4, 4, 4)
2 rations in second 12 hours (6, 6)
1 ration in third 12 hours (12)

Fluid calculations are based on time of burn injury, not time of admission to hospital.

b. Maintenance

100 c.c. dextrose 5%/normal saline (2:1) per hour for a 70kg man

Formulae only serve as guidelines. The key to successful resuscitation is frequent bedside assessment e.g., assess:

Clinical
- i. Conscious level and orientation
- ii. Peripheral perfusion
  - warmth of limbs
  - capillary refilling
  - venous filling
- iii. CVP
- iv. Hourly urine output

Laboratory Parameters - Haematocrit, Electrolytes

12. Wound Care

a. Superficial burns - aim for spontaneous healing by preventing infection

b. Deep burns - dead tissue separates as slough which is invariably infected and may lead to septicaemia. Aim to get rid of slough, prevent infection and obtain early coverage of new areas by skin grafting.

13. Topical Wound Treatment

a. Exposed methods

b. Closed methods

Preference for closed methods

Materials for dressings

Antibacterial agents - silver sulphadiazine
- silver nitrate
- furacin
- sulphamylon
- eusol

Removal of slough

14. Surgical Treatment

a. Early tangential excision

Technique vs concept - before 5 days
- prevent zone of stasis
- indications

b. Delayed excision

Techniques - Humby knife
- Avulsion
- CO_2 laser

15. Skin Grafting

Knives
Donor sites
Treatment of recipient areas
Handling of grafting
After treatment - dressings
- exposure
Delayed application
Graft storage
Graft failure
Repeated use of donor site
Priority areas
Use of homograft
allograft

16. General Care

Asepsis and Antisepsis - washing hands is vital
Patients morale
Nutrition - oral
- hyperalimentation
Blood transfusion
Iron and vitamins
Antibiotics - prophylactic
- therapeutic
- Pseudomonas aeruginosa
- septicaemia
Pseudomonas vaccine

Prevention of avoidable complications
- pressure sores
- urinary sepsis
- muscle wasting
- joint contractures - physiotherapy
- splintage

17. Burns of Special Areas

Eyelids, ears, nose, lips, air passages, scalp, neck, arms, hands, trunk, perineum, lower limbs, feet.

18. Special Burns

Electrical - flash, hot element, arcing lightning, contact
- high/low tension

Chemical
Hot liquid - tar
Hot metal
Radiation

19. Burn Scars

a. hypertrophy — Treatment: antipruritics
b. keloids — steroids, pressure, surgery
c. contractures
   i. early - prevention, stretching
   ii. established - release: - SSG, splintage flaps
d. unstable scars - Marjolin's ulcer

20. Other Complications

Infections - septicaemia
- lung, urinary tract, drip site
Malnutrition
Respiratory - upper airway, lower airway
Renal
Liver
Anaemia
Psychiatric

21. Rehabilitation

Home, work, society

22. Burn Prone People

23. Social Pathology of Burns

24. Team Work

a. Medical
   Burns surgeon
   Renal physician
   Anaesthetist
   Psychiatrist
   Bacteriologist (Clinical)
   Intensive care physician

b. Nursing and Paramedical
   Infection control nurse
   Physiotherapist
   Occupational therapist
   Dietitian
   Medical social worker
   Rehabilitation officer

25. The Burns Unit

Centralisation of facilities, expertise.
Infection control

DISEASES OF THE SALIVARY GLANDS

Major salivary glands - parotid, submandibular

Minor salivary glands - occur throughout the upper respiratory tract especially hard palate and tonsillar regions.

Diseases
- Inflammatory diseases and stones
- Tumours

I. NON-OBSTRUCTIVE SIALADENITIS

1. Acute suppurative parotitis

Elderly, debilitated, poor oral hygiene, dehydration, post-operative

S/S - fever, pain, diffuse swelling and tenderness over the parotid gland.
Stensen's duct orifice - inflamed purulent discharge

Pathogen - staph. aureus

Treatment
- rehydration
- specific antibiotics i.v. high dose
- improve oral hygiene
- stimulate secretion
- surgical drainage

2. Recurrent Sialadenitis

Children, adolescents
No identifiable cause
Recurrent attacks of acute distension of one or more salivary glands
Usually subsides spontaneously in 12-24 hours
If swelling persists for more than 24 hours, secondary bacterial infection may occur
Problem usually disappears with adulthood

Sialogram - chronic distension of peripheral duct system (sialectasis)

Treatment
- treat superimposed infection
- surgical excision if frequent suppuration

3. Acute viral parotitis (mumps)

Caused by a RNA virus - paramyxovirus
An acute, contagious epidemic disease, affects children, rare before 1 year old.
History of contact 2-3 weeks ago.

S/S Fever, malaise
Bilateral parotid involvement (80% cases)
Other salivary glands may be involved
Parotids of rubbery consistency, enlargement lasts for 1-2 weeks.

Complications - CNS - meningitis, encephalitis
orchitis
oophoritis
pancreatitis etc.

Investigations - virus can be isolated from saliva, urine or CSF
serological test

Treatment - no specific treatment

4. Sjogren's syndrome (Sicca syndrome, Mikulicz's disease)

Syndrome consists of keratoconjunctivitis sicca
xerostomia
swelling of salivary glands (usually parotids)
lacrimal glands may be affected

Associated with rheumatoid arthritis
polyarteritis nodosa
SLE
Hashimoto's thyroiditis

An autoimmune disease
Affects middle aged women

S/S dry mouth or eyes
joint symptoms
bilateral parotid swelling

Diagnosis - Biopsy of palatal salivary glands
Serology - hypergamma-globulinaemia
rheumatoid factor
ANF
anti-thyroglobulin antibodies

II. OBSTRUCTIVE SIALADENITIS

1. Stones (Sialolithiasis) - more common in submandibular gland than parotid
   often multiple
   tend to recur
   secondary infection may occur

   S/S - eating would lead to swelling and pain
   stones may be seen or felt
   X-ray - submandibular stones radio-opaque
   - parotid stones radiolucent
   Sialography

   Treatment - stone near/at duct orifice may be removed transorally
   - for multiple, recurrent stones, surgical removal of gland

2. Ductal strictures

   Cause - trauma
   stones
   recurrent infections
   S/S - recurrent distension of gland with eating
   - recurrent or chronic suppuration

   Treatment - dilatation with lacrimal dilators
   - surgical excision of gland

III. Tumours

Common benign tumours
- pleomorphic adenoma
- Warthin's tumour

Common malignant tumours
- adenoid cystic carcinoma
- adenocarcinoma
- squamous carcinoma
- malignant pleomorphic adenoma

Tumours of variable malignancy
- muco-epidermoid tumour
- acinic cell tumour
- oncocytoma

Rarer tumours
- haemangioma
- lymphangioma
- sarcoma
- lipoma
- acidophilic cell adenoma
- metastatic tumour
- benign lympho-epithelial lesion

1. Benign tumours

a. Pleomorphic adenoma (benign mixed cell tumour)

Most common salivary gland tumour 60%
Benign parotid tumours 90%
More common in women around 40 years of age
Usually unilateral
Symptomless except "the lump"
No pain, very slow growing
Facial nerve paralysis never present

Usual site - tail of parotid just anterior to lobe of ear
Smooth, superficial, round and mobile
Encapsulated, but small excrescences may project from capsule
Sometimes multilobulated
Greyish-white

Histologically, both epithelial and mesenchymal elements present. Epithelial element consists of small basophilic cells arranged in trabecular pattern, mesenchymal stroma may be myxoid, fibroid, chondroid or combination.
The cartilaginous stroma is believed to be mucin secreted by the myoepithelial cells.

Treatment - superficial parotidectomy

Recurrence after primary surgery 5%

b. Warthin's tumour (papillary cystadenoma lymphomatosum)

Second most common benign tumour
Only seen in parotid gland
M : F = 7 : 1
Average age of onset 70 years (range 30-74)
Bilateral 10%
Slow growing
Site - in mid-zone of gland rather than tail
Soft, fluctuant or solid

Encapsulated
Grossly, appear as cysts with multiple papillary projections from the wall
Cysts contain brown mucoid fluid

Histologically composed of proliferating salivary gland cells in lymphoid tissue

Treatment - superficial parotidectomy

Recurrence rate - low 2%

2. Malignant Tumours

a. Adenoid cystic carcinoma

Equal sex distribution
Any age, peak - sixth decade
Comprise small percentage parotid tumours but most common malignant tumour of submandibular and minor salivary gland
Slow growing - usually present for 5 years before presentation

Signs of malignancy
- pain
- skin fixation
- facial nerve paralysis

Tumour poorly demarcated - may present as an area of tender induration rather than discrete lump

Histology - uniform basaloid cells in clumps, masses or cords.
"Swiss cheese" picture

Tend to spread along nerve sheaths and ultimately invade cranial cavity
L.N. secondary 10%
Recurrence after surgery - 50%
5-year survival 50% 20-year survival 15%
Patients eventually develop distant metastasis 60%

b. Adenocarcinoma

Equal sex incidence
Wide age range
May present as asymptomatic mass or with frank signs of malignancy
Histology varies
5-year survival 40%

c. Squamous cell carcinoma

M : F = 2 : 1
Commonest in 7th decade
Rapid growth
Causes pain, ulceration, facial paralysis
L.N. metastasis 50%
Poor prognosis

d. Malignant pleomorphicadenoma

2-5% pleomorphic adenomas will turn malignant
Average age 50
Sudden rapid enlargement of a long standing parotid mass that becomes painful
Facial nerve paralysis not present
L.N. metastasis 10%

3. Tumours of variable malignancy

a. Muco-epidermoid carcinoma

Salivary tumours 10%
Most common malignancy of parotid gland. In submandibular gland, second in frequency to adenoid cystic carcinoma
Equal sex incidence
Peak - 5th decade
In children, the most common malignant salivary tumour
May or may not be encapsulated
Solid or cystic

Histologically : 2 major cell types
- epidermoid cells
- mucous cells

Classified into : high, intermediate, and low grades. The more mucous cells, the lower the grade, the better the prognosis

5-year survival low grade 90%
Higher grade 40%

b. Acinic cell tumour

Uncommon
Exclusively affects parotid
Tumour cells resemble normal acinar cells with variable lymphoid tissue

c. Oncocytoma

Uncommon
Also affects parotid only
Oncocytes are derived from intralobular ducts or acini

4. Approach to parotid swellings

Unilateral - most tumours
Bilateral - Warthin's tumour can be bilateral
Involve more than 1 gland - sialectasis
stones
benign lympho-epithelial lesions
endocrine conditions e.g. myxoedema
Cushing's syndrome
Diffuse swelling of whole gland probably not a tumour
Fluctuation in size of gland and pain with eating - inflammatory or stones

Signs of malignancy - pain
skin fixation, ulceration
facial nerve involvement
L.N.
distant metastasis

Bimanual palpation - for stones and deep lobe tumours

Duct orifice - stones
discharge

5. Staging (American Joint Committee for Cancer Staging and Results Reporting, 1976)

Tumour
T1 0-3 cm Solitary mobile
T2 3.1-6 cm Solitary mobile or skin fixation
T3 >6cm Multiple nodules, deep fixation, facial nerve dysfunction

Stage I : T_1 N_0

Stage II : T_2 N_0

Stage III : T_3 or any tumour size with N_1

6. Biopsy

a. A discrete salivary gland mass should not be biopsied.
90% of parotid swellings are pleomorphic adenoma.
High chance of local recurrence after biopsy.

b. Needle biopsy not helpful.

7. Treatment of parotid tumours

Benign parotid tumours -

Superficial parotidectomy since pleomorphic adenoma tends to recur after enucleation because of its bosselated surface.
Less chance of facial nerve damage

Total conservative parotidectomy -
Deep lobe tumour
Conservation of facial nerves

Malignant parotid tumour -

If facial nerve palsy present
Total radical parotidectomy
Removal of involved skin
Removal of facial nerve and nerve grafting
Radical neck dissection if L.N. involved
For adenoid cystic carcinoma the facial nerve should be resected widely

Malignant parotid tumour

With no facial nerve paralysis, can preserve facial nerve during operation

Submandibular gland tumour

More likely to be malignant but usually encapsulated
Remove entire gland

Combined surgery and irradiation give the best result for high grade malignant tumours

Tumours of the salivary gland are usually radiosensitive, though not radiocurative

Irradiation could palliate pain and slow tumour growth in locally advanced and metastatic tumours

8. Prognosis - depends on clinical staging and histologic type and grade

9. Long term follow-up is necessary to detect late recurrences

TUMOURS OF THE ORAL CAVITY AND PHARYNX

PREMALIGNANT LESIONS

1. PRECANCEROUS LESIONS

- Leucoplakia
- Erythroplakia

2. PRECANCEROUS CONDITIONS

- Syphilis
- Sideropenic dysphagia
- Oral submucous fibrosis

3. LEUCOPLAKIA

Aetiology

- Tobacco
- Candida
- Virus
- Alcohol

Histology

- Most have hyperkeratosis
- Epithelial dysplasia 10-25%
- Malignant transformation 0.13-6%

Common Sites

- Buccal mucosa
- Labial commissure
- Labial mucosa

Factors Influencing Malignant Potential

- Duration of follow-up
- Age
- Location
- Type
- Adjacent area
- Dysplasia
- Tobacco (decrease)

Treatment

- Excision + grafting - severe dysplasia, carcinoma-in-situ
  - nodular type
- Remove local factor
- High dose vitamin A
- Cryosurgery

4. COMPARISON OF MALIGNANT POTENTIAL

	Leucoplakia	Erythroplakia
No dysplasia	80	0
Mild to moderate dysplasia	12	9
Severe dysplasia to Carcinoma-in-situ	5	40
Carcinoma	3	51

5. ERYTHROPLAKIA

Features

- Bright red
- Velvety

Common Sites

- Floor of mouth
- Tongue

ORAL CANCER

1. TYPES OF ORAL CANCER

- Squamous cell carcinoma
- Verrucous carcinoma
- Salivary tumours
- Lymphoma
- Basal cell carcinoma

2. AETIOLOGY

- Tobacco
- Alcohol
- Dental hygiene
- Liver cirrhosis
- Dietary deficiencies
- Industry - textile
- Low income

3. FEATURES

- Age - middle or elderly
- Male > Female
- Second primary 10%
- Common sites - between edge of tongue and alveolus
- Appearance - infiltrating with ulceration
  exophytic papillary growths rare
- Microscopy - well or moderately differentiated (poorly differentiated rare)

4. TENDENCY TO METASTASISE TO REGIONAL LYMPH NODE

Tongue (post 1/3) oropharynx	>	Tongue (anterior 2/3)	>	Lip Floor of mouth Buccal mucosa Hard palate Gum

- Large tumour > small tumour
- Poorly differentiated > well, moderately differentiated

5. CLINICAL DETECTION OF LYMPH NODE METASTASIS

- Reliability - 70-80%
- Tongue - worse

6. EFFECT OF NODE METASTASIS ON SURVIVAL

- Histology negative 50-70%
- Metastasis present 20%

7. T CLASSIFICATIONS

- TIS - carcinoma in situ
- T1 - $\leq$ 2cm
- T2 - $\geq$ 2 $\leq$ 4
- T3 - > 4cm
- T4 - Extension to muscle, bone, skin etc.

8. N CLASSIFICATION

- N0 - not palpable
- N1 - mobile - homolateral
  - N1a not significant
  - N1b significant
- N2 - mobile - contralateral
  - N2a not significant
  - N2b significant
- N3 - fixed

9. M CLASSIFICATION

- M0 - no distant metastasis
- M1 - distant metastasis present

10. STAGE-GROUPING

- Stage I - T1, no significant LN
- Stage II - T2, no significant LN
- Stage III - T3, no significant LN, or any T, N1b
- Stage IV - N2b or N3 or N1

11. MANAGEMENT OF ORAL CANCER

- Establish diagnosis
- Assess extent of disease
- Assess general status
- Surgery vs radiation
- Lymph node metastasis
- Clinically negative neck

12. OTHER METHODS OF TREATMENT

- Chemotherapy
- Cryosurgery

13. RECURRENCE PATTERN

- Most common failure - primary site
- 90% local recurrence appear in 2 years
- Salvage of failure poor - 16%

14. CARCINOMA OF LIP

- Definition of lip
- Light skin race
- Aetiological factor - sunlight, pipe, chronic cheilitis, actinic keratosis
- Male
- Lower lip

15. CARCINOMA OF FLOOR OF MOUTH

- Pain - ear
- Excessive salivation
- Small area, early invasion
- Late cases

16. CARCINOMA OF ALVEOLUS

- Mandible 4 times
- Loose teeth
- Bony destruction

17. CARCINOMA OF BUCCAL MUCOSA

- Mucosa often bitten
- Infection - trismus
- South-East Asia
- Betel nut + tobacco chewing
- Late cases usually

18. CARCINOMA OF TONGUE

Site - Anterior lesion : posterior lesion = 4:1
- Edge 48%
- Dorsum 19%
- Undersurface 14%
- Extensive 14%

Presenting symptoms	- mass or ulcer of tongue	89%
	- difficulty in swallowing	4.5%
	- cervical lymphadenopathy	3.5%
	- ear pain	1%
	- bleeding	1%
	- hoarseness	1%
Symptoms	- pain	61%
	- ear pain	29%
	- excessive salivation	25%
	- difficulty in swallowing	25%
	- slurring of speech	23%
	- ankyloglossia	21%
	- cervical lymphadenopathy	21%
	- bleeding	15%
	- hoarseness of voice	6%
	- dyspnoea	3.5%
	- trismus	2.5%

TUMOURS OF ORO- AND HYPOPHARYNX

1. OROPHARYNGEAL CANCER

Anatomy

- Anterior wall - tongue posterior to the vallate papillae (base of tongue or posterior third)
- Lateral wall - tonsils, faucial pillars and glossotonsillar sulci
- Posterior wall - oropharyngeal wall
- Superior wall - inferior surface of soft palate and uvula

Pathology - squamous carcinoma 85-90%
adenoid cystic carcinoma 5-10%
malignant lymphoma 5-10%

Clinical Features - dysphagia, hoarseness, pain
indirect laryngoscopy

2. HYPOPHARYNGEAL CARCINOMA

Anatomy and Relative Site Incidence

- Piriform fossa 55%
- Postcricoid space 40%
- Posterior pharyngeal wall 5%

Aetiological Factors - smoking and drinking
sideropenic dysphagia - female (3x)

Pathology - squamous carcinoma

Local Spread - piriform fossa - lateral - thyroid cartilage
medial - larynx
postcricoid space - anterior - larynx, trachea
posterior - prevertebral fascia
posterior pharyngeal wall - prevertebral fascia

Clinical Features - dysphagia, sore throat, hoarseness
laryngeal expansion, fixation
loss of laryngeal crepitus
lymph nodes
indirect laryngoscopy

Investigations - lateral X-ray of neck
barium swallow
direct laryngoscopy

3. TREATMENT OF ORO- AND HYPOPHARYNGEAL CANCER

- surgery - resection, reconstruction
- radiotherapy - as adjuvant
- chemotherapy - adjuvant or palliative

DISEASES OF THE OESOPHAGUS

I. SURGICAL ANATOMY OF THE OESOPHAGUS

(References: Bailey and Love's Short Practice of Surgery, p.759,
Clinical Anatomy by Harold Ellis, pp.44-47)

Note : Length and measurements from upper incisor.
Immediate relationship to trachea, recurrent laryngeal nerve, left atrium and aorta in neck, chest and abdomen.
Blood supply from branches of inferior thyroid artery, descending thoracic aorta and left gastric artery.
Lymphatic drainage to supraclavicular and subdiaphragmatic lymph nodes.

II. SYMPTOMS AND SIGNS

Main symptoms - dysphagia (distinguish from oropharyngeal cause)
Other symptoms - pain, hoarseness, regurgitation, haematemesis.
Minimal local physical signs except in advanced carcinoma or after perforation, must look for lymphadenopathy, hepatomegaly, ascites and constitutional upsets.

III. INVESTIGATIONS

1. Blood tests
2. Plain chest X-ray
3. Barium swallow (± screening)
4. Motility and pressure study
5. Oesophagoscopy (flexible and rigid)

IV. CAUSES OF DYSPHAGIA

(2 types : oropharyngeal and oesophageal)

Oesophageal causes:

1. In the lumen e.g., foreign body
   (including food but must exclude underlying oesophageal lesions)
2. In the wall:
   a. stricture e.g., after caustic ingestion
   b. tumour
   c. diverticulum
   d. spasm e.g., achalasia
3. Outside the wall e.g., retrosternal goitre, aortic aneurysm
4. General causes e.g., hysteria, Myasthenia gravis, scleroderma

V. SOME COMMON DISORDERS OF THE OESOPHAGUS

1. Foreign body e.g., Fish bone, chicken bone, coins and denture; voluntarily swallowed or by accident

Present with pain and dysphagia, blood staining of sputum.
May have features of perforation of oesophagus, mediastinal abscesses and fatal rupture of aorta.

Treatment - oesophagoscopic removal.

2. Perforation

Causes: Instrumentation, foreign body, penetrating wounds, tumour and spontaneous perforation
Features: Pain, dysphagia, fever, subcutaneous emphysema
X-ray: Subcutaneous or mediastinal emphysema, hydropneumothorax
Gastrografin or barium swallow confirms perforation.

Treatment: Nil by mouth
Drainage of abscesses
Surgical repair

3. Caustic stricture

Caused by ingestion of strong acids or alkalis.
Stricture develops as late complication.
Treat by repeated bouginage or bypass operation.

4. Achalasia (cardiospasm)

Disorganised peristalsis with failure of relaxation of lower end of oesophagus.
Features: Insidious onset of slowly progressing dysphagia in adults around their third decade.
Regurgitation with aspiration pneumonia.
X-ray: Dilated and tortuous oesophagus (as mediastinal shadow on plain chest X-ray)
Rat-tail appearance or pencil-shaped narrowing of lower end of oesophagus.
Lack of gas bubble in stomach

Treatment: Heller's operation (oesophago-cardiomyotomy)
Hydrostatic dilatation

VI. OTHER DISORDERS OF THE OESOPHAGUS

Oesophageal atresia
Plummer-Vinson (Paterson-Kelly) syndrome (sideropenic dysphagia)
Oesophageal diverticulum
Oesophageal varices
Oesophagitis

VII. TUMOURS OF THE OESOPHAGUS

1. Benign - rare e.g., papilloma, leiomyoma, submucous lipoma
2. Malignant -
   primary e.g., squamous cell carcinoma, adenocarcinoma
   secondary e.g., from bronchial carcinoma

   Sites: mid-third (50%)
   lower third (30%)
   upper third (20%)

Macroscopic: ulcer, stricture or fungating mass

Spread:

a. direct invasion through oesophageal wall and to adjacent organs
b. lymphatic:
   i. submucosal
   ii. paraoesophageal and tracheobronchial lymph nodes
   iii. supraclavicular and subdiaphragmatic lymph nodes
c. blood-borne to lungs and liver

Clinical features of carcinoma of oesophagus:

a. Usually elderly males (50-80 years of age) with rapidly progressing dysphagia
b. Pain, hoarseness, lymphadenopathy, irritating cough, weight loss

Investigations:

a. Barium swallow
b. Oesophagoscopy, biopsy, and bronchoscopy
c. Mediastinoscopy, CAT scan
d. Cardiopulmonary and nutritional work-up

Treatment :

a. Resectable lesions - oesophagectomy and reconstruction
b. Non-resectable lesions - bypass operation, intubation
c. Radiotherapy for non-resectable lesions or adjunct to surgery
d. Chemotherapy - limited benefit
e. Gastrostomy or total parenteral nutrition may be necessary

PEPTIC ULCERATION

Peptic ulceration may occur wherever mucosa comes into contact with the acid secretions of the stomach. It can therefore occur at the following sites:

1. In the duodenum as a duodenal ulcer
2. In the stomach as a gastric ulcer
3. Ulcer at the lower end of oesophagus
4. In a Meckel's diverticulum containing ectopic gastric mucosa
5. Any segment of bowel surgically anastomosed to the stomach (anastomotic ulcer)

The normal integrity of the mucosa of the stomach and duodenum is maintained by a balance between factors that tend to destroy or digest the mucosa and those that protect it.

Attacking Factors	Defending Factors
Acid peptic digestion (pH < 4)	Dilution (non-parietal secretion)
Drugs (Salicylates, steroids)	Emptying
Trauma	Neutralisation (Bicarbonate from bile and pancreas)
Ischaemia	Mucosal barrier Rich blood supply

NO ACID - NO ULCER

Whenever this balance between opposing factors is upset a peptic ulcer will result. Such ulcers may be acute or chronic. In this Lecture, we will consider chronic peptic ulcers.

I. AETIOLOGY

Duodenal Ulcer

These are 4 times more common than gastric ulcers and males are three times more likely to develop as females.

Special factors associated with aetiology:

1. Family history - Patients with a positive family history are three times more liable than normal controls.
2. Blood group and secretor status - Those belonging to blood group O and non-secretors have a higher incidence.
3. Drugs - Salicylates, steroids, anti-inflammatory drugs.
4. Increased parietal cell mass and G-cell mass.

5. Diet - Those with high refined carbohydrates, low residue and non-masticatory and low in buffering protein.
6. Endocrine - a. Zollinger-Ellison syndrome
   b. Hyperparathyroidism
7. Cirrhosis of liver
8. Chronic bronchitis
9. Stress and anxiety

Gastric Ulcer

Occurs later in life than duodenal ulcers and has a peak incidence in the 5th decade of life. Twice as many men as women are affected.

1. Drugs - Alcohol, Indomethacin, salicylates, steroids
2. Antral stasis
3. Duodenal reflux of bile - mucosal damage

II. SYMPTOMATOLOGY

Duodenal Ulcer	Gastric Ulcer
Periodic epigastric pain, relieved by food, and antacids.	Periodic epigastric pain, exacerbation with food.
Appetite good	Appetite poor
Nocturnal epigastric pain	No nocturnal pain
No nausea or vomiting	Nausea, vomiting
Upper social classes	Poorer class of patients
No loss in weight	Loss of weight

III. DIAGNOSIS OF GASTRIC AND DUODENAL ULCERS

1. Almost always by upper G.I. tract endoscopy
2. Barium studies
3. Measurements of acid secretion
   a. Basal acid secretion
   b. Maximum secretion
   c. Peak acid output
4. Serum gastrin
5. Biopsy of gastric ulcer

IV. TREATMENT

Duodenal Ulcer

Medical treatment consisting of antacids and H_2-Receptor Blockers, e.g. Cimetidine.

Surgical treatment is indicated for

1. Bleeding
2. Perforation
3. Obstruction
4. Failed adequate medical treatment

Surgical treatment consists of either truncal vagotomy and drainage operation (such as pyloroplasty or gastrojejunostomy) or truncal vagotomy and antrectomy. Highly selective vagotomy is still under trial. Polya type of partial gastrectomy is done less frequently.

Gastric Ulcer

Medical treatment consists of treatment with antacids and H_2-Receptor Blockers; carbenoxolone sodium is also used.

Surgical treatment is indicated for haemorrhage, perforation, obstruction and intractability and the need to rule out malignant change. The Billroth 1 gastrectomy is the operation of choice for gastric ulcers.

V. ZOLLINGER-ELLISON SYNDROME

Is a syndrome with gastric hypersecretion and hypergastrinaemia. This may be due to:

1. Gastrinoma
2. Antral G-cell hyperplasia
3. Retained gastric antrum (after surgery)
4. Hypercalcaemia

The condition is diagnosed by serum gastrin estimations and acid secretion studies.

Treatment

Total gastrectomy. Recently H_2-Receptor Blocking drugs have been used with some success.

STOMACH AND DUODENUM - OTHER DISEASES

I. HYPERTROPHIC PYLORIC STENOSIS (CONGENITAL)

This affects boys four times as often as girls. Vomiting starts about one week after birth and becomes progressively more frequent and projectile. Vomitus does not contain bile. Metabolic alkalosis may occur. A lump may be palpable in the epigastrium. Ramstedt's operation is done to correct this condition.

II. ACQUIRED PYLORIC STENOSIS

1. Duodenal ulcer 56%
2. Carcinoma of antrum 36%
3. Gastric ulcer (prepyloric) 4%
4. Adult hypertrophy of pylorus 2%
5. Rare causes (Hodgkin's disease, pancreatic heterotopia) 2%

III. ACUTE GASTRITIS

This is usually due to alcohol, drugs, corrosive chemicals, and some items of food, septicaemia, in patients recovering from multiple injuries and in extensively burnt patients. In severe cases, surgery in the form of under-running of bleeding ulcers, together with vagotomy and a drainage operation may be necessary.

IV. CHRONIC GASTRITIS

1. Chronic superficial gastritis
2. Chronic atrophic gastritis
3. Chronic granulomatous gastritis

V. DUODENITIS

Is a cause of non-ulcer dyspepsia. It is diagnosed endoscopically and proven by biopsy of the duodenum.

VI. GASTRIC CANCER

Peak incidence is 40-60 years of age. Ratio of male:female = 3:1.

Aetiological Factors

1. Gastric polyp]
2. Pernicious anaemia] Pre-malignat conditions
3. Gastric ulcer]
4. Nitrites in foodstuffs are converted to nitrosamines which are carcinogenic.
5. Doubtful association with blood group A.

Macroscopic Types

1. Cauliflower-like growth
2. Malignant ulcer
3. Colloid carcinoma
4. Carcinoma secondary to a chronic gastric ulcer
5. Leather-bottle stomach

Most common site is the prepyloric region; but those occurring in pernicious anaemia tend to be fundal and polypoid.

Spread

1. Direct spread - transverse colon, pancreas etc.
2. Lymphatic spread - to lymph nodes around stomach and coeliac axis
3. Blood stream - to liver, lungs
4. Transperitoneal spread - to pelvic peritoneum

Symptomatology

May present as dyspepsia of recent onset, or as progressive loss of weight with anaemia and anorexia. Sometimes it may present as pyloric obstruction or as an epigastric lump. Occasionally the primary may be silent and the first evidence of disease may be from its metastasis to other area, e.g. jaundice, or ascites.

Investigations

The most useful one is endoscopic biopsy. Others that may be helpful are acid secretion studies and cytology after brushing the tumour.

Treatment

For operable tumours, lower radical partial gastrectomy, upper radical partial gastrectomy or total gastrectomy may be required, depending on the site of the tumour.

For inoperable tumours, palliative gastro-jejunostomy or exclusion gastro-jejunostomy may be performed.

Chemotherapy

5-Fluorouracil, Mitomycin C and BCNU have been found to be of some use in prolonging remissions. Adjuvant chemotherapy is now under trial.

GASTROINTESTINAL BLEEDING

I. COMMON CAUSES OF GASTROINTESTINAL BLEEDING

Upper gastrointestinal:

1. Duodenal ulcer, chronic and acute
2. Gastric ulcer, chronic and acute
3. Oesophageal varices
4. Gastritis
5. Gastric carcinoma

Lower gastrointestinal:

1. Haemorrhoids
2. Anal fissure
3. Colorectal carcinoma
4. Colitis
5. Diverticulosis
6. Colonic polyps

UNCOMMON CAUSES OF GASTROINTESTINAL BLEEDING

Upper gastrointestinal:

1. Gastric leiomyoma, leiomyosarcoma
2. Mallory-Weiss syndrome
3. Aortoduodenal fistula
4. Stomal ulcer
5. Haemobilia
6. Meckel's diverticulum
7. Angiodysplasia
8. Bowel gangrene - ischaemia, intussusception

Lower gastrointestinal:

1. Angiodysplasia
2. Bowel gangrene - ischaemia, volvulus

II. DIAGNOSIS OF GASTROINTESTINAL BLEEDING

Symptoms:

1. Haematemesis (UGI)
2. Coffee-ground vomitus (UGI)
3. Ulcer pain, vomiting (UGI)
4. Melaena, tarry stool (UGI)
5. Fresh bleeding per rectum (LGI)
6. Change in bowel habits (LGI)

Physical findings:

1. Tachycardia
2. Hypotension, shock
3. Melaena, tarry stool (UGI)
4. Fresh blood or coffee-ground material aspirated from Ryle's tube (UGI)
5. Fresh blood found on rectal examination (LGI)
6. Abdominal masses, ascites

III. INITIAL MANAGEMENT

1. Estimate amount of blood loss
2. Complete blood picture, RFT, LFT, PT, PTT
3. Type and crossmatch for blood
4. Start intravenous drip - volume replacement
5. Insert Ryle's tube and perform ice saline lavage (UGI)
6. Monitor blood pressure and pulse frequently (every ½ hr)
7. Nil by mouth

If bleeding is substantial - continuous or with tachycardia and hypotension:

8. Insert CVP line and monitor CVP
9. Insert Foley's catheter and monitor urine output
10. Monitor intake and output

IV. INITIAL INVESTIGATIONS

1. Suspected UGI bleeding - upper endoscopy to inspect:
   - oesophagus
   - gastro-oesophageal junction
   - stomach
   - duodenum

2. Suspected LGI bleeding - proctoscopy
   - sigmoidoscopy
   - superior and inferior mesenteric angiograms

V. EMERGENCY SURGICAL TREATMENT

Emergency operations indicated if:

1. High rate of blood loss
2. Continuous blood loss
3. Rebled after initially stopping

Common emergency operations performed:

Pathology	Operation
Oesophageal varices	
Duodenal ulcer	Truncal vagotomy, pyloroplasty and plication of bleeding vessel
Gastric ulcer	Partial gastrectomy, gastroduodenostomy
Gastritis	Truncal vagotomy, pyloroplasty
Gastric carcinoma	Subtotal gastrectomy, gastrojejunostomy
Mallory-Weiss syndrome	Plication of tear
Haemorrhoids	Haemorrhoidectomy
Anal fissure	Excision and plication
Diverticulosis	Partial colectomy
Angiodysplasia of colon	Partial colectomy

VI. NON-OPERATIVE TREATMENT FOR GASTROINTESTINAL BLEEDING

1. Pitressin infusion - systemic
   regional (selective)
   - effective in variceal bleeding, gastritis and diverticulosis
2. Embolisation
3. Endoscopic diathermy

VII. FURTHER INVESTIGATIONS

1. Coeliac angiogram
2. Upper and lower gastrointestinal series
3. Colonoscopy

BENIGN DISEASE OF THE BILIARY TRACT

Bile

500-1500 ml/day

Contains: bile salts
lecithin
cholesterol
bilirubin
fatty acid
inorganic salts

Regulation of Flow

Regulated by: hepatic secretion
*gallbladder contraction
*choledochal sphincteric resistance

*cholecystokinin-pancreozymin (CCK-PZ)

Enterohepatic Circulation

Entire bile salt pool 2-4 g.
Primary bile salts

cholate	40%
chenodeoxycholate	40%

Secondary Bile Salts

deoxycholate	20%
lithocholate	

Resorption of Bile Salts

By terminal 200 cm of ileum
95% reabsorbed

6-8 cycles/day
Normal loss 10-20%

Gallstones

Cholesterol gallstones - pure cholesterol stones
mixed cholesterol stones

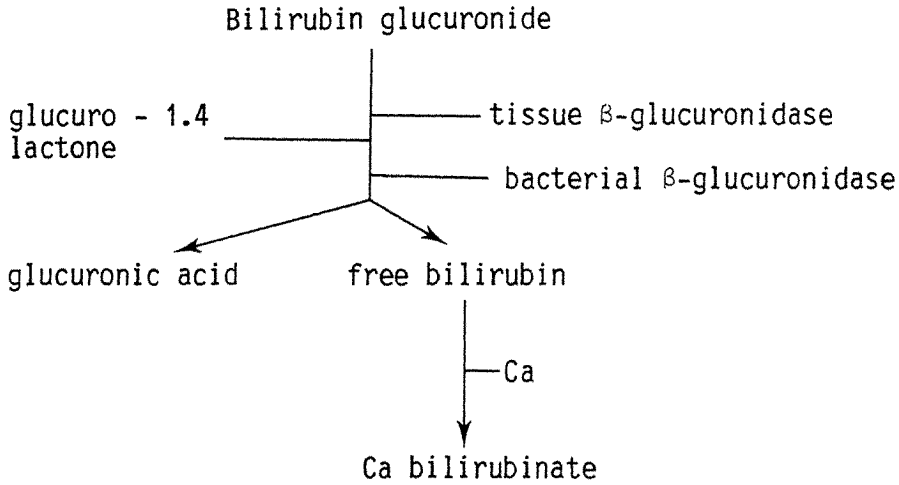
Pigment stones - bilirubin stones (black stones)
calcium bilirubinate stones - RPC

Rare stones - calcium carbonate stones
fatty acid - calcium stones

Formation of Cholesterol Gallstones

Cholesterol-holding capacity of micelles
Cholesterol secretion
Bile salt pool

Formation of Calicium Bilirubinate Stones



Diagnostic Examination of the Biliary Tree

Plain Abdominal Film

10-15% of gallstones are opaque
air in biliary tree

Oral Cholecystogram

Tyropanoate or iopanic acid taken the night before.

Cause of non-opacification

1. poor absorption - ileus, vomiting or diarrhoea
2. poor excretion - bilirubin level over 40 μmol/l

Highly reliable (>95% true positive)

Intravenous Cholangiogram

Iodipamide given IV
X-ray with tomography
Not satisfactory if bilirubin is raised
Major bile ducts, gallbladder opacified
Not reliable in studying gallbladder

Endoscopic Retrograde Cholangiopancreatography (ERCP)

Endoscopically cannulate the bile and/or pancreatic duct
Requires expertise
Ampulla visualised
Upper ducts not visualised if there is common duct obstruction

Complications : hyperamylasaemia 8%, acute pancreatitis 1%
introduce infection - cholangitis

Percutaneous Transhepatic Cholangiography (PTC)

Direct needle puncture of intrahepatic duct
Requires dilated ducts
Lower part of duct not visualised if there is common duct obstruction

Complications : bleeding, bile leak - peritonitis, introduce infection

Ultrasonography

Can detect : gallbladder stones
dilated ducts (intrahepatic or extrahepatic)
space-occupying lesions in liver

>90% accurate in expert hands for gallstones

Asymptomatic Gallbladder Stones

30-50% of all patients with gallbladder stones come to surgery

Indications for surgery

1. diabetes mellitis
2. non-visualising gallbladder on OC
3. large stones (>2 cm)
4. many small stones
5. a calcified gallbladder

Gallstones and Chronic Cholecystitis

Symptoms and signs : biliary colic, fatty food intolerance
Investigations : OC - gallbladder stones or non-opacification
ultrasound

Treatment - cholecystectomy, operative cholangiogram
dissolution of stones?

Complications - acute cholecystitis, common duct stones, Ca gallbladder

Acute Cholecystitis, Empyema

Symptoms and signs: acute RUQ pain, fever, nausea and vomiting,
Murphy's sign

Investigations: leucocytosis, mildly elevated bilirubin, and
alkaline phosphatase
ultrasound

Differential Diagnosis

perforated peptic ulcer
acute pancreatitis
acute appendicitis
acute viral hepatitis

Treatment: conservative - mild symptoms
(IV fluids + antibiotics)

surgery - severe symptoms, palpable tender gallbladder

Complications : empyema,
perforation - pericholecystic abscess
free perforation
cholecystenteric fistula - gallstone ileus

Gallstone Ileus

Females
Average age - 70

Symptoms and Signs

- that of bowel obstruction
- history compatible with acute cholecystitis (30%)

Investigation

Plain X-ray abdomen - gallstones in bowel, air in biliary tree, dilated small bowel loops and air fluid levels

Treatment : emergency laparotomy and removal of stones
cholecystectomy later

Choledocholithiasis

Primary - formed in bile ducts - RPC
Secondary - formed in gallbladder

Recurrent Pyogenic Cholangitis

Aetiology

Bacterial infection characterised by

1. high incidence of mixed infections (up to 5 species of bacteria)

2. 30% incidence of anaerobic infection

Bacteria Involved

Aerobic:	E. coli	66%
	Klebsiella aerogenes	54%
	Streptococcus faecalis	30%
	Pseudomonas species	14%
Anaerobic:	Clostridia species	24%
	Bacteroides fragilis	8%

Portal of Entry

1. ascending via ampulla
2. haematogenous via hepatic artery, portal vein
3. via lymphatics

Clinical features

Sex : male=female
Age : 40's

Fever, jaundice, RUQ or epigastric pain

Liver palpable	50%
Gallbladder palpable	30%
Spleen palpable	20%
Rebound tenderness	5%

Investigations

Blood test - obstructive jaundice, hyperamylasaemia
Ultrasonography - gallstones, dilated duct, liver abscess

Treatment Conservative - IV fluids, antibiotics, nil by mouth

Surgery - empyema of gallbladder, liver abscess, impending sepsis, no improvement with conservative treatment.

Other investigations - ERCP, PTC

Principles of Treatment

Cholecystectomy, removal of all stones from bile duct, relief of all biliary stricture

Prognosis

About 20% will require reoperaton due to recurrence of cholangitis

Uncommon Causes of Biliary Obstruction or Cholangitis

1. biliary atresia
2. choledochal cyst
3. parasitic infection - clonorchiasis, ascariasis
4. pancreatitis
5. sclerosing cholangitis - ulcerative colitis 25%
   thyroiditis
   retroperitoneal fibrosis

LIVER ABSCESSES

Liver abscesses

Pyogenic - cholangitic
- non-cholangitic

Amoebic

Diseases predisposing non-cholangitic pyogenic abscess :

ulcerative colitis
acute appendicitis
acute diverticulitis
perforated peptic ulcer

Symptoms and Signs

fever, chills and rigor
jaundice
hepatomegaly

Investigations :

ultrasonography, CAT scan
ERCP
anti-amoebic titre

Treatment

amoebic abscess - flagyl

pyogenic abscess

solitary or large
- external drainage + antibiotics

multiple small
- antibiotics

cholangitic abscess
- common duct exploration and drainage

non-cholangitic abscess
- remove primary focus

Liver cysts

Simple cyst
Polycystic liver
Hydatid cyst
Neoplastic cyst - cystadenoma

Simple cyst, polycystic liver

congenital
arises from bile duct elements

Treatment of simple cyst

excision
fenestration

Hydatid disease

Clinical features - exposure in endemic areas
RUQ pain
hepatomegaly
jaundice

Treatment of hydatid cyst

Excision

Deroofing and omentopexy
after sterilization (25% NaCl)
(0.25% $AgNO_3$)

Common duct exploration -
if rupture into bile ducts has occurred

NEOPLASMS OF THE LIVER, BILIARY TRACT AND PANCREAS

NEOPLASMS OF THE LIVER

Benign : Adenoma - no ductal elements
Focal nodular hyperplasia
Haemangioma

Malignant : Hepatocellular carcinoma
Sarcomas
Carcinoid tumour

Adenoma

Focal Nodular Hyperplasia

More common in females, associated with steroid intake, malignant potential.
Some regress with withdrawal of contraceptives.

Hepatocellular Carcinoma

Second most common cancer in Hong Kong.

Male : Female = 4:1

80% HBsAg positive, 80% cirrhotic.

Pathology

nodular form
massive form
diffuse form
encapsulated 5-15%

Symptoms and Signs

abdominal distention, epigastric mass, jaundice
epigastric, RUQ discomfort, shock with haemoperitoneum - from rupture.

Blood Investigations

CBP, LFT, RFT
serum alphafetoprotein (80%)
serum HBsAb, HBsAg
BSP retention test

Other Investigations

hepatic arteriogram, superior mesenteric arteriogram
- look for portal vein invasion.
ultrasonography, CAT scan
peritoneoscopy

Peritoneoscopy

to assess resectability, to assess condition of liver,
to perform biopsy under visual guidance

Prerequisite for Surgical Resection

adequate liver function reserve
assessed by - BSP retention, serum albumin, PT, APTT.
tumour confined to one lobe
no metastatic spread

Surgery for HCC

to provide access for regional chemotherapy ± implantable pump
for rupture of tumour - plication, packing
ligation of hepatic artery
surgical resection for cure

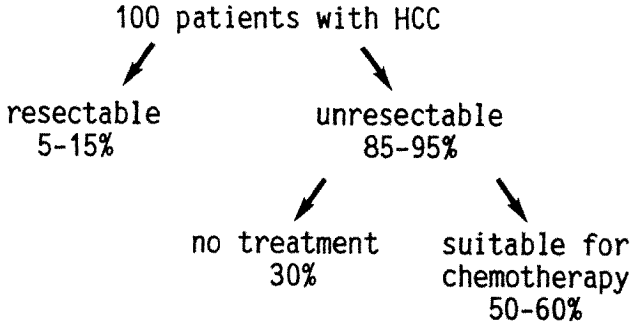
Nonoperative Treatment

systemic chemotherapy - adriamycin 25% response
- interferon 25% response

regional chemotherapy - by cannulation of hepatic artery
25% response

embolisation - for rupture
- as definite treatment

radiotherapy - ineffective



NEOPLASMS OF THE BILIARY TRACT

Benign : Cystadenoma

Malignant : Gallbladder - adenocarcinoma
- squamous cell ca

Bile ducts - cholangiocarcinoma
- cystadenocarcinoma
- mucoepidermoid carcinoma

Adenocarcinoma of the Gallbladder

old females, 85% associated with gallstones,
usually presented late with advanced carcinoma.

Treatment

cholecystectomy - for those discovered incidentally and confined to mucosa

cholecystectomy, partial hepatectomy,
regional node dissection - for more advanced tumour

most are unresectable at presentation

Other Therapies

chemotherapy - ineffective
radiotherapy - ineffective

Prognosis

poor - 5% 5-year survival
best prognosis are those incidentally discovered at cholecystectomy for gallstones and confined to mucosa.

Cholangiocarcinoma

more than 80% associated with clonorchiasis

Symptoms and Signs

jaundice, palpable gallbladder - for lower CBD tumour
haemobilia, fever and sepsis

Blood investigations

CBP, RFT, LFT, CEA

Other investigations

ERCP - brushings of duct
bile for cytology

PTC

Ultrasonography and CAT scan

Treatment

few are resectable
curative treatment - by resection
for lower CBD tumours - Whipple's operation
for hilar and intrahepatic tumours - partial hepatic resection

Palliative treatment

Non-operative

insertion of endoprosthesis - endoscopically
- percutaneously

Operative

hepaticojejunostomy
cholangiojejunostomy

Other treatment

chemotherapy - ineffective
radiotherapy - localised radiotherapy
effective for palliation

Carcinoma of the Ampulla

Symptoms and signs

jaundice, palpable gallbladder, UGI bleeding, fever and sepsis.

Investigations

upper endoscopy - ampullary biopsy
ERCP
PTC
hypertonic duodenography

Treatment

curative treatment
local excision - for old individuals with small tumours
Whipple's operation
30% 5-year survival

Palliative treatment

Nonoperative - endoscopic papillotomy
- insertion of endoprosthesis endoscopically

Operative - hepaticojejunostomy

NEOPLASM OF THE PANCREAS

Benign : adeoma
cystadenoma

Malignant : adenocarcinoma duct cell
acinar cell
cystadenocarcinoma
carcinoid tumour
islet cell tumours
gastrinoma, insulinoma, glucagonoma, others.
others

Adenocarcinoma of the Pancreas

head 73%, body 20%, tail 7%
most are advanced on presentation

Symptoms and signs

weight loss, jaundice, pain, anorexia, nausea and vomiting, pruritus, abdominal mass, palpable gallbladder.

Blood investigation

CBP, LFT, RFT, serum CEA.

Other investigations

barium meal, upper endoscopy, ERCP,
ultrasonography and CAT scan
celiac angiography - 70% curative
percutaneous aspiration cytology
ERCP + collection of pancreatic juice for cytology - 90% accurate

Treatment

curative treatment
- partial pancreatectomy + splenectomy for tumour of tail
- Whipple's operation for tumour of head

Palliative treatment

Operative - bypass operations (triple bypass)

Nonoperative - radiotherapy
intraoperative, implantation

- chemotherapy
ineffective

Islet Cell Tumours

Symptoms and signs

gastrinoma - recurrent ulcers
insulinoma - hypoglycaemia

Investigations

blood - serum gastrin, serum insulin
other - CAT scan, arteriography

Treatment

gastrinoma - resection of pancreas if localised
- resection of stomach if disseminated
- cimetidine, ranitidine

other islet cell tumours - resection if localised
- chemotherapy with streptozotocin and 5-fluorouracil, if widespread

PORTAL HYPERTENSION AND SPLEEN

I. CLASSIFICATION OF PORTAL HYPERTENSION

1. Extrahepatic Presinusoidal Block

a. Infantile or neonatal umbilical vein sepsis
b. Pyelophlebitis from intra-abdominal sepsis
c. Malignant tumour invading portal vein
d. Previous splenectomy
e. Idiopathic

2. Intrahepatic Perisinusoidal Block

a. Portal space infiltration e.g., Hodgkin's disease, Schistosomiasis
b. Sarcoidosis
c. Congenital hepatic fibrosis
d. Cirrhosis

3. Intrahepatic Postsinusoidal Block

1. Cirrhosis : alcoholism, post necrotic, biliary
2. Wilson's disease
3. Haemochromatosis
4. Veno-occlusive disease

4. Extrahepatic Postsinusoidal Block

a. Obstruction of the hepatic vein
b. Obstruction of the vena cava
c. Cardiac disease e.g., constrictive pericarditis, congestive heart failure

5. Arteriovenous Shunt

Aetiology of Portal Hypertension

1. Changes secondary to portal vascular bed block
2. Presence of regenerating nodules
3. Transmission of hepatic arterial pressure

Normal free portal vein pressure 15-20 cm H_2O

Portal hypertension > 25 cm H_2O

Methods of Measuring Portal Vein Pressure

1. Directly at laparotomy
2. Transumbilical portal pressure
3. Percutaneous splenic pulp pressure
4. Wedge hepatic venous pressure
5. Transhepatic portal pressure

Clinical Features

1. Jaundice
2. Splenomegaly
3. Ascites
4. Peripheral oedema
5. Spider naevi
6. Caput medusa
7. Palmar erythema
8. Clubbing
9. Muscle wasting
10. Testicular atrophy
11. Gynaecomastia
12. Alopecia

Investigations

1. Complete blood picture
2. Liver function test
3. Prothrombin index
4. Electrolytes
5. Barium swallow
6. Fibreoptic upper endoscopy
7. Superior mesenteric arteriogram/splenoportovenogram

CHILD'S CLASSIFICATION OF HEPATIC FUNCTIONAL RESERVE

(Clinical and laboratory classification of patients with cirrhosis).

Group Designation	A (minimal)	B (moderate)	C (advanced)
serum bilirubin (mg%)	below 2.0	2.0-3.0	over 3.0
serum albumin (gm%)	over 3.5	3.0-3.5	under 3.0
ascites	none	easily controlled	poorly controlled
neurologic disorder	none	minimal	advanced coma
nutrition	excellent	good	poor, "wasting"

II. MANAGEMENT OF OESOPHAGEAL VARICEAL BLEEDING

Resuscitation
Intravenous fluid replacement (C.V.P. monitoring, if necessary)
Emergency upper endoscopy
Sedate and reassure
Anti-hepatic coma regime (in patients with poor hepatic reserve)
- Gastric lavage
- Enema
- Neomycin
- Lactulose

Parenteral Vitamin K

1. Medical (Non-operative) Therapy

Intravenous pitressin
Oesophagogastric tamponade
Injection sclerotherapy of varices

Pitressin therapy
- 20 units in 200 ml 5% Dextrose intravenously in 20 mins
- Decreased splenic blood flow
- Contraindicated in ischaemic heart disease

Oesophagogastric tamponade
- Blakemore sengstaken's tube (3 lumen)
- Minnesota tube (4 lumen)

Injection sclerotherapy
- Rigid Negus oesophagoscope (general anaesthesia)
- Flexible endoscope (local anaesthesia)
- Sclerosant: Ethanolamine oleate
  Sodium tetradecylsulphate

Complications of sclerotherapy
- Pyrexia
- Tachycardia
- Retrosternal discomfort
- Perforation of oesophagus
- Perioesophageal leakage with mediastinitis
- Oesophageal stricture
- Mucosal sloughing and ulceration

Advantages
- Avoid emergency surgical procedure
- Can be repeated
- Effective even after shunting procedure
- Can be done under local anaesthesia

Disadvantages
- Palliative procedure
- Not useful for fundic varices

2. Surgical Therapy

a. Transthoracic ligation of varices
b. Transabdominal plication of varices
c. Porta azygos disconnection
d. Oesophageal transection
e. Porta systemic shunt

Porta Systemic Shunt

Emergency versus Elective
Prophylactic versus Therapeutic

Types

1. Portacaval shunt
   - end to side
   - side to side
   - double

2. Splenorenal shunt
   - end to side
   - side to side
   - distal

3. Mesocaval shunt
   - end to side
   - interposition

4. Renoportal shunt

Complications

1. Post shunt encephalopathy
2. Thrombosis of shunt
3. Peptic ulceration

III. SPLEEN

Indications for Splenectomy

1. Haematological disorder
2. 1^0 or 2^0 hypersplenism
3. Others - ruptured spleen, etc.

Haematological Disorders

1. Hereditary spherocytosis
2. Hereditary elliptocytosis
3. Thalassaemia major
4. Idiopathic thrombocytopenic purpura
5. Autoimmune haemolytic anaemic
6. Myelofibrosis
7. Staging for Hodgkin's lymphoma

1^{o} Hypersplenism

1. Splenomegaly, pancytopenic hyperplastic marrow
2. No obvious secondary aetiology detectable
3. Good response to splenectomy

2^{o} Hypersplenism

1. Primary liver disease
2. Extrahepatic portal or splenic vein obstruction
3. Collagen disease
4. Haematological disorder
5. Acute infection
6. Chronic infection
7. Miscellaneous cause e.g., amyloidosis

Other Indications for Splenectomy

1. Splenic trauma
2. Pathological rupture of spleen
3. Splenic cyst
4. Part of cancer operation

Haematological Consequences of Splenectomy

1. Increased white cell count
2. Increased platelets
3. Increased number of abnormal looking red blood cell count

Complications of Splenectomy

1. Immediate
   - Pneumothorax
   - Bleeding
   - Subphrenic abscess
   - Inadvertent damage to stomach, pancreas
   - Venous thrombosis

2. Late
   Fatal sepsis in children - pneumococcal infection (2-7%)
   Recurrent infection (5x normal)

Rupture of Spleen

Classification

1. Immediate death - shock
2. Initial shock - recovered, signs of ruptured spleen
3. Delayed type (up to days after trauma)

Diagnosis

Trauma to left upper quadrant
Hypotension
Tachycardia
Haemoperitoneum
Peritonitis

Radiological Features

Fracture to left lower ribs
Obliteration of splenic outline
Obliteration of Psoas shadow
Indentation of left side of stomach air bubble
Elevation of left hemidiaphragm
Free fluid between gas filled intestinal loops

Clinical Features

Kehr's sign
Ballance sign

Treatment

Splenectomy
Autotransfusion

DISEASES OF THE PANCREAS

Pancreatitis

Aetiology

gallstones	55%
alcohol	15%
unknown	15%

Others

hyperlipidaemia
hypercalcaemia
drug induced - corticosteroids
thiazides
azathioprine
contraceptives
postoperative
tumours
parasites
vascular

Pathogenesis

1. Obstruction - secretion

   Obstruction alone
   - exocrine atrophy

   Obstruction + stimulation
   - pancreatitis

2. Reflux - common channel theory

   Bile reflux, especially infected bile, duodenal juice reflux

3. Vascular - anoxia

Pancreatitis

Acute pancreatitis - no permanent histological change
Chronic pancreatitis - permanent histological change has occurred

Acute Pancreatitis

Symptoms and signs
- epigastric pain radiating to back
- nausea and vomiting
- dehydration, tachycardia
- abdominal tenderness
- decreased or absent bowel sounds
- abdominal mass
- Grey Turner's sign, Cullen's sign

Laboratory Findings

serum amylase > 1000 μmol/min.l

serum amylase also elevated in the following acute abdominal conditions :
- small bowel obstruction
- mesenteric infarction
- perforated ulcer
- gangrenous cholecystitis

leukocytosis
bilirubin, alkaline phosphatase
SGOT, SGPT, LDH
urinary amylase

Urinary amylase - creatinine clearance ratio > 5%

$$\frac{\text{Urine amylase}}{\text{Serum amylase}} \times \frac{\text{Serum creatinine}}{\text{Urine creatinine}} \times 100\%$$

Other non-acute conditions with raised serum amylase

- renal failure
- chronic sialadenitis
- salivary tumours
- ovarian tumours
- liver disease

Investigations

Plain X-ray abdomen - sentinel loop
colon cut-off sign
gallstones

Ultrasonography - gallstones, dilated bile ducts
collections of pus or fluid

ERCP after recovery

Factors of Poor Prognosis :

1. Age > 50
2. Leucocytosis > 19,000/mm^3
3. low pO_2
4. elevated serum creatinine
5. elevated serum glucose
6. low serum calcium
7. presence of methaemalbumin in serum
8. presence of DIC

Medical Treatment :

1. Nasogastric suction, if nauseated
2. IV fluid replacement
3. Analgesics
4. Anticholinergics - atropine?
6. Calcium replacement
7. Oxygen
8. Antitrypsin enzymes - aprotinin?
9. Somatostatin?

Surgical Treatment :

1. For biliary pancreatitis

   should be done within 10 days after complete resolution of symptoms
   30% with another attack within 3 months

   Procedure

   cholecystectomy,
   common duct exploration and T-tube drainage,
   $\pm$ sphincteroplasty

2. for pancreatic abscess

3. for haemorrhagic pancreatitis diagnosed by :
   a. $\uparrow$ serum methaemalbuminemia
   b. Cullen's or Grey Turner's signs
   c. DIC
   d. $\downarrow$ haemoglobin

   Procedure

   partial or complete excision of pancreas
   drainage

4. for pancreatic pseudocyst, diagnosed by

   a. epigastric mass
   b. ultrasonography, CAT Scan

   Procedure

   cystogastrostomy
   cystoduodenostomy
   cystojejunostomy
   external drainage

5. for other complications
   haemorrhage
   fistula

Miscellaneous Methods of Treatment

1. Peritoneal lavage for severe acute pancreatitis

   some early improvement in renal function and PO_2

   die later from septic complications

2. Nutrition

   a. via enteral tube in jejunum
   b. total parenteral nutrition

Chronic Pancreatitis

Aetiology

alcoholism
hypercalcaemia
hyperlipidaemia
familial pancreatitis

Symptoms and Signs :

persistent or recurrent abdominal pain
pancreatic insufficiency

Laboratory and investigational findings :

diabetes mellitus
calcifications on X-ray
ERCP
secretin - CCK stimulation test

Medical Treatment :

- analgesics
- enzymes
- insulin

Surgical Treatment :

- pancreatectomy
- internal drainage of pancreatic duct
- sphincteroplasty

BENIGN CONDITIONS OF THE COLON, RECTUM AND ANUS

I. HAEMORRHOIDS

Dilated veins formed by radicles of the superior, middle and inferior rectal veins.

Types

1. Internal haemorrhoid
2. External haemorrhoid
3. Internal-External haemorrhoid

Associated conditions

1. Carcinoma of rectum
2. Pregnancy
3. Straining at micturition
4. Chronic constipation

Clinical features

1. Bleeding
2. Prolapse
3. Discharge
4. Pain
5. Anaemia

Degrees of haemorrhoid

1. First degree - no prolapse
2. Second degree - prolapse with spontaneous reduction
3. Third degree - prolapse with manual reduction
4. Fourth degree - irreducible prolapse

Complications

1. Profuse haemorrhage
2. Strangulation
3. Thrombosis
4. Ulceration

Treatment

1. Conservative treatment - diet and suppository
2. Injection - 5% phenol in almond oil
3. Rubber band ligation
4. Cryosurgery
5. Haemorrhoidectomy

II. THROMBOSED EXTERNAL HAEMORRHOID

Thrombosis of the subcutaneous external haemorrhoidal veins of the anal canal.

Clinical features

1. Sudden pain
2. Lump in the anus

Treatment

1. Conservative - analgesic
   - suppository
2. Evacuation of blood clot

III. ANORECTAL ABSCESS

Organisms

1. E. coli
2. Staph. aureus
3. Proteus

Aetiology

1. Infection of an anal gland (90%)
2. Blood borne infection
3. Extension of a cutaneous boil

Types

1. Perianal
2. Ischiorectal abscess
3. Submucous abscess
4. Pelvirectal abscess

Clinical features

1. Pain
2. Swelling
3. Fever

Treatment

1. Incision and drainage
2. 1-stage or 2-stage

Prognosis

Abscesses that rupture spontaneously or are drained without removing the fistulous connection will frequently recur until the underlying cause is removed.

IV. ANORECTAL FISTULA

It is a tract lined by granulation tissue communicating between the anal canal and the skin. Most of them originate in the anal crypts at the anorectal junction. The crypt becomes infected and the infection extends along one of the several well-defined planes, and an abscess occurs. When the abscess is opened or when it ruptures, a fistula is formed.

Types

1. Low level - subcutaneous
   submucosal
   intermuscular
   ischiorectal

2. High level - very rare
   iatrogenic

Clinical features

Persistent seropurulent discharge from an opening around the anus.

Treatment

Lay open the fistula tract and allow it to heal by granulation

V. ANAL FISSURE

1. Acute - a superficial tear
2. Chronic - a chronic ulcer at the anal verge with exposure of the underlying fibres of the internal sphincter

Clinical feature

Pain in anus

Differential diagnosis of anal pain

1. Perianal haematoma
2. Anal Fissure
3. Strangulated haemorrhoid
4. Anorectal abscess

Treatment

1. Analgesic
2. Laxative
3. Local ointment or suppository
4. Digital dilatation
5. Sphincterotomy

VI. RECTAL PROLAPSE

Partial prolapse

1. Prolapse of the mucous membrane and submucosa of the rectum
2. Occurs more often at the extremes of life

Complete prolapse

1. Prolapse of all layers of the rectal wall
2. More common in females, associated with prolapse of the uterus

Treatment

1. Partial prolapse : Conservative treatment
   Submucous injection of phenol
   Thiersch operation
2. Complete prolapse : Poor risk patient - Thiersch operation
   Good risk patient - Transabdominal fixation of rectum

VII. POLYPS OF COLON AND RECTUM

Type

1. Neoplastic : Adenoma - Tubular adenoma
   - Tubulovillous adenoma
   - Villous adenoma
   Carcinoma
2. Hamartomas : Juvenile polyp
   Peutz-Jeghers polyp
3. Inflammatory : Inflammatory polyp (pseudopolyp)
   Benign lymphoid polyp
4. Unclassified : Hyperplastic (metaplastic) polyp

Much evidence to support adenoma - carcinoma transformation

Clinical feature

Fresh blood per rectum

Differential diagnosis of rectal bleeding

1. Haemorrhoid
2. Carcinoma of rectum and colon
3. Polyp
4. Colitis - bacterial, amoebic, ischaemic
5. Other inflammatory conditions, e.g. ulcerative colitis diverticular disease

Diagnosis

1. Barium enema
2. Colonoscopy

Treatment

Colonoscopic polypectomy

VIII. SIGMOID COLON VOLVULUS

Predisposing factors

1. Redundant sigmoid colon
2. Long pelvic mesocolon with narrow attachment
3. Band of adhesion

Clinical feature

Symptom and sign of intestinal obstruction

Diagnosis

Typical plain X-ray abdominal film showing a large distended loop of bowel with no haustral marking arising up out of the pelvis to as high as the diaphragm.

Treatment

Viable colon : decompression by sigmoidoscope (rigid or flexible) followed by elective colectomy
Nonviable colon : emergency resection

IX. OTHER BENIGN CONDITIONS

Ulcerative colitis
Diverticular disease
Crohn's disease

MALIGNANT CONDITIONS OF THE COLON, RECTUM AND ANUS

I. INTRODUCTION

The incidence of carcinoma of the colon and rectum is rising in developed countries. In Hong Kong, it is the third most common cause of death. Each year, about 13 per 100,000 men and 9 per 100,000 women die of a lower G.I. malignancy.

The aetiology of sporadic cases has been attributed to dietary and other environmental factors. Known predisposing conditions include ulcerative colitis, familial polyposis, villous adenoma, and a previous colorectal cancer.

Approximately 60% of all lesions are situated in the lower sigmoid and rectum. Sigmoid and caecal carcinomas are the next most common sites. There is a recent trend towards more colonic lesions, perhaps due to improved diagnostic aids in their detection.

II. PATHOLOGY

Adenocarcinoma spreads by lymphatic routes, direct invasion to contiguous structures, and by haematogenous metastases to the liver, lung, brain and other sites.

Staging by TNM classification

Stage

1. within bowel wall
   a. to submucosa
   b. to muscularis propria
2. beyond muscularis propria
3. lymph nodes involved
4. any distant metastases

III. SYMPTOMS

Local :

- Pain → perforation
- Diarrhoea
- Constipation → obstruction
- Positive occult blood in stool → bleeding

Right colon lesions
- Weakness, anaemia
- Vague discomfort
- Mass
- Obstruction-perforation

Left colon lesions
- Altered bowel habits
- Blood in stool
- Obstruction

Rectal lesions
- Bleeding
- Altered bowel habits → tenesmus

Systemic :

- ↓ weight
- ↓ appetite
- ↓ energy
- Dyspnoea
- Ascites, oedema

IV. INVESTIGATIONS

Proctosigmoidoscopy

Asymptomatic cancer	: 0.2 - 1.0%
Five years survival	: symptomatic cases - 50%
	asymptomatic cases - 88%
Perforations	: 1 in 20,000
Deaths	: 0

Barium enema

False-positive CA	: up to 21%
False-negative CA	: up to 15%

Colonoscopy

Detects 42% of polyps missed by barium enema
30% of carcinomas with a prior unsatisfactory barium enema

Complications :	perforation	0.2 - 1.9%
	bleeding	0.9%
	death	0.13%

V. SURGERY

Surgical excision remains the most effective treatment for primary localised carcinoma of the colon and rectum. In addition to removing the primary tumour, the entire regional lymph nodes draining the area of the carcinoma must be resected. It must be emphasised that it is the removal of the draining lymph nodes that determines the extent of bowel resection.

Colonic lesions may be resected with restoration of intestinal continuity. Rectal lesions as low as 6 cm from the anal verge may be resected with preservation of the anal sphincter. However, more distal rectal lesions may not permit an adequate and safe distal margin to allow preservation of the anal sphincters. In such rectal cancers, an abdomino-perineal resection with a permanent end colostomy is usually required.

Bowel preparation. Mechanical bowel preparation and the use of antibiotics are essential to reduce septic complications and anastomotic leakage. This involves dietary measures as well as either repeated enemas or whole-gut irrigation. Systemic antibiotics should include antibiotics directed against gram-negative organisms (e.g., aminoglycosides) and anaerobic bacteria (e.g., metronidazole).

VI. SURVIVAL

5-year survival

I - 80%
II - 65%
III - 30%
IV - 5%

VII. POST-OPERATIVE MANAGEMENT

Follow-up
Colostomy care
Hb, LFT's, sigmoidoscopy
Serial CEA
Colonoscopy, barium enema
Liver scan, CT scan, hepatic angiography

VIII. CHEMOTHERAPY

Adjuvant versus therapeutic
5 FU, CCNU, others
Regional hepatic perfusion

IX. RADIOTHERAPY

Adjuvant versus therapeutic
Pre-, post- and intra-operative

X. CARCINOMA OF THE ANUS

Types : epidermoid
Basal cell (transitional) carcinoma
malignant melanoma
Bowen's disease
Paget's disease

XI. TREATMENT

Local resection plus minor radiotherapy
AP resection

ACUTE ABDOMEN, INCLUDING APPENDICITIS

ACUTE ABDOMEN

I. Definition

Any condition requiring rapid decision making and operative intervention.

- Peritonitis - localised/diffused
- Intestinal obstruction if untreated
- Trauma - blunt/penetrating

II. Common Lesions Requiring Emergency Operation

Trauma	58%
Acute appendicitis	16%
Intestinal obstruction	15%
Perforated ulcer	3%
Acute cholecystitis	2%
Others	6%

III. Common Problems Mimic Acute Abdomen

1. Gynaecological lesions
   - Salpingitis
   - Ectopic pregnancy
   - Ovarian cyst - ruptured/twisted
2. Renal lesions
   - Ureteral stones
   - Pyelonephritis
3. Lesions of the gastrointestinal tract
   - Acute gastroenteritis
   - Mesenteric adenitis
   - Typhoid fever

IV. History

1. Mode of onset of pain
2. Character of pain
   - Localisation - with/without shift
   - Rapidity of onset
   - Constant/colicky
   - Radiation
3. Associated symptoms
   - Anorexia, nausea, vomiting
   - Diarrhoea, constipation
   - Chills/fever
   - Symptoms of pelvic inflammatory diseases, pregnancy (female patients with suspected appendicitis)

V. Physical Examination

1. Inspection - General condition
   Male genitalia
   Hernial orifice
2. Vital signs - Blood pressure, pulse, respiratory rate,
   Temperature
3. Cough, tenderness
4. Spasm
5. Localisation of maximal tender site e.g., McBurney's point
6. Costovertebral tenderness
7. Deep palpation
8. Rebound tenderness
9. Rectal and pelvic examination
10. Special signs for Acute Appendicitis
    - The followings depend on stretching the posterior abdominal muscles together with the overlying inflamed peritoneum
    a. Cope's sign - Obturator internus
    b. Psoas sign - psoas muscle

VI. Laboratory Investigations

Blood analysis : Haemoglobin, white-cell count, amylase, renal and liver functions, blood gases

Urine analysis

Peritoneal fluid:

a. Four quadrant abdominal paracentesis
b. Peritoneal lavage
   - Invasive investigation but of great value in assessment of suspected haemoperitoneum from trauma
   - False-positive rate - high

VII. Radiological Studies

Plain films of the chest, abdomen - supine/erect,
KUB (kidney, ureter, bladder)
Note: Outlines of visceral organs, psoas shadow

Gases - Dilated loops of bowel
Biliary tree
Under the diaphragm

Abnormal calcification - gall stone, faecalith, aorta

ACUTE APPENDICITIS

I. Common Sites of Appendix

Retrocaecal	74%
Pelvic	21%

II. Pathology

1. Non-obstructive - commences in mucous membrane
2. Obstructive - 1/3 cases
   faecalith - commonest agent
   rapid inflammatory process
   perforation at site of impaction

III. Special Features

1. Infant - high perforation rate
   high mortality - poor localisation of peritonitis
   by undeveloped greater omentum
2. Aged - frequent gangrene and perforation
   minimal/vague physical signs
3. Pregnancy - increasing mortality as term near
   - gravid uterus displaces appendix up
   - premature labour - perforated : 50%
   non-perforated : 30%

IV. Management

1. Acute Appendicitis

Early diagnosis and early appendicectomy
Preoperative antibiotic
Incision: grid-iron, Lanz's, Rt. paramedian
Primary closure/delayed primary closure

2. Appendix Mass

Component: greater omentum, caecal wall, loops of small intestine
Ochsner-Sherren regime: conservative treatment but prepared for operation
To stop if: rising pulse rate
spreading abdominal pain
increase in size
Not indicated in: a. uncertain diagnosis
b. extremes of age

3. Appendiceal Abscess

Common sites - around McBurney's point
retrocaecal, subcaecal, retrorectus, post-ileal
Drainage
Interval appendicectomy

INTESTINAL OBSTRUCTION

I. Approach

Recognition
Classification and causes
Preoperative management
Surgery

II. Symptoms and Findings

Anorexia, nausea, vomiting
Constipation - obstruction
Abdominal discomfort, pain
Abdominal distension
Dehydration

III. Strangulation vs Non-strangulation

Nearly always small bowel
Distinction not clearcut -
1/3 of strangulations not suspected

Suggestive findings -
shock, high fever,
severe, constant pain
bloody stools
abdominal rigidity

IV. Aetiology

1. Small Bowel

Adhesions		60%
External hernia		20%
Neoplasm		10%
intrinsic	3%	
extrinsic	7%	
Miscellaneous		10%
intussusception, volvulus, gallstone ileus, F.B.		

2. Large Bowel

Carcinoma	90%
Volvulus	5%
Miscellaneous	5%
diverticulitis, pseudo-obstruction	

V. Preoperative Management

Blood-work : Hb, WBC, RFT,
amylase
X-match

Plain X-ray (serial) - small vs large bowel
complete vs incomplete
likely cause

Ryle's tube, Foley
I.V. drip/CVP line
Close monitoring
Antibiotics
*K^+, urine output

VI. Surgery

1. Small Bowel

Lysis of adhesions
Hernia repair
Bowel resection and anastomosis

Mortality : 5-25%

2. Large Bowel

Left colon - right transverse colostomy
tube caecostomy (L.A.)
loop ileostomy
resection $\pm$ anastomosis

Right colon - resection and anastomosis
caecostomy
bypass

Mortality : 15-30%

CHEST AND ABDOMINAL INJURIES

MANAGEMENT OF CHEST INJURIES

I. Diagnosis of Major Conditions

- ventilatory insufficiency
- circulatory insufficiency

Principle : recognition, resuscitation, repair

II. Recognition

Assess ventilatory exchange
Inspect thoracic movement
Palpate pulse

III. Initial Resuscitation

Cover sucking chest wound
Establish open airway
Release tension pneumothorax
Drain haemothorax
Tap pericardial tamponade

IV. X-ray

Widening of mediastinum
'Wet lung'
Elevated diaphragm
Elevation of stomach bubble

V. Reassessment

Ventilatory and circulatory state

Indications for Early Thoracotomy

1. Massive haemorrhage
2. Rapidly reforming cardiac tamponade
3. Widened mediastinum
4. Ruptured oesophagus
5. Ruptured aorta
6. Ruptured diaphragm + herniation of abdominal content
7. Uncontrolled air leak - ruptured trachea or major bronchi
8. Significant chest wall defect

Thoracic Cage

1. Soft tissue injury
2. Subcutaneous emphysema
   - Through a. disruption in pleura and intercostal muscles
     b. outward dissection of mediastinal emphysema
     c. direct connection with external wound
   - Diagnosis a. crepitus in skin
   - Treatment b. underlying cause
3. Rib fractures
   - Diagnosis a. localised chest pain ± subcutaneous and bone crepitus
     b. X-ray
   - Treatment a. analgesics
     b. intercostal nerve block
4. Flail chest
   - Diagnosis a. paradoxical movement of chest wall
   - Treatment a. stabilise the flail segment
     b. positive pressure ventilation

Pleural Space

1. Haemothorax
   - Diagnosis a. shock and respiratory embarrassment
     b. blood on thoracocentesis
   - Treatment a. restore blood volume
     b. chest tube drainage
     c. thoracotomy for massive haemorrhage
2. Pneumothorax
   - Treatment a. chest tube drainage
3. Tension pneumothorax
   - Diagnosis a. ventilatory and circulatory impairment
     b. tracheal ± mediastinal shift
   - Treatment a. chest tube drainage

Lungs

1. Lung parenchymal trauma
   - Types a. contusion
     b. laceration
     c. haematoma
   - Treatment (depends on severity)
     a. chest tube drainage
     b. suture ligation ± resection
2. Tracheobronchial tree
   - Diagnosis a. mediastinal and neck emphysema
     b. persistent pneumothorax
     c. unilateral atelectasis
   - Treatment a. repair

Oesophagus

Diaphragm

Heart

Aorta and Great Vessels

MANAGEMENT OF ABDOMINAL INJURIES

I. Mode of Injury

1. Penetrating
   - from within (swallowed foreign bodies)
   - from without (stab, bullet)
2. Non-penetrating
   - crushing force
   - shearing force

II. Structures Involved

1. Solid organ - liver, spleen, kidney, pancreas
2. Hollow viscera - bowel, gall bladder
3. Others - mesentery, omentum, diaphragm
4. Abdominal wall

III. Types of Injury

Contusion, haemorrhage, perforation

IV. Diagnosis

1. Mode of injury
2. Circulatory state
3. Location of wounds (entry, exit)
4. Abdominal examination
   area of tenderness, referred pain
   distension by fluid (blood, bile) or gas
   abnormal mass
   peritonitis

V. Indications for Laparotomy

1. Tenderness with rigidity
2. Peritonitis
3. Fluid or air in peritoneal cavity
4. Systemic signs of acute blood loss + trauma

VI. Management

1. Initial resuscitation
   a. airway
   b. circulation
   c. nasogastric tube - diagnostic
      - gastric decompression
2. Biochemical studies
   Blood - haematocrit, haemoglobin
   white cell count
   amylase
   Urine - red blood cells
   sugar or albumin

3. X-ray studies (depend on clinical urgency)
   - Foreign bodies
   - Bone fractures
   - Outline of solid organs
   - Free gas beneath diaphragm
   - Emphysema
   - Position of solid and hollow viscera

4. Special procedures
   - Abdominal tapping
   - Peritoneal lavage

Abdominal Wall

Haematoma - evacuation
ligation of bleeder
Penetrating wound - excision/debridement
surgical toilet

Spleen

Diagnosis - LUQ tenderness Kehr's sign
Hypotension + pallor
Rib fracture
Treatment - splenectomy

Liver

Superficial laceration to massive tissue destruction
Treatment - suture
resection
packing
Other important structures - CBD, PV, HA

Pancreas

Bleeding points - suture + abdominal drainage
Crushed tissue - resection
Main pancreatic duct - internal drainage

Duodenum

Simple laceration - primary closure
Severe laceration - repair + defunctioning

Stomach

Repair/resection + lavage

Small Bowel and Mesentery

Repair/resection + 1^{o} anastomosis

Colon, Rectum, Perineum

Choice of repair
1. 1^{o} closure $\pm$ proximal colostomy
2. Resection $\pm$ 1^{o} anastomosis $\pm$ proximal colostomy
3. Exteriorisation

Factors affecting choice
1. Location of injury
2. Extent of injury
3. Associated injury
4. Amount of peritoneal soiling
5. Length of time since injury