THE UNIVERSITY OF HONG KONG

Department of Surgery

LECTURES FOR MEDICAL STUDENTS

VOLUME 2 August 1994

CONTENTS

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

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

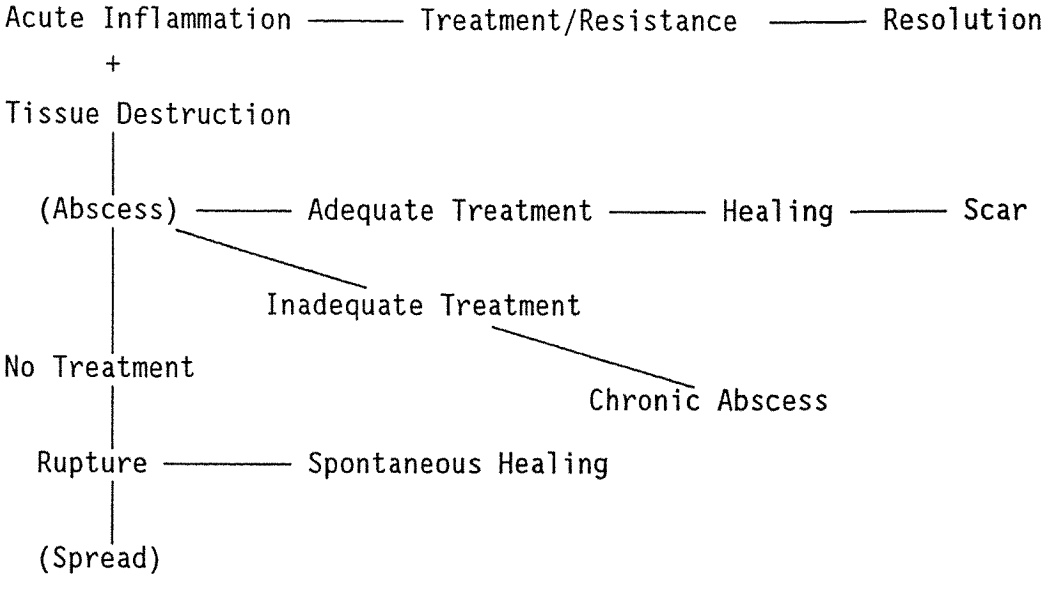
1. Scalp - under aponeurosis
   necrosis of bone
   venous thrombosis - intracranial

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)

INFECTIONS 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 (NOSOCOMIAL 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

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)

a.	Intracellular Fluid Space (ICF)	40%	24 litres
b.	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 in the body is maintained, i.e., there is always interchange of fluid of different compartment.

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_4	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.

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 <u>not</u> 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 Hypenatraemia	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 of fluid and electrolyte disturbance

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

CID-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
      $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2$

2. Body pH = 6.1 + log $\frac{HCO_3}{pCO_2}$ (controlled by kidney) (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 ± 2 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 may be difficult 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
   a. Activities energy expenditure
   b. Stress energy expenditure

2. Metabolic response to starvation
   - mobilization of body fat mainly

3. Metabolic response to trauma
   - mobilization of muscle protein mainly

4. Harmful effect of malnutrition on outcome
   - increased incidence of postoperative complication due to poor poor wound healing, anastomotic healing and chest infection

5. Definition of malnutrition
   a. subjective global assessment
   b. weight loss > 10%

6. Beneficial effect of perioperative nutritional support
   - reduce postoperative complication in severely malnourished patients

7. Means of perioperative nutritional support
   a. Enteral - NG tube
      gastrostomy
      jejunostomy
   b. Parenteral - Broviac or Hickman catheter

8. Components of nutrition
   a. Carbohydrates/glucose
   b. Protein/amino acid
   c. Fat/fatty acid
   d. Fluid and electrolytes
   e. Vitamins and trace elements

9. Basal requirement
   Energy 30 Kcal/Kg/Day
   Nitrogen 200 - 300 mg N/Kg/Day
   Additional requirement is necessary to replete malnutritional state and to meet major stress e.g. sepsis, burn, trauma

10. Monitoring during nutritional therapy
    - body weight
    - body temperature
    - CBP RFT LFT sugar
    - input and output
    - urine for sugar and ketone

11. Complication of nutritional therapy
    Enteral
    - aspiration pneumonia
    - vomiting
    - diarrhoea
    - bloating
    - dehydration
    Parenteral
        Catheter-related
        - wound sepsis
        - catheter sepsis
        - catheter embolism
        Nutrient related
        - electrolyte disturbance
        - dehydration
        - pulmonary edema
        - hyper or hypoglycaemia
        - fatty liver
        - cholestasis

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EVALUATION OF SURGICAL RISKS

I. Patient
II. Time of operation
III. Nature of operation

I. Patient

A) Age

1. Elderly patient
   - Physiological age
   - Associated medical disease
   - Occult cancer
   - Modify fluid & drug administration
   - Slow recovery
2. Paediatric patient
   - Premature & new born infant
   - Heat loss
   - Dosage of drug
   - Blood and fluid replacement
   - Postoperative position

B) Compromised Host

1. Nutrition
   - Weight loss over 20%, increase operative mortality and infection rate
2. Immune competence
   - Cytotoxic drugs, immunosuppressant & steroids increase operative complication
3. Radiotherapy
   - Reduces host resistance to infection & wound healing

C) Medical Conditions

1. Pulmonary function
   - Smoking
   - Tubuculosis, asthma, etc lead to mortality & morbidity
2. Cardiac disease
   - Recent infarction, myocardial ischaemia
   - Hypertension
3. Blood disease
   - Anaemia
   - Clotting problem
4. Endocrine problem
   - Diabetes affects wound healing
   - Thyrotoxic crisis
5. Others
   - Renal, liver disease, etc

D) Drugs

1. Sensitivity to some drugs
   - Penicillin, aspirin, morphine, barbiturates, etc
2. Continuation of drugs
   - Depressants, tranquilizers affects anaesthestic agents

E) Obesity

1. Affects wound healing
2. Associated medical disease
3. Anaesthetic risk

II. Time of operation

A) Emergency procedure
- Increase risks
- Perform minimal procedure

B) Elective operation
- Surgical procedure can be planned

III. Nature of operation

A) Neurosurgical operation
- Conscious level
- Mental state
- Postoperative problems

B) ENT, Head & Neck
- Airway problems

C) Cardiothoracic
- Maintenance of perfusion and ventilation

D) Abdominal procedures
- Preoperative and postoperative problems

E) Urological
- Postoperative monitoring

F) Orthopaedic
- Fixation and mobilization

G) Special situations
- More than one region affected
- Microvascular free tissue transfer
- Reconstruction

CARDIOPULMONARY RESUSCITATION

Cardiac Arrest (Clinical Death)

- sudden, potentially reversible cessation of circulation and respiration.

Mechanism of Cardiac Arrest

1. Pump failure
   - Asystole (95%)
   - Ventricular fibrillation (5%)
   - Extreme bradycardia

2. Circulatory obstruction
   - e.g., obstruction of right ventricular outflow due to massive pulmonary embolism.

Recognition of Cardiac Arrest

1. Unconsciousness
2. Apnoea or gasps
3. Deathlike appearance (cyanosis or pallor)
4. Absence of pulse in large arteries (e.g., carotid or femoral)

Differential Diagnosis

1. Simple fainting
2. Vasovagal reaction
3. Epilepsy
4. Cardiac conduction disturbance
5. Hypovolaemic shock
6. Acute myocardial infarction
7. Pulmonary oedema

Results of Sudden Complete Cessation of Circulation

1.	Unconsciousness	15 sec.
2.	Iso-electric EEG	15-30 sec.
3.	Agonal gasping	30-60 sec.
4.	Apnoea and maximal pupillary dilatation	30-60 sec.
5.	Permanent brain damage	5 min.

Preventive Measures

1. Sound knowledge of drugs, anaesthetic agents, electrolyte balance, etc.

2. Recognition and proper management of patients at risk;

   a. Identification of patients with
      Previous arrest;
      Prior acute myocardial infarction;
      Unstable angina pectoris;
      Documented coronary artery disease involving two to three vessels or recurrent ventricular arrhythmia
   b. Public education
   c. Coronary Ambulance Service
   d. Coronary Care Unit
   e. Intensive Care Unit

Cardiopulmonary Resuscitation

Basic Life Support	Airway control Breathing support Circulatory support
Advance Life Support	Drugs and fluids Electrocardiography Fibrillation treatment

A. BASIC LIFE SUPPORT

Airway Control

Commonest site of airway obstruction is hypopharyngeal - relaxed tongue and neck muscles fail to lift the base of the tongue from the posterior pharyngeal wall, when the patient's head is in the flexed or mid-position.

Triple airway manoeuvre
Backward tilt of the head
(in patients with suspected neck injury, use moderate tilt; maximal backward tilt of the head might aggravate a spinal cord injury)

Forward displacement of the mandible - jaw thrust

Opening of the mouth

Manual clearing the airway of foreign matter e.g., vomitus or blood (dentures - if firmly in place, leave them in position)

Breathing Support

Direct mouth-to-mouth ventilation

Take a deep breath, seal your mouth around the patient's mouth (mouth and nose in infants and small children) with a wide open circle, and blow forcefully into adults, gently into children (use only puffs for infants to avoid lung rupture). When blowing into the mouth, prevent air leakage through the nose, either by pinching it with one hand or by pressing your cheek against the nostrils while blowing. While blowing, watch his chest to see whether it rises with your inflation.

When you see the patient's chest rises, stop inflation; release the seal of your mouth against the patient's mouth, turn your face to the side; and allow the patient to exhale passively.

When his exhalation is finished, give him next deep inflation. Volume is more important than rhythm. Repeat inflations in adults about every 5 sec. (12 per min.); in children about every 3 sec. (20 per min.)

Mouth-to-nose ventilation

Mouth-to-adjunct ventilation

Exhaled air, which contains 16-18% O_2, has been found to be an adequate resuscitative gas, provided that the patient's lungs are normal and the operator uses about twice normal tidal volumes. This usually results in arterial pCO_2 values of 20-30 mmHg and an arterial pO_2 values of over 75 mmHg in the patient with normal lungs.

Circulatory Support

External cardiac compression

1. Position yourself to either side of the patient.

2. Locate the xiphoid-sternal junction.

3. Place the heel of one hand over the pressure point at the lower half of the sternum and place the heel of the other hand on top of the first hand.

4. Push the sternum downward toward the spine about 1 to 2 in. (4 to 5 cm) in adults. The force required varies and should not be more than necessary for sternal displacement.

5. Hold the sternum down for about 1 sec. (50% of the cycle), then release rapidly and wait for another 1 sec. (other 50% of the cycle) to let the chest filled with blood.

6. Reapply pressure every sec. or at a slightly faster rate. The presently recommended rate is 60 per min. for two operators (with ventilation interposed after every fifth compression) and 80 per min. for one operator (alternating 15 compressions with two quick lung inflations). In small children, compress the sternum with one hand only; in infants with the tips of two fingers.

In small infants, the rescuer may encircle the infant's chest with both hands and compress the midsternum with both thumbs.

The heart in infants and small children lies higher in the chest, and the danger of injuring the liver is greater; apply cardiac compressions over the midportion of the sternum. Press down only about in. (1 to 2 cm) in infants, and 1 to 1 in. (2 to 4 cm) in small children. In children and infants, compression rates of 100 to 120 per min. are recommended at present. Since backward tilt of the infant's head lift his back, the back should be supported by one of the rescuer's hands, a folded blanket or other support.

Augmentation of Blood Flow during External CPR

This is possible by restraining the abdomen, by hand or by pressure suit (military anti-shock trousers, MAST). These measures however can damage the liver and other abdominal organs and require high lung inflation pressures which require a tracheal tube. A safer method of augmentation of blood flow is an intravenous fluid load.

Monitoring the Effectiveness of CPR

In the presence of two operators, the ventilating operator should:

a. intermittently palpate the carotid pulse; and

b. check whether a spontaneous pulse has returned, at first after one min. of CPR and every few min. thereafter, during brief interruption of external cardiac compressions.

Blood Flow during Cardiac Compression

Cardiac pump mechanism - until recently, the mechanism by which blood flows during external cardiac massage was attributed to compression of the heart between the sternum and vertebral column. When a pulse was generated during external cardiac massage, it was inferred that compression occurred in a manner analogous to internal cardiac compression, during which the hand directly squeezed the heart to produce forward blood flow.

Thoracic pump mechanism - the new explanation for a blood flow during cardiac massage. Increased intrathoracic pressure generatd during cardiac compression is transmitted equally throughout the thorax but unequally to the vessels in the neck. The resultant arteriovenous pressure gradient in the extrathoracic vessels explains blood flow. The heart, rather than functioning as a pump, merely serves as a conduit through which blood circulates.

Sternal compressions can produce systolic blood pressure peaks of 100 mmHg and more, but the diastolic pressure is usually not more than 10 mmHg and the systolic central venous pressure (and intracranial pressure) is increased almost as much as arterial pressure, leaving only a minimal perfusion pressure. (This is not the case in open chest cardiac compressions, during which venous pressure is not significantly increased). External cardiac compressions result in a cardiac output and carotid artery blood flow of usually less than 30% of normal flow, sometimes less than 10%. This would not be enough to maintain or restore consciousness and can be borderline for maintaining viability of cerebral neurons during prolonged CPR.

ADVANCED LIFE SUPPORT (Restoration of Spontaneous Circulation)

Spontaneous circulation should be restored as promptly as possible after initiation of basic life support, since external cardiac compressions produce only borderline blood flow, which may be inadequate to keep the brain and heart viable for longer than a few minutes of CPR. Restoration of spontaneous circulation usually requires:

Administration of drugs and fluids
Electrocardiographic diagnosis
Fibrillation treatment

in varying sequences depending on circumstances.

Witnessed, ECG monitored arrest - ventricular tachycardia or fibrillation:

1. If a defibrillator is immediately available, administer external electric countershock within 30 sec. of the patient's collapse. Do not delay countershock for administration of drugs or basic life support.

2. If the first countershock fails to restore a spontaneous pulse immediately, start closed-chest CPR and repeat countershocks every 1-2 minutes.

3. Give adrenaline 0.5-1.0 mg IV (adult dose), followed by sodium bicarbonate 1 mEq/Kg IV as soon as possible after the initiation of basic life support. If countershock failed, circulate the drugs by cardiac compressions for at least one minute before repeating countershock. Do not use bicarbonate if there has been prompt initiation of CPR and minimal tissue acidosis, as it may lead to alkalaemia with intractable ventricular fibrillation.

4. If countershock fails to convert the rhythm or if a spontaneous pulse is achieved but then reverts rapidly to ventricular fibrillation or ventricular tachycardia, give lignocaine 100-200 mg IV, followed by an infusion of 1-3 mg per min. (adult dose). Then repeat countershock.

Witnessed arrest - asystole or electromechanical dissociation

Unwitnessed arrest:

1. Start basic life support as soon as possible.

2. Give adrenaline in 0.5-1.0 mg IV (adult dose). (Dilution is not necessary.) Repeat this dose, or even a larger dose (1-2 mg) every 2-5 mins. If there is no intravenous route available, give the adrenaline via needle puncture of a peripheral vein, or via the endotracheal route.

3. When cardiac arrest has lasted 2 minutes or longer, or tissue hypoxia has existed prior to arrest, give sodium bicarbonate, 1 mEq/Kg IV, slowly into a running infusion. In these circumstances, sodium bicarbonate combats the acidaemia that would otherwise offset adrenaline's action.

4. One half of the above dose of bicarbonate may be repeated blindly but not more than every 5-10 mins. of CPR, least alkalaemia and hyperosmolality develop. Once arterial pH values are available, bicarbonate administration should be guided by such measurements and accompanied by moderate hyperventilation.

ROUTES FOR DRUGS AND FLUIDS

1. Peripheral intravenous route.

2. Intrapulmonary route - Intratracheal instillation of selected drugs is recommended in situations where an intravenous route is not readily available. Adrenaline, lignocaine, atropine, and other drugs that do not cause tissue damage, can safely be given via the endotracheal tube, using 1-2 times the intravenous dose, diluted in 10 ml of sterile water. Bicarbonate, however, must <u>not</u> be given.

3. Intracardiac route - the blind intracardiac injection of drugs is not recommended during closed-chest CPR, as it may produce pneumothorax, injury to a coronary artery and prolonged interruption of external cardiac compressions. Inadvertent injection into cardiac muscle rather than a cardiac chamber may, in addition, lead to intractable dysrhythmias. Intracardiac injection of adrenaline should be considered only in rare instance that a vein is inaccessible, and the endotracheal route has not been established, and should be done via a long, thin (e.g. 22 gauge) needle through the fifth intercostal space parasternally into a heart chamber. The paraxiphoid approach (needle insertion to the left of the xiphoid process, and advancement cephalad, posteriorly, and laterally) is less likely to damage the anterior descending coronary artery. The position of the needle must be confirmed by free aspiration of blood.

4. Central venous route.

Useful Drugs in CPR

1. adrenaline
2. Sodium bicarbonate
3. Vasopressors
    a. Noradrenaline
    b. Metaraminol
4. Cardiotonics
    a. Isoproterenol (Isuprel)
    b. Dopamine
5. Calcium chloride
6. Lignocaine; procainamide; bretylium
7. Propranolol (Inderal)
8. Atropine
9. Nitroprusside or nitroglycerin for infusion
    - Nitroglycerin tablets
10. Morphine or pethidine
11. Furosemide (Lasix)
12. Methylprednisolone (Solu-Medrol), or dexamethasone (Decadron)
13. 50% dextrose (for empirical use in coma of unknown aetiology)
14. Bronchodilators
    a. Aminophylline
    b. Terbutaline
15. Diphenhydramine (Benadryl), an antihistaminic
16. Naloxone (Narcan, a narcotic antagonist)
17. Barbiturate, short-acting (pentobarbital), or ultra-short-acting (thiopental)
18. Diazepam (valium); and diphenylhydantoin (phenytoin)
19. Chlorpromazine (Largactil) as vasodilator, and for psychiatric emergencies
20. Muscle relaxant; succinylcholine (Scoline) and pancuronium (Pavulon)
21. Mannitol
22. IV fluids

Technique of External Electric Countershock

1. Basic life support ongoing
2. Turn synchronised switch of defibrillator off
   Turn main power switch on
3. Set energy level to desired reading (approximately 3 Joules/Kg)
4. Charge the paddles
5. Lubricate the paddles with electrode paste. Interrupt the rescuer's chest compressions as briefly as possible (15-20 sec. maximum for countershock. Place paddles on chest. Negative paddle - just to the right of the upper sternum, below the right clavicle. Positive paddle - just below and to the left of the left nipple.
6. Apply firm pressure with the paddles against the chest.
7. Confirm ECG diagnosis.
8. Clear the area.
9. Fire the defibrillator.
10. Leave paddles in place 5 sec. to ascertain rhythm.
11. If a pulse is not palpable within 5 sec., resume basic life support
12. If VF continues after 1 min. CPR, repeat countershocks with 3, 4, 5 joules/kg.

Complications of External CPR

1. Fractured ribs/sternum.
2. Laceration of liver.
3. Ruptured heart.
4. Tension pneumothorax.
5. Embolisation of marrow to pulmonary circulation.

Failure of External CPR

1. Cardiac tamponade.
2. Tension pneumothorax.
3. Ruptured aorta or heart.
4. Abnormal thoracic cage -
   - crushed chest
   - severe kyphoscoliosis
   - pectus excavatum
   - severe emphysema with fixation of rib cage

Indications of Open-chest CPR (for Trained Physicians Only)

1. When intrathoracic pathology is suspected, e.g. cardiac tamponade/uncontrollable haemorrhage following penetrating wounds of the chest, crushing chest injury or cardiothoracic surgery.

2. When External CPR fails to produce a palpable femoral or carotid pulse as occasionally is the case in patients with chest or spine deformities or severe emphysema with barrel-chest.

3. As the last step in treating intractable ventricular fibrillation or electromechanical dissociation, when prolonged closed-chest CPR and repeated external defibrillation attempts have failed; this may be the case in suspected massive pulmonary thromboembolism (when the open technique permits breaking-up or removing the embolus or in deep hypothermia (when the open technique permits direct rewarming of the heart for defibrillation).

4. For cardiac arrest in the operating room in a patient whose chest is already open.

Technique of Open-chest CPR (Intubated Patients Only)

1. Cut through skin and muscles directly overlying the 4th or 5th left intercostal space. Pierce the intercostal structures bluntly with a handle or bandage scissors and tear open the intercostal space with your fingers. Insert a rib spreader if available.

2. Immediately compress the heart, without at first opening the pericardium by placing the fingers of the right hand behind the heart and the thenar and thumb in front of the heart. Take care not to pierce the atrium or ventricle with your thumb. If the heart is large, use one hand behind and one hand in front of the heart to compress it.

3. Usually one can diagnose VF, inject drugs and defibrillate through a closed pericardium (one can see and feel the wormlike motions of VF). Whenever you are not certain, however, and thus choose to open the pericardium, take care not to interrupt compressions or injure the heart or vagus nerve. In intractable VF or when the first dose of adrenaline has failed to restart cardiac action, open the pericardium to allow direct inspection of the heart and to prevent injury to coronary vessels from multiple needle punctures.

4. Drug therapy

   a. When drugs are necessary, they should be injected into the cavity of the left ventricle, not into the myocardium.
   b. Start with adrenaline 0.5 mg/70 kg.
   c. Atropine and lignocaine may also be given safely via the intracardiac route.
   d. Do not give bicarbonate intracardiac - use the intravenous route.

5. Defibrillation

   a. Use two insulated paddle electrodes.
   b. Place one electrode behind the LV, the other over the anterior surface of the heart.
   c. DC countershock is preferred.
   d. Start with 0.5 watt-seconds (joules)/kg body weight. If the shock is ineffective at this low energy level, increase the energy level gradually with subsequent shocks. (High energy shocks applied directly to the heart are more likely than external countershock to produce heart damage, including myocardial burns.)

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 metabolism

      Catecholamines initiate lipolysis

      Tissue lipids ⟶ Free fatty acids → ↑ serum FFA
      Serum triglycerides (FFA)

   c. Carbohydrate metabolism

      - Catecholamines increase liver glycogenolysis → ↑ blood glucose

      - Aerobic metabolism $\xrightarrow{\downarrow \text{ perfusion}}$ Anaerobic metabolism

        i.e. Glucose pyruvate $\xrightarrow{\downarrow \text{ perfusion}}$ Lactate → ↑ blood lactate
        → ATP (small quantities)

2. Cell

   Breakdown of the sodium pump

3. Circulation

   Functional shunting
   Protein extravasation

	Pulmonary Arterial	Systemic Vascular	Cardiac Output	Oxygenn 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 treatment.

   a. Crystalloids : e.g. Hartmann solution and normal Saline
   b. Colloids : e.g. blood, plasma, serum albumin, plasma substitutes such as Haemaccel, 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

HEAD INJURIES

Head injury may be sustained in a variety of ways such as by blunt trauma e.g., by traffic related accidents, falls, missiles, and blows. Head injuries may be very trivial, and may not require hospitalisation, or may be so severe as to require surgical intervention or care in an Intensive Care Unit.

Classification

Depending upon the nature and extent of the injury, head injuries (H.I.) may be classified as:

I. Minor injuries
II. Major or serious head injuries
III. Associated injuries
IV. Sequelae

I. MINOR HEAD INJURIES

These patients are usually fully conscious or have had no loss of consciousness. These injuries are:-

1. Scalp Injuries

Before going into scalp injuries it is worthwhile to remember the various layers of the scalp which are: skin, subcutaneous tissue, galea aponeurotica, loose connective tissue and the pericranium. Remember injuries of the scalp that do not involve the galea aponeurotica can be treated by simple steri-tape - of course depending upon the size of the laceration. The arteries of the scalp are either superficial or deep to the galea and therefore during closure of the skin by surgical suture it is important to define whether the galea is cut or not. If it is then it must be closed by a subcutaneous suture because not only it will approximate the scalp edges together but it will also help to stop arterial bleeding.

Scalp injuries may be sustained by road accidents, incised wounds by knife, chopper, glass, sharp edge etc., or these may be burns due to thermal, chemical, electrical or radiation energy. Scalp lacerations may be (depending upon their nature):

- linear
- irregular
- stellate
- incised

Scalp injuries do not all require an X-ray of the skull but those who have sustained injury due to R.T.A. or assaults and glass should have it. Treatment of scalp injuries is perfect haemostasis, thorough cleansing to remove all the hair and debris, earth, etc. from under the edges of the scalp, debridement if necessary and as appropriate and good skin closure. Usually there is no need to give antibiotics. If the wound is really dirty, then these may be given.

2. Scalp Haematomas

These are quite frequently seen and commonly in children. In a baby it must be remembered that if there is a scalp haematoma, Hb must be checked and you would find that it has dropped to around 11G%. There are two kinds of scalp haematomas:

(a) Subgaleal Haematoma : These are usually very large and a child may lose as much as 150 ml of blood in it. These are usually soft to tense on palpation. The characteristic feature is that if it is on one side of the scalp you can move it across the midline and across the suture lines i.e., from one bone to another. These must not be aspirated because these tend to recur - only to make the child a little more anaemic, and may get infected. A crepe bandage may be given. Slowly they resolve completely.

(b) Subperiosteal Haematoma : This usually feels very tense and is small. In contrast to (a) above you cannot move it from one place to the other and this is, of course, due to the periosteum being attached to the suture lines. Treatment like (a) above.

3. Head Injuries without Loss of Consciousness

These patients do not need hospitalisation; however, if in doubt get X-ray skull performed. If there is no fracture of the skull and there is no history of epilepsy, real subconjunctival haemorrhage, C.S.F. leak from the ear or nose, bleeding from the ear, Battle's sign or Racoon eye syndrome, etc. you would be perfectly justified not to admit him.

II. SERIOUS HEAD INJURIES

Classification

Cerebral contusion
intracranial haematomas
- EDH
- SDH
- ICH

Fractured skull: Simple / Compound
- Linear
- Comminuted
- Depressed
- Indented

Mechanism of Cerebral Trauma

Major cerebral trauma is usually due to R.T.A., falls, blunt or sharp blows, falling objects, etc.

Various Factors involved in H.I.

1. Biochemical Factors. The scalp is usually about a centimeter thick and it has some compressibility and tensile strength. Therefore it serves to protect the brain and the underlying structures. The main factor in preventing injury to the brain is, of course, the skull. From the protection point of view the thicker the skull the better it is - it has nothing to do with 'the thick skulled' quotation. The dura offers little protection to the brain from trauma but it is extremely important - for if the dura is torn open it must be closed, otherwise the C.S.F. may leak.

2. Dynamic Factors. You may remember from your knowledge of physics, that there are usually three kinds of forces i.e., acceleration, deceleration and deformations and these forces can produce four different kinds of stresses to the brain i.e., compressive, decompressive, shearing and tensile. Without going into much detail I think you can imagine what would happen to the brain if it is compressed and then suddenly decompressed. Shearing stress is the worst of all because it tears off the grey matter from the white matter.

3. Vascular Factors. Due to injury the blood vessels may rupture producing intracranial haematomas or petechial haemorrhages in the vital parts of the brain, or the blood vessels may go into spasm producing ischaemia of the brain.

The combined effect of all the above factors is to produce loss of consciousness. The consciousness or the state of awareness is maintained by the reticular formation which extends from the medulla to the basal ganglia. All the above factors initiated by trauma may produce:

Cerebral Contusion

Common sites:
- frontal lobes
- temporal lobes
- brain near the - sphenoid ridge
  - orbital roof
- anterior half of the brain

Contre-coup injury is usually diagonally opposite to the site of injury. This injury may even be worse than the original injury. The worst damage is done by the sphenoid ridge which is a sharp edge and it may shear the Sylvian vein and the brain. Besides these the other sites for contre-coup injury are the frontal lobes, tips of the temporal lobes, medial parts of the cerebral hemispheres and the occipital lobes.

Pathologically, the cerebral contusion is just like any other laceration. Therefore there is some blood, damaged brain, coagulated blood vessels and necrotic brain. There is usually progressive oedema of the brain, and as the skull is a rigid box therefore the patient's condition starts to deteriorate and develop neurological signs. Now there are only two options open: either you investigate the patient and get a CT scan or angiogram done if facilities exist. If not then that is where clinical judgment helps. As in these patients there may be some lateralising signs, you perform an exploratory burr-hole. Remember this is only investigation and is not a substitute for craniotomy or craniectomy except in the case of chronic S.D.H.

If there are no localising signs or if the B.H. are negative then you give them a conservative treatment, the bases for which include:

Respiration. This is the single most important factor in deterioration of the level of consciousness in H.I. patients. Always make sure there is adequate ventilation; if in doubt intubate the patient or get on with tracheostomy. Even a few minutes' hypoxia in H.I. can produce permanent brain damage. As you know, hypoxia increases CO_2 retention which increases cerebral vasculature permeability and thus more cerebral oedema. The condition of the patient may deteriorate suddenly. Thus most unconscious patients are best treated in the Intensive Care Unit.

Circulation. If the patient is bleeding profusely the bleeding should be stopped by pressure bandage, or if it is arterial bleeding by ligature. If the patient is in shock, resuscitate the patient. Some patients with H.I. have high blood pressure; this is only in response to H.I. and they do not need anti-hypertensive agents. In fact, sudden lowering of BP in these patients will produce cerebral ischaemia and brain damage.

Cerebral Oedema. Traumatic cerebral oedema is a real problem to treat. In contrast to oedema due to cerebral tumours it does not respond very well to steroids and in later stages even to hyperosmolar agents. The commonly used measures are :-

- dexamethasone 10 mg I.V. and 4 mg Q6H
- mannitol 20%, 200 mls given I.V. in 20 minutes
- glycerol orally/Ryles tube, one ounce TID
- hyperventilation to lower the CO_2 retention
- prop up the head by 30^{o}
- keep jugular venous drainage free of compression
- never set up a jugular C.V.P. line

Treatment of Concomitant Injuries. As these patients may have fractures of other bones, ribs, spine, etc. these may be treated accordingly.

Nursing Care. During this period of unconsciousness the nurses help tremendously. Since the head is propped up, secretions, saliva, blood or vomitus may gravitate into the lungs. Therefore the patient needs frequent observation, suction, turning the patient, cleansing, care of the eyes, nose, mouths, etc.

Vomiting. Deeply comatose patients rarely vomit but others, who are more salvageable, often do. Don't give antiemetics because they interfere with observations.

Hydration and Feeding. Usually I.V. fluids are given at a rate of 2L/24 hours. Feeding is a problem only of the chronically comatosed patient. In an unconscious patient always keep a watch on serum electrolytes and urea every other day.

Following conservative treatment there are only three possibilities. Either the patient deteriorates and dies, develops localising signs, or recovers. If he develops localising signs then you investigate him to find if he has a clot or perform an exploratory burr hole.

III. INTRACRANIAL HAEMATOMAS

1. Extradural Haematoma (E.D.H.) :

Originates from
- middle meningeal vessels
- dural venous sinuses
- diploic vessels

It is usually seen in young male adults. Usually it is unilateral. The classic picture of L.O.C., lucid interval, focal signs and progressive unconsciousness is seen in only 15% of patients. Usually there is bradycardia, temporal bogginess, and X-ray skull reveals a fracture in 90% of patients. Diagnosis is made by clinical history, CT scan or exploratory burn hole. Treatment is by evacuation of the clot. Prognosis is excellent and mortality is around 5%.

2. Acute Subdural Haematoma (S.D.H.). If it collects within 24 hours it is called acute; 24 hours to 10 days subacute, and > 10 days chronic. It is the commonest of the intracranial haematomas, and carries a high mortality of 50 to 80%. Fractured skull is seen in only 50% of the cases and nearly 20% are bilateral. The commonest sites are the frontal and temporal regions.

3. Intracerebral Haematomas. These haematomas are within the brain, and therefore depending upon their site they produce localising signs e.g., hemiplegia, etc. Diagnosis is usually made clinically, by CT scan or angiography. On B.H. these may be missed. Treatment is surgical evacuation. The mortality is around 35%.

Skull Fracture

Skull fracture is the result of a concentrated force. Since the force is at one point, the L.O.C. is not as frequent as in acute S.D.H. Linear skull fractures are the commonest and seen in 80% of the cases, and in 50% these are in the mid-portion of the skull. Depressed fractures may tear the dura and damage the brain. Diagnosis can be made clinically. X-ray skull is diagnostic, CT scan is not required. Closed (simple) depressed fractures are elevated. Compound depressed fracture is excised and bone discarded. Linear fractures need only observations. Depressed fractures are associated with open dura in 50% cases. No L.O.C. in 50%, and 50% of these fractures are in frontal region. Mortality is low. However, if these are associated with meningitis or brain abscess and coma lasting over 24 hours then mortality is 35%. Treatment is by debridement, thorough cleansing, removal of bone fragments, hair, etc. Always repair the dura and stop the bleeding.

Post traumatic syndrome is frequently seen and comprises:

Headache	80%
Dizziness	50%
Nervous instability	20%

Epilepsy is common after H.I. and may start any time, usually within the first few months. Incidence is:

Minor H.I.	5%
Penetrating H.I.	50%
Laceration of brain)	
Prolonged coma)	90%
Infection (meningitis))	

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 $\pm$ 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

TRAUMATIC INJURIES TO THE GENITO-URINARY TRACT

About 8-10% of all injuries seen in the emergency department involve the genito-urinary system to some extent. Many of these are subtle and difficult to define and require systematic diagnostic expertise. Early diagnosis and appropriate management is essential to prevent complications.

There are two general principles:

1. Uncommon for more than one part or more than one side to be injuried at the same time
2. Very common to be associated with damage to other organs which tend to dominate the clinical picture

I. INJURY TO KIDNEY

These are the most common injuries of the urinary system. A pathological kidney is more readily ruptured from mild trauma.

Aetiology

1. Penetrating (10%) - gun shot
   stabbed by knife
   iatrogenic - percutaneous renal biopsy

2. Blunt (90%) - traffic accident
   contact sport
   falling from height

Classification

1. Renal contusion - haematoma with an intact renal capsule and collecting system

2. Minor lacerations - superficial cortical disruptions that do not involve the deep renal medulla or the collecting system

3. Major lacerations - disruptions extending through the cortex and into the deep medulla, which may involve the collecting system

4. Vascular injuries - occlusions or tears of the renal artery or vein

Clinical Features

1. History of injury
2. Haematuria (95%) either microscopic or gross. The degree of injury does not necessarily correspond to the degree of haematuria
3. Flank bruises, visible mass and tenderness
4. Abdominal distension, ileus and vomiting
5. Features of fracture of lower rib cage
6. Hypovolaemia and/or shock

Investigations

1. IVU - establish the presence or absence of both kidneys, clearly define the renal parenchyma, and outline the collecting system and ureters

2. CT scan - noninvasive
   clear delineation of parenchymal lacerations
   sensitive detection of urinary extravasation
   outline of nonviable tissue
   detection of associated injuries

3. Arteriography - defines parenchymal lacerations and vascular injuries
   non-visualization of a kidney on IVU
   a. avulsion of renal artery and vein
   b. renal artery thrombosis
   c. major vascular spasm
   d. absence of the kidney

Treatment

Governed by

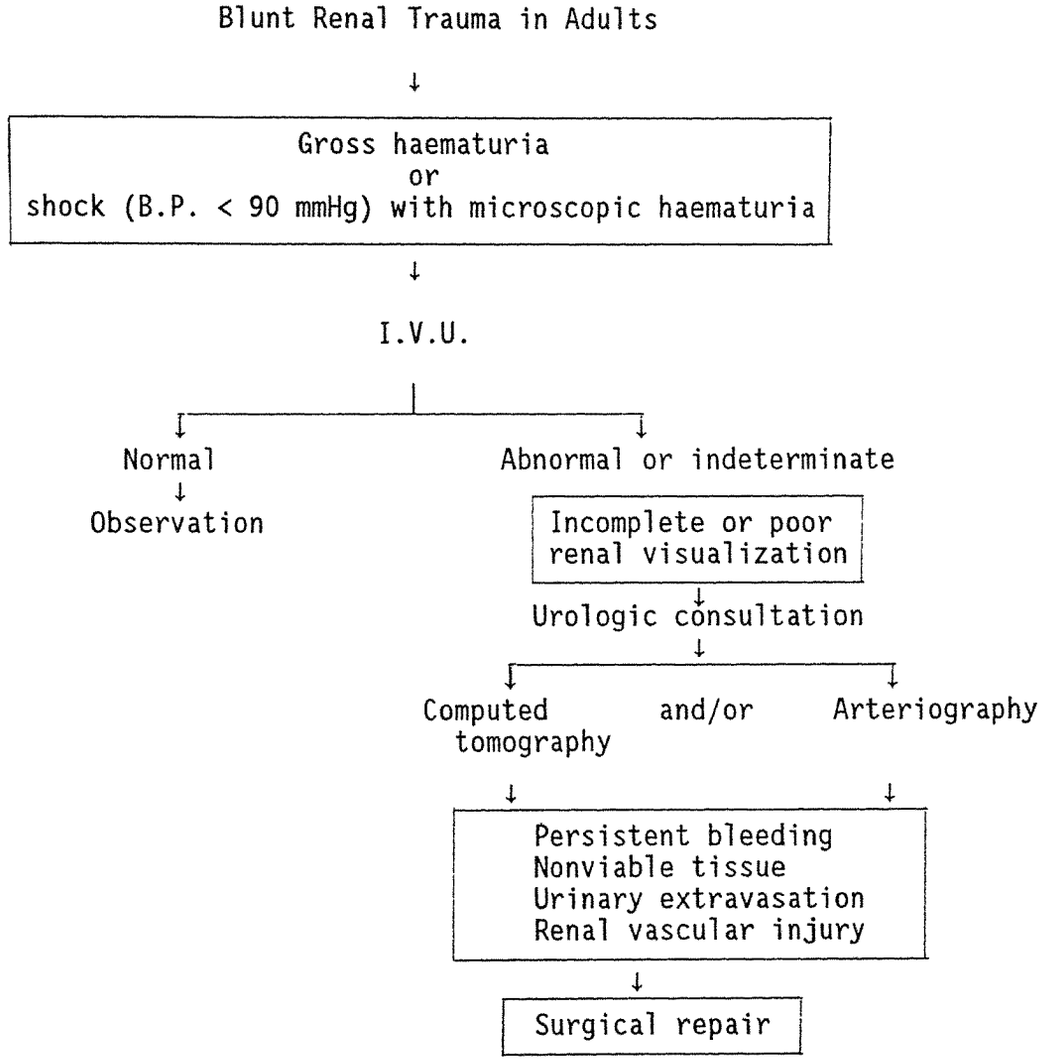
1. Accurate diagnosis of extent of injury
   a. contusions
      85% - 90% of blunt renal injuries
   b. vascular injuries
      operative intervention and reconstruction when recognized early
   c. minor and major laceration
      highly variable

2. Clinical parameters and associated major injuries

Indications for operation

1. expanding or uncontained haematoma
2. extensive urinary extravasation
3. nonviable renal parenchyma
4. vascular injury
5. a penetrating renal injuries

Management Flow Chart



Complications

1. Early (< 6 weeks)

   a. Persistent or recurrent bleeding
   b. Urinary extravasation (urinoma) - percutoneous drainage
   c. Abscess - open drainage
   d. fistula formation to bowel or skin

2. Late

   a. Hypertension - renin-mediated
   b. Hydronephrosis - IVU within 3 months of major renal injury
   c. Traumatic A-V fistula - mainly by stab wounds

II. INJURIES TO URETER

External trauma seldom injures the ureter because of its position and size.

Aetiology

1. Iatrogenic - operative trauma

   a. pelvic surgery - hysterectomy/rectal operations
   b. vascular surgery - aneurysmectomy
   c. endoscopic manipulation - ureteroscopy
   d. spinal operations

2. Penetrating injury from accidents

Ureter is Vulnerable

1. It courses the pelvic cavity and is always near to the vessels
2. It is mobile and can be displaced to abnormal location
3. Ureteric blood supply is delicate
4. The ureter is adherent to back of overlying peritoneum
5. Significant abnormalities occurred 3 - 5%

Clinical Features

1. Injury suspected or recognised during surgery

2. Delayed manifestations :

   a. anuria or deterioration in renal function
   b. urinary fistula to incision or vagina (75%) - after 1-2 weeks
   c. peritonitis
   d. loin pain
   e. asymptomatic

Investigations

1. Urinalysis
2. Renal function test
3. IVU $\pm$ cystogram
4. Retrograde or antegrade ureterogram

Treatment

1. Prevention - ureteric catheterisation in selected case
   care during surgery
   IVU prior to all major pelvic surgery

2. Surgical treatment
   a. timing
      - < 48 hours post-operative - immediate surgery
      > 48 hours - delayed after complete investigations
   b. surgical options
      i. direct end-to-end anastomosis
      ii. reimplantation of ureter
      iii. Psoas hitch technique
      iv. Boari bladder flap
      v. transuretero-ureterostomy
      vi. renal vascular relocation (autotransplantation)
      vii. ileal replacement of ureter

III. INJURIES TO BLADDER

Two important physiological factors in bladder injuries.

1. Degree of bladder distension - increasingly vulnerable as it fills
   a. empty bladder deep in pelvis behind pubic bone
   b. empty bladder has thick wall and minimal intraluminal pressure
   c. more distension - less force required for perforation
   d. infantile bladder more abdominal in position

2. Status of lower muscle
   a. co-ordinate contraction offers protection from impact injury
   b. multiple pregnancies result in lax recti
   c. intoxicated individuals have less co-ordinated contraction

Aetiology

1. Blunt Trauma
   a. direct blow to abdomen - intraperitoneal
   b. pelvic fracture - incidence 10% (20% in pubic arch)
      80% extraperitoneal - antero-lateral wall

2. Penetrating injuries
   a. iatrogenic - pelvic surgery
      transurethral surgery
   b. external violence

Clinical Features

1. History of injury
2. Haematuria
3. Lower abdominal pain
4. Abdominal distension ileus
5. Associated injury - musculo-skeletal

Investigations

1. Ascending urethrocystogram, if urethral injury is suspected
2. Cystogram - static cystogram with full vesical distension and a film taken after drainage

Classification

1. Blunt trauma
   a. contusion - wall continuity intact
   b. interstitial rupture - incomplete tear of bladder wall
   c. intraperitoneal rupture - dome (weakest part of bladder), contrast material outline loops of bowel
   d. extraperitoneal rupture - almost exclusively seen with pelvin fractures, flame shaped areas of extravasation

2. Penetrating trauma

Treatment

1. Penetrating injuries
   a. external violence - exploration
   b. iatrogenic - individualise

2. Bladder contusion
   urethral catheter drainage
   associated sacral injury - intermittent catheterization

3. Interstitial ruptures
   catheter drainage for 10 days

4. Intraperitoneal ruptures
   formal surgical repair

5. Extraperitoneal ruptures
   uninfected urine and isolated injury - catheter drainage
   otherwise surgical repair

IV. INJURY TO URETHRA

Urethral injuries are uncommon and occur most often in men; they are rare in women and children.

Various parts of the urethra may be injured to various extent. According to mechanism of trauma, the urethra can be divided into two anatomical division :

1. Posterior urethra - prostatic
   membranous
2. Anterior urethra - bulbous
   penile

Aetiology

Posterior urethral injuries

1. Fracture of the pelvis
   motor vehicle accidents
   fall, crush
   sporting accidents
2. Penetrating injuries
   external violence : gunshot or stab
   urethral instrumentation

Anterior urethral injuries

1. Straddle injury
   fall : fence, ladder
   kick
   bicycle
2. Penetrating injuries
   external violence
   urethral instrumentation

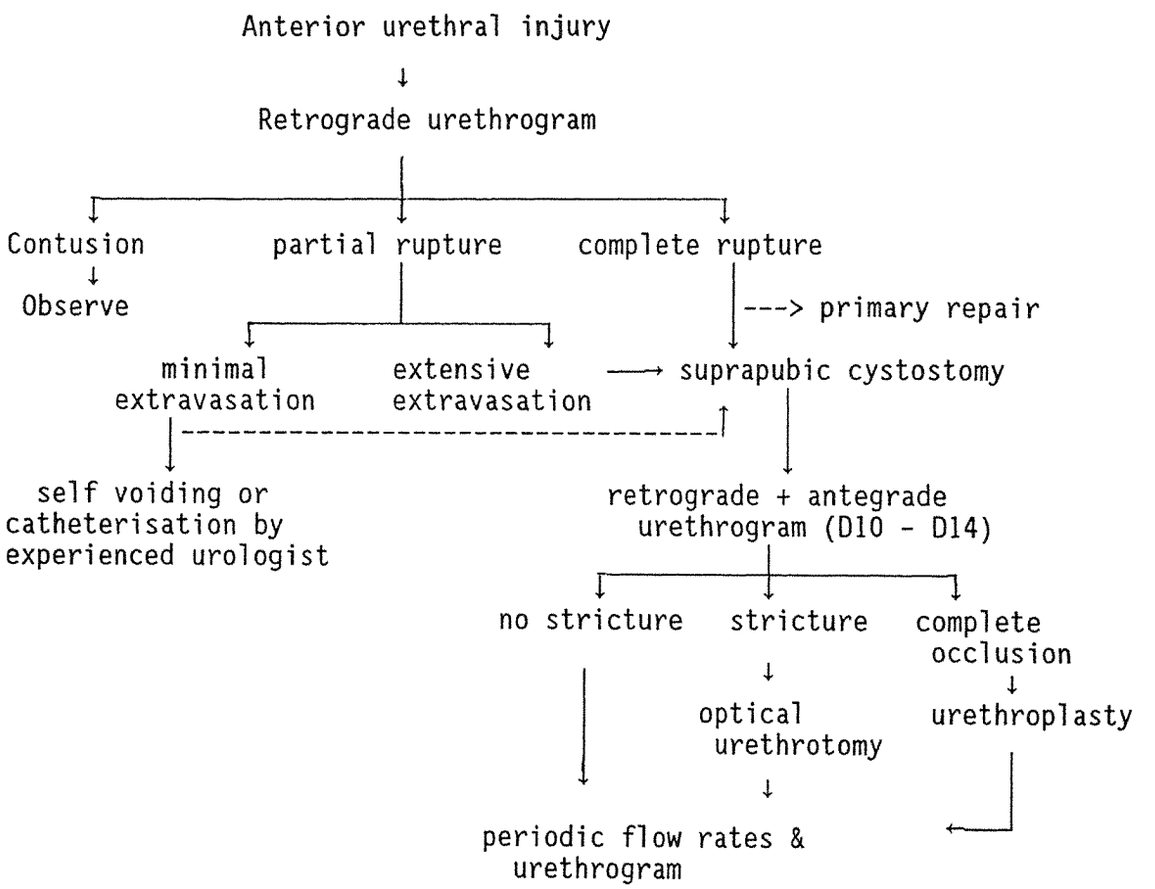
Clinical Features

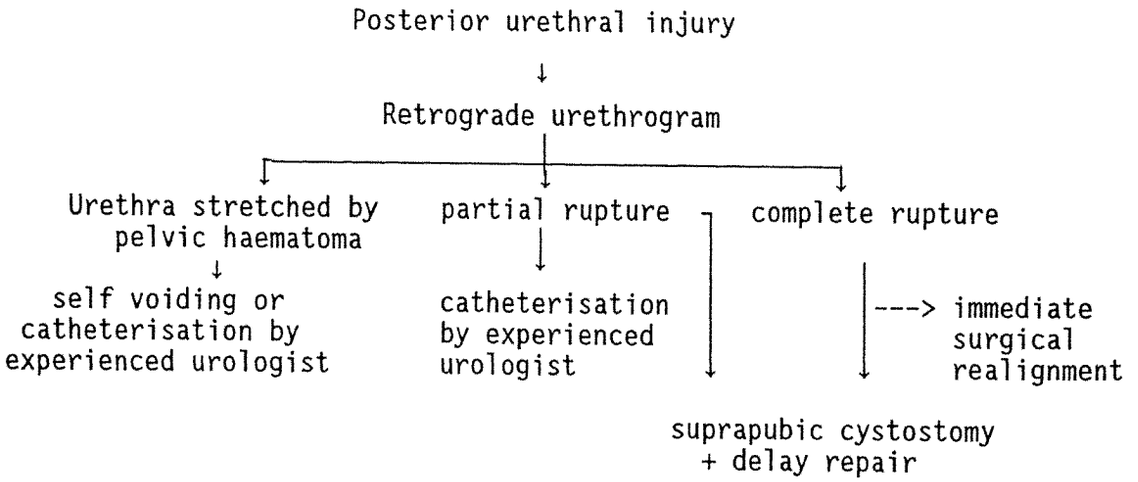
1. History of perineal trauma or fracture of bony pelvis
2. Bloody urethral discharge (spontaneous or after milking of urethra)
3. Inability to pass urine (90%)
4. Distended bladder
5. Floating prostate
6. Perineal and scrotal bruising and haematoma
7. Associated injury e.g., ano-rectal canal

Investigations

1. KUB - fractured pelvis
2. Urethrogram (ascending) - water soluble contrast

Management Flow Chart





Complications

1. Stricture - 20-50%
2. Impotence - 25% if complete tear of posterior urethra
3. Incontinence - 10-15% depending on site and severity

V. INJURY TO PENIS

Disruption of tunica albuginea of the penis can occur during overactive sexual intercourse. At presentation the patient has pain and haematoma and possibly bloody urethral discharge. Surgical repair may be required if severe injury is present.

VI. INJURY TO TESTIS AND SCROTUM

Blunt injury to testis causes severe pain and referred lower abdominal tenderness. Haematoma may be formed. If rupture has occurred primary repair should be done. Ultrasonography would be a useful pre-operative diagnostic technique.

The common scrotal injury is laceration. Primary debridement and repair should be performed.

ACUTE ABDOMEN, INCLUDING APPENDICITIS

ACUTE ABDOMEN

I. Definition

Any condition requiring rapid decision making and usually requiring operative intervention.

- Peritonitis - localised/diffuse
- Intestinal obstruction with evidence of strangulation
- Trauma - blunt/penetrating

II. Common Lesions Requiring Emergency Operation

Trauma
Acute appendicitis
Intestinal obstruction with strangulation
Perforated viscus, including perforated peptic ulcer
Acute cholecystitis
Recurrent pyogenic cholangitis

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
4. Medical
   - M.I. Bronchopneumonia. Diabetes.

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 disease, 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. Localisation of maximal tender site e.g., McBurney's point
5. Costovertebral tenderness
6. Guarding, rebound tenderness, rigidity
7. Rectal and pelvic examination
8. Bowel sounds - increased or absent in ilevs

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. - Invasive investigation but maybe of 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

Gas - Dilated loops of bowel + fluid levels
Biliary tree
Under the diaphragm

Abnormal calcification - gallstone, faecolith, aorta

VIII. Ultrasound Examination

Very useful in detection of gallstones and acute cholecystitis and also intra-abdominal and intra-hepatic abscesses and some of the complications of pancreatitis.

IX. CT scan sometimes indicated.

ACUTE APPENDICITIS

I. Common Sites of Appendix

Retrocaecal	74%
Pelvic	21%

II. Pathology

1. Non-obstructive - commences in mucous membrane
2. Obstructive - faecolith - commonest agent
   rapid inflammatory process
   perforation at site of impaction or gangrene

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 important
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 regimen	: 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 sometimes indicated
Interval appendicectomy

INTESTINAL OBSTRUCTION

I. Approach

Recognition
Classification and causes
Preoperative management
Surgery

II. Symptoms and Findings

Nausea, vomiting
Constipation - may become absolute
Abdominal discomfort, colicky pain
Abdominal distension
Dehydration

III. Strangulation vs Non-strangulation

Nearly always small bowel
Distinction not always easy -
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 - on table lavage to empty colon
followed by resection and primary anastomosis
If unsuitable for this may require Hartmann's procedure

Right colon - resection and anastomosis
caecostomy
bypass

Mortality : 15-30%

GASTROINTESTINAL BLEEDING

I. DEFINITION

1. Haematemesis : vomit gross blood; due to lesion proximal to ligament of Treitz
2. Melena : tarry stool; due to lesion in upper GI tract, small bowel or right colon
3. Haematochezia : passage of bright red blood through rectum; due to colonic lesions

II. 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

III. 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

IV. INITIAL MANAGEMENT

1. Estimate amount of blood loss
2. Complete blood picture, RFT, LFT, PT, APTT
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 2 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 catheter and monitor urine output
10. Monitor intake and output

V. 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

VI. 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 oversewing of bleeding vessel
Gastric ulcer	Partial gastrectomy, gastroduodenostomy
Gastritis	Truncal vagotomy, pyloroplasty
Gastric carcinoma	Subtotal gastrectomy, gastrojejunostomy
Mallory-Weiss syndrome	Oversewing of tear
Haemorrhoids	Haemorrhoidectomy
Anal fissure	Excision and plication
Diverticulosis	Partial colectomy
Angiodysplasia of colon	Partial colectomy

VII. NON-OPERATIVE TREATMENT FOR GASTROINTESTINAL BLEEDING

1. Pitressin infusion - systemic
   regional (selective)
   - effective in variceal bleeding, gastritis and diverticulosis
2. Somatostatin infusion
3. Endoscopic treatment - injection methods e.g. adrenaline
   - thermal methods e.g. heater-probe

VIII. FURTHER INVESTIGATIONS

1. Coeliac angiogram
2. Upper and lower gastrointestinal series
3. Colonoscopy
4. T^{C}-labelled RBC scan

IX. PROGNOSTIC FACTORS

1. Mortality rate for acute GIB = 10%
2. Clinical prognostic factors :
   * age > 60
   * concurrent cardiac, pulmonary, hepatic or renal diseases
   * transfusion requirement > 5 units
   * shock on admission
   * endoscopic stigmata
   * oesophageal varices

APPROACH TO UROLOGICAL PATIENTS

In the work-up of any patient the history is of paramount importance. It will be necessary to discuss here only those urological symptoms that are brought to the physician's attention by the patient.

I. SYMPTOMS

Change in Urine Appearance

Normal urine colour varies from light straw colour to deep amber colour depending on the concentration. Alteration in colour is often the most alarming to the patient.

1. Red urine - Haematuria
   Porphyria - exposed to sunlight
   Haemoglobinuria
   Colouring agent - in food and juices
   e.g., Rhodamine B
   Drugs - phenolphthalein

2. Cloudy urine - Microscopic haematuria
   Alkaline urine with precipitation of phosphate
   Pyuria
   Chyluria

3. Air in urine - Pneumaturia

Change in Urine Volume

The urine may be increased (polyuria) or diminished (oliguria) or absent (anuria). One must take into consideration the intake of the patients and loss of body fluid through other channels when interpreting these symptoms.

1. Polyuria
   - diuretic intake
   psychological cause
   diabetes mellitus
   diabetes insipidus

2. Oliguria
   - pre-renal condition causing decreased renal perfusion
   primary renal parenchymal disease
   urinary tract obstruction

Change in Micturition Habit

1. Frequency - (increase in frequency)
   polyuria
   small bladder capacity
   hypersensitive detrusor muscle - sensory
   motor
2. Continence - stress incontinence
   urge incontinence
   overflow incontinence
   enuresis

3. Act of micturition
   Obstruction - hesitancy
   slow/weak stream
   intermittency
   terminal dribbling
   sensation of incomplete voiding
   retention of urine
   Instability - urgency
   frequency
   dysuria
   nocturia

Pain

Pain is usually associated with inflammation or obstruction. Two types of pain: local and referred.

Local pain is felt in or near the involved organ. Thus pain from a diseased kidney (T10-12) is felt in the costovertebral angle and in the flank, in the region of and below the 12th rib. Pain from an inflamed testicle is felt in the gonad itself. Prostatic pain may be felt in the perineum.

Referred pain originates in a diseased organ but is felt at some distance from that organ. This is explained by the common segmental innervation of the area or organ, ureteric colic may be associated with pain radiating down the ipsilateral testicle (T11-12). The burning pain with voiding in cystitis is felt in the glandular urethra in male (S2-3). Pain from testicular torsion may be felt in umbilical area. Pain from prostatitis may radiate to groin and suprapubic areas.

Genital Symptoms

1. Urethral discharge
2. Haemospermia - blood in seminal fluid
3. Sexual dysfunction - erectile, ejaculatory
4. Infertility - primary
   secondary

Systemic Symptoms

1. Fever - with infection
2. Weight loss - chronic infection or malignancy
3. Symptoms of uraemia - multi-systemic
4. Anorexia

II. PHYSICAL SIGNS

Examination of the Urological System

1. General examination - with particular attention to the cardiovascular system and neurological system
2. Examination of abdomen for tenderness or abnormal masses along anatomical site of urinary tract
3. The external genitalia and perineum
4. Examination of the pelvic organ by per rectal or per vaginal examination
5. Examination of the urine by chemical and microscopic methods

III. EXAMINATION OF THE URINE

The most simple and rewarding but the most poorly performed laboratory test.

The three fallacies are:

1. Improper collection
2. Not examined when fresh
3. Incomplete examination of the sediment

Collection of Urine

1. Adult - mid-stream urine
   catheterised urine
   STAMEY'S Localisation of infection
   - VB_1 : initial stream 10 ml
   - VB_2 : mid stream
   - prostate massage
   - EPS/VB_3 : expressed prostatic secretion
     end stream of urine
2. Children - "mid-stream catch"
   suprapubic aspiration
   catheterised urine

Urinalysis

1. Inspection - colour of urine
   deposit
   volume of each micturition
2. Chemical examination - protein
   sugar
   Ketone
   pH 5.5-6.5
3. Microscopy - red blood cell
   pus cell
   cast
4. Microbiological study - simple Gram's stain followed by microscopy and culture for pyogenic or other specific organisms
5. Cytological examination - full stream urine fixed with equal volume of 50% alcohol
6. Special test of differential functions of parts of renal tubules
7. Eary morning urine (EMU) for acid fast baccilli (AFB)
8. 24 hour urine collection for calcium, oxalate, phosphate, urate in patients with urolithiasis

IV. UROLOGICAL INVESTIGATION

1. Renal Function Tests

   (a) Serum electrolytes
   (b) Blood urea and creatinine
   (c) Serum arterial blood acid/base status

2. Liver function tests

3. Prostate specific antigen (PSA)
   Specific glycoprotein secreted by prostatic epithelial cells
   A tumour marker used for monitoring of patients with adenocarcinoma of prostate

V. ROENTGENOGRAPHIC EXAMINATIONS IN UROLOGY

1. Plain film of Abdomen (KUB)

   (a) Renal shadows - size
   position
   shape
   (b) Calcification - location
   shape
   (c) Psoas shadows
   (d) Skeletal shadows
   (e) Gas patterns

2. Excretory Urogram (Intravenous Urogram - IVU) ± Tomography

Fundamental X-ray study of urinary tract

Principle

An iodine based contrast medium given intravenously is eliminated by glomerular filtration but not reabsorbed by the tubules. Water reabsorption causes a progressive increase in concentration of contrast (30-50x that of plasma) enabling opacification of the urinary tract.

Application

(a) Qualitative test of renal function
(b) Demonstration of anatomy of urinary tract, particularly of upper urinary tract
(c) Qualitative test of bladder function

3. Retrograde and antegrade ureterography

Principle

Introduction of a contrast medium into the upper urinary collecting system, with the aid of endourological technique:

Retrograde - from below - cystoscopic
Antegrade - from above - percutaneous

Application

Demonstration of anatomy of urinary tract, when IVU is not useful, e.g., non-visualised or poorly visualised kidney.

4. Micturiting Cystourethrography (Voiding Cystourethrogram)

Principle

Contrast medium is introduced into bladder via urethral or suprapubic catheter. Serial X-rays are then taken when the patient micturites.

Applications

(a) Anatomy of bladder and urethra
(b) Competency of vesico-ureteric junction e.g., U-V reflux
(c) Functional status of bladder/sphincter mechanism

5. Retrograde (Ascending) Urethrogram

Principle

Introduction of contrast medium into urethra with concurrent radiographic imaging.

Application

Anatomy of urethra e.g., before and after urethroplasty, and assessment of urethral injury.

6. Renal Angiography

Principle

Introduction of contrast into arteries (arteriography) or veins (venography) of urological organ after percutaneous cannulation. Therapeutic embolisation may be carried out, if indicated.

Applicatons

(a) Visualisation of anatomy and pathology of arteries in renal disease e.g., renal cell carcinoma, AVM
(b) Demonstration of venous drainage for staging of disease e.g., renal cell carcinoma with infiltration and tumour thrombus in the IVC

7. Lymphography

Principle

Injection of contrast material after cannulation of superficial lymphatic vessel (on the dorsum of foot) with ascending opacification of lymphatic system in inguinal, pelvic, retroperitoneal and mediastinal regions.

Applications

(a) To demonstrate pathology in lymph nodes which may be involved in neoplasm e.g., testicular and prostatic carcinomas
(b) To demonstrate abnormal lymphatic channel e.g., chyluria

VI. COMPUTERISED AXIAL TOMOGRAPHY

Principle

Computerised axial tomography differs from conventional radiology. The X-ray tubes and detector system are on opposite sides of the patient, and during a scan they rotate around the patient while recording information about the internal structure of their transverse cross section through which the X-ray beam is passing. Through a complex series of mathematical manipulations the computer reconstructs the cross-sectional image which bears remarkable resemblance to photographs from standard textbooks of cross-sectional anatomy. Different tissue density will be clearly shown in the final image.

Applications

(a) Differentiaton of renal mass
(b) Differentiation of adrenal mass
(c) Retroperitoneal pathology
(d) Evaluation of stage of bladder carcinoma with reference to depth of infiltration

VII. ULTRASONIC EXAMINATION IN UROLOGY

Principle

Ultrasound consists of sound waves with frequency of over 18,000 Hz which cannot be appreciated by human ear (medical ultrasound 1.5 MHz. When the beam strikes a boundary surface between tissues of different density, a portion of the beam is reflected as echoes, when detected by the transducers these echoes are converted to weak electrical impulse recorded as dots on a cathode ray screen.

Applications

(a) Differential diagnosis of consistency of renal mass
(b) Evaluation of renal size of non-visualising kidney
(c) Diagnosis of perirenal and retroperitoneal mass
(d) Assistance in percutaneous approach to kidney and collecting system
(e) Evaluation of intravesical and prostatic pathology (special intraluminal ultrasonic transducer 7.5 MHz)
(f) Scrotal mass differentiation : with doppler duplex scan

VIII. RADIOISOTOPIC UROLOGICAL STUDIES

Principle

The radiopharmaceuticals (eg. DMSA, DTPA) when injected intravenously are taken up and handled by the kidney in different ways and the radioactivity can be measured with accuracy by external imaging (DMSA) or clearance study (DTPA).

Applications

(a) For evaluation of regional function e.g., perfusion and structure e.g., cyst (DMSA)
(b) Measurement of overall kidney function and split function (DTPA)
(c) Assessment of obstruction (DTPA)
(d) Assessment of vesicoureteric reflux (DTPA)

IX. ENDOSCOPIC EXAMINATION IN UROLOGY (ENDO-UROLOGY)

Principle

Surgical procedure for the examination of inside of urinary tract by means of instrument introduced through an external opening which may be natural or artificial.

Instruments

- urethrocystoscope
- uretero-renoscope
- percutaneous nephroscope

Applications

(a) Direct inspection - diagnostic e.g. biopsy
(b) Retrograde and antegrade - radiological assessment of urinary tract
(c) Split renal function test
(d) Therapeutic - resection of tumour (TURBT) and BPH (TURP)
- removal of stone

X. URODYNAMIC STUDY

Principle

The study of the hydrodynamics of urine transport. The various methods of objective measurement of bladder and sphincter function include:

1. Uroflowmetry - basic test of objective urinary flow
2. Urethral pressure profile
3. Cystometry - intravesical volume/pressure change
4. Cineradiological study of micturition
   - bladder neck function
   - detrusor-sphincteric dyssynergia
   - reflux uropathy
5. Electromyography

TUMOURS OF THE GENITO-URINARY TRACT

I. KIDNEY

Parenchymal

A. Benign tumours

1. Adenoma
   a. small cortical lesions with a papillary histology
   b. cause and clinical incidence : unknown
      7 - 22% at autopsy
   c. incidental tumours : nephron-sparing surgery
   d. symptomatic tumours : radical nephrectomy

2. Angiomyolipoma
   a. 50% of patients with tuberous sclerosis
      (80% of tuberous sclerosis patients have angiomyolipoma)
   b. unencapsulated yellow or gray lesion of multicentric origin
   c. fat on CT scan is pathognomonic
   d. asymptomatic lesion and < 5 cm : observe

3. Juxtaglomerular cell tumour
   a. profound hypertension
   b. arise from pericytes with renin secretory granules

B. Malignant tumours

1. Renal cell carcinoma

1.1. Pathology
commonest renal parenchymal tumour
arises from proximal convoluted tubules
areas of necrosis and haemorrhage
spread : haematogenous
lymphatic
local

1.2. Symptoms and Signs
   a. urological symptoms:
      haematuria, pain and flank mass (triad), acute varicocele
   b. metastatic deposits:
      lung, bone and brain
   c. non-specific signs of malignancy:
      pyrexia, anaemia
   d. non-metastatic effects of malignancy:
      hypertension (↑ renin)
      polycythaemia (↑ erythropoietin)
      hypercalcaemia (↑ ectopic PTH)

1.3. Investigations
- a. IVU : calyceal distortion
- b. ultrasound : renal vein and caval tumour thrombus
- c. CT scan : extrarenal spread, lymphadenopathy, tumour thrombus within the renal vein or IVC, or liver metastases
- d. arteriography and inferior vena cavography : selected cases
- e. percutaneous fine needle biopsy

1.4. Treatment
- a. localised disease : radical nephrectomy
- b. disseminated disease : radiotherapy/chemotherapy - ineffective; immunotherapy/hormonal therapy - variable results

2. Nephroblastoma (Wilm's tumour)

2.1. Pathology

highly malignant renal tumour arising from embryonal cells
children under age 6
bilateral : 10%
spreads mainly via the blood stream

2.2. Symptoms and Signs

most appear healthy and symptom free
non-tender abdominal mass : 90%
abdominal pain, hypertension, or haematuria (30%)

2.3. Investigations
- a. IVU : calyceal distortion
- b. ultrasound/CT scan : solid renal mass, contralateral renal disease, liver secondaries, retroperitoneal lymphadenopathy, tumour thrombus
- c. CT scan of chest
- d. arteriography/inferior vena cavography : significant morbidity

2.4. Differential diagnosis
- a. hydronephrosis, polycystic and multicystic renal conditions
- b. neuroblastoma : elevated urinary V.M.A., displacement of an otherwise normal looking kidney on IVU

2.5. Treatment

various combination of all 3 modalities of surgery, radiotherapy, and chemotherapy

II. UROTHELIAL NEOPLASMS

1. Transitional cell carcinoma (TCC)

a. etiology
   - diffuse multicentric change
   - exposure to multiple carcinogens excreted in the urine
   - heavy smokers or heavy analgesic consumers; long exposure to organic solvents in the dye, leather and paint industries

b. behaviour
   - multiple lesions on initial diagnosis
   - "recurrences" : new tumours

c. pathogenesis
   - papillary lesions of medium or low grade :
     - non-invasive or only superficially into the lamina propria
   - non-papillary lesions of high grade:
     - muscle invasion and dissemination
   - not a single disease process that progresses
     - atypical changes ---> superficial lesions ---> invasive carcinoma

d. spread
   - direct extension, or via lymphatics
   - thin walls of calyces, pelvis, and ureter :
     - less resistance to invasion

2. TCC of Renal Calyces, Pelvis and Ureter

2.1. Symptoms and Signs

a. haematuria (90%)
b. dull renal angle pain (30%) :
   calyceal, pelvic, or ureteric obstruction
c. acute ureteric colic due to clots

2.2. Investigations

a. IVU $\pm$ retrograde pyeloureterogram :
   - filling defect (ddx uric acid calculi, blood clots, sloughed renal papillae, and renal adenocarcinoma)

b. urinary cytology
c. cystoscopy :
   - co-existing TCC of bladder

2.3. Treatment

a. nephroureterectomy and regular follow up cystoscopy
b. segmental resection : selected cases
c. radiotherapy/chemotherapy : little value

3. TCC of Bladder

3.1. Symptoms and Signs
- a. painless haematuria ± clot retention
- b. irritative bladder symptoms (c.i.s.)
- c. obstruction : ureteric orifice, bladder neck

3.2. Investigations
- a. cytology
- b. cystoscopy and biopsy
- c. TUR + EUA for staging

3.3. Treatment
- a. carcinoma-in-situ (3 - 4%)
  - high grade malignancy of urothelium
  - resistant to radiotherapy
  - intravesical BCG/chemotherapy ± radical cystectomy
- b. superficial disease (stage Ta, T_1 and low grade, minimal T_2) (70%)
  - recurrences : 50 - 70%
  - increase in grade and/or stage : 20%
  - transurethral resection ± intravesical BCG/chemotherapy
- c. muscle invasive disease (stage T_2, T_{3a} and T_{3b}) (25%)
  - radical cystectomy and urinary diversion/ bladder replacement
  - radiotherapy
- d. disseminated disease
  - chemotherapy ± palliative resection/ radiotherapy

III. PROSTATE GLAND, TESTIS AND PENIS

1. Carcinoma of Prostate

1.1. Epidemiology
- incidence of latent cancer : same
- mortality rates : striking difference

1.2. Aetiology ?
- viral
- hormonal : excess of androgenic hormones at puberty
- familial
- environmental : Cadmium

1.3. Pathology
- cancer area : firm and gritty, no well-defined edge
- invasion : capsule, seminal vesicle, bladder, rectum
- histology : adenocarcinoma

1.4. Clinical Features

a. incidental : found incidentally during histological examination of prostatectomy specimen
b. clinical : outflow obstruction and abnormal rectal examination
c. occult : only metastatic disease is symptomatic
d. miscellaneous : intestinal obstruction, ureteric obstruction, haemorrhages and subcutaneous bruising (fibrinolysins)

1.5. Investigations

a. serum acid phosphatase
b. prostate-specific antigen
c. transrectal ultrasound
d. bone scan/x-ray
e. CT scan/MRI
f. prostatic biopsy

1.6. Treatment

a. incidental cancer (T_{0a}/A_1) :
observation
b. early prostatic cancer (T_{0b}, T_1 & T_2/A_2, B_1 & B_2) :
radiotherapy/radical prostatectomy
c. locally advanced prostatic cancer (T_3, T_4/C) :
local control to minimize urethral and ureteral obstruction (DXT or interstitial implants)
and androgen deprivation
d. disseminated disease (N_{1-4} & M_1/D_1 & D_2) :
i. hormonal -
orchidectomy
oestrogens
LHRH analogues (chemical castration)
anti-androgens
surgical ablative procedures (adrenalectomy/hypophysectomy)
medical ablative procedures (aminoglutethimide & spironolactone)
ii. chemotherapy -
toxic and ineffective
iii. radiotherapy -
for bone pain

2. Carcinoma of Testis

2.1. Epidemiology and Etiology

a. 3 peak age groups
i. infants and children
ii. young adults (20 - 40)
iii. male above 50

b. maldescent
cryptorchid testis 20 - 40 x,
account for 10% of germ cell tumours,
not affected by orchidopexy,
10% on contralateral normal testis

2.2. Pathology

a. classification
   i. germ cell tumours
      seminoma
      embryonal carcinoma
      teratoma
      choriocarcinoma
      yolk sac tumour
   ii. sex cord-stromal tumour
   iii. lymphoid tumours
   iv. secondary tumours

b. natural history
   disseminate primarily by metastatic spread to regional LN (except choriocarcinoma)
   distribution of metastases :
      retroperitoneal LN
      lungs
      liver
      mediastinal LN

c. tumour markers
   αFP & ßHCG

2.3. Clinical Features

a. testicular swelling/pain
b. symptoms of metastases
c. age predilections :
   1st decade - yolk sac tumours
   2nd decade - choriocarcinoma
   3rd decade - embryonal carcinoma
   4th decade - seminoma

2.4. Clinical Staging

I. No evidence of disease outside the testis
II. Infradiaphragmatic node involvement
III. Supradiaphragmatic node involvement
IV. Metastases to extralymphatic sites

2.5. Treatment

a. Nonseminomatous germ cell testis tumours
   i. low-stage : retroperitoneal LN dissection
   ii. high-stage : chemotherapy ± surgery

b. Seminoma
   i. low-stage : radiotherapy
   ii. high-stage : chemotherapy ± surgery

3. Carcinoma of Penis

3.1. Pathology
squamous cell carcinomas and metastasize to the inguinal lymph nodes

3.2. Clinical Features
elderly, unwashed, uncircumcised man
enlarged inguinal LN

3.3. Treatment
circumcision
amputation/radiotherapy
lymph node dissection

URINARY TRACT INFECTIONS

I. SPECIFIC INFECTIONS

Tuberculosis

1. Mycobacterium tuberculosis
2. Entry: pulmonary, haematogenous spread, ascending vs descending
3. Slow progression, long lag time
4. Granulomatous reaction, caseation
5. Symptoms/signs: incidental
   "cystitis", sterile pyuria
   epididymis, prostate
6. Laboratory findings
   - acid-fast bacilli (AFB)
   - early morning urine for bacilli culture
   - CXR
   - IVP
7. Treatment
   systemic - anti-TB treatment
   surgical - drainage
   nephrectomy
   diversion
   replacement

Gonorrhoea

1. Neisseria gonorrhoea
2. Urethral infection
3. Symptoms/signs: urethral discharge, dysuria
   asymptomatic female
   complications:
   - acute pelvic inflammatory disease
   - Fitz-Hugh-Curtis syndrome (perihepatitis)
   - urethral stricture
4. Labortory: Gram stain
   CO_2 atmosphere culture
5. Treatment: penicillin
   tetracycline
   spectinomycin (trobicin)
   dilation/incision of strictures
   drainage of abscesses

Others

1. Schistosomiasis
2. Filariasis
3. Candidiasis
4. Trichomoniasis

II. NON-SPECIFIC INFECTIONS

Epidemiology

1. 10-20% females at least one episode
2. End stage renal failure

Pathogenesis

1. Organisms - 80% E. coli, other enterobacteriaceae in faecal flora
2. Ascending route
   - Rectum - introitus-urethra-bladder
   - Rectum - urethra-bladder/prostate
3. Haematogenous
4. Catheter - associated
5. Predisposing factors - stasis, anomalies, foreign body

Symptoms/Signs

1. Lower tract - frequency, urgency
   - dysuria, haematuria
2. Upper tract - fever, chills
   - flank pain/tenderness

Laboratory Investigations

1. Methods of urine collection
   - midstream
     VBI, VB2, EPS, VB3
     suprapubic aspiration
     catheterisation
2. Microscopy - pyuria/bacteriuria
3. Culture - 105 organisms/ml: significance@@ E
4. Upper tract localisation: antibody-coated bacteria

Acute Pyelonephritis

1. Ascending route (by reflux) most common
2. Fever/chills/flank pain
3. Leucocytosis/flank tenderness/normal IVP
4. Treatment - parenteral antibiotics
5. Special situations: pregnancy
   male infants

Chronic Pyelonephritis

1. Recurrent pyelonephritis during renal development

2. Could be unrecognised and silent and discovered on presentation with uraemia or hypertension

3. IVP shows small contracted kidney with multiple scarring and delayed function

Renal Carbuncle/Perinephric Abscess

1. Staph. aureus/Gram negative organisms

2. Haematogenous/ascending/complicated infections

3. Incomplete or no treatment

4. Necrosis and abscess formation + rupture into perinephric@@ C space

5. Fever/chills/sepsis/flank mass and tenderness

6. Drainage is mandatory

Acute Cystitis

1. Ascending infection

2. 'Honeymoon cystitis' in females

3. Instrumentation/catheterisation in males

4. Symptoms/signs: frequency, urgency, urge incontinence, suprapubic pain, dysuria

5. Investigations: microscopy and culture/sensitivity only

6. Organisms: enterobacteriaceae
   80% E. coli

7. Treatment: antibiotics
   symptomatic relief (?)

Prostatitis

1. Symptoms: frequency, dysuria, urgency,
   perineal pain,
   fever, chills, retention

2. Classifications: acute prostatitis] bacterial
   chronic prostatitis]
   abacterial prostatitis (mycoplasma, chlamydia)
   prostatodynia

3. Prostatic fluid: leucocytes, pH, culture

4. Lower tract localisation/3-glass technique

5. Treatment:
   acute prostatitis - aminoglycosides
   chronic prostatitis - sulfamethoxazole-trimethoprim
   as prophylaxis + treatment of
   acute episodes
   abacterial prostatitis - tetracycline; erythromycin
   prostatodynia - smooth/skeletal muscle relaxants

6. DDX: torsion of testis, testicular tumours
   tuberculous epididymitis

7. Organisms - unknown

8. Treatment - antibiotics
   symptomatic

Urethritis/Orchitis

References :

1. Stamey, T.A. 'Pathogenesis and treatment of urinary tract infections', Williams & Wilkins, 1980.

2. Smith, D.R. 'General Surgery', Lange Series Chapters 12, 13, Lange Medical Publications, 1981.

CALCULOUS DISEASE OF THE GENITO-URINARY TRACT

Clinical Stone Formation

1. Supersaturation for the precipitating salt (continuous/ intermittent)
   a. nucleation (birth of crystals from solution)

Activity product	
Spontaneous Nucleation	Unstable
Formation product	
Heterogeneous Nucleation Crystal Growth	Metastable
Solubility product	
Crystal Dissolution	Undersaturated

   b. crystal growth ± aggregation

2. Inhibitors (nucleation or crystal growth)
   e.g. pyrophosphate, citrate, Mg ($Ca \cdot PO_4$)
   glycosaminoglycans (Ca·oxalate)

3. Crystal retention
   anatomic abnormalities or adherence to epithelium
   (may involve a specific molecular interaction)

Epidemiology

1. Increased dietary protein intake
   ↑ urinary calcium
   ↓ urinary citrate

2. Decreased dietary fiber
   ↑ G.I. absorption of calcium

Aetiology

1. Idiopathic calcium urolithiasis (70%)

2. Secondary urolithiasis (15%)
   a. infection (triple phosphate)
   b. obstruction (uric acid bladder stone)
   c. urinary diversion
   d. drugs
   e. enteric hyperoxaluria (Ca·oxalate)

3. Uric acid lithiasis (10%)
   too much uric acid or excessively acid urine
   a. idiopathic
      normal serum & urine uric acid levels, low urine pH
   b. gout
   c. myeloproliferative diseases
   d. low urine output states
      ileostomies or chronic diarrhoea

4. Hypercalcemic disorders (5%)
   a. primary hyperparathyroidism
   b. immobilization (resorption of bone)

5. Renal tubular syndromes (<1%)
   a. renal tubular acidosis
   b. cystinuria

6. Enzyme disorders (<1%)
   a. xanthinuria (deficiency of xanthine oxidase)
   b. primary hyperoxaluria

Presentation

1. Usually silent unless infection/obstruction occurs
2. Flank pain and typical renal colic with radiation
3. Haematuria/smoky urine, clots uncommon
4. Fever and flank tenderness, septicaemia with obstructive pyonephrosis or pyelonephritis
5. Calculous anuria
6. End-stage renal failure
7. Bladder obstructive symptoms and cystitis

Investigations

1. Urinalysis
   haematuria, bacteriuria, pyuria, pH, culture, presence of crystals not significant
2. Renal function and serum calcium and urate
3. Plain X-ray KUB - 95% opaque
4. Intravenous urography for position and obstructive effects
5. Radio-isotopic renography for proper renal function and obstruction
6. 24-hour urine collection for calcium, phosphate, oxalate, urate and cystine
7. Stone analysis and stone fragments for culture
8. Post-operative 24-hour urine and IVU for progress and recurrence

Goals of Therapy

1. Eliminating the symptomatic calculus
2. Achieving a stone-free state
3. Eradicating stone-related bacteriuria
4. Choosing the procedure that
   a. has the lowest morbidity
   b. is least invasive
   c. is least costly
   d. provides the lowest likelihood of residual fragments

Surgical Management - treatment of primary metabolic disorder first
- nephrectomy should not be done for stones

1. Stones not requiring intervention

   a. calyceal stones without obstruction
   b. medullary sponge and calcifications
   c. small non-obstructing ureteric stones < 5 mm

2. Treatment for renal stones

   a. extra-corporeal shockwave lithotripsy
   b. percutaneous nephrolithotripsy
   c. pyelolithotomy and nephrolithotomy as reserve

3. Treatment for ureteric stones

   a. extra-corporeal shockwave lithotripsy
   b. ureteroscopic lithotripsy
   c. endourological pushing and basketing
   d. ureterolithotomy as reserve

4. Treatment for bladder stones

   a. mechanical endoscopic litholapaxy
   b. ultrasonic and electrohydraulic lithotripsy
   c. suprapubic lithotomy
   d. treatment of obstruction such as TUR prostate

Medical Management

1. Aim:
   prevent the formation of new stones or further growth of old stones

2. Principles:

   a. reduction of urinary supersaturation
   b. increase in net inhibitory activity

3. Dietary Advice:

   a. high fluid intake to maintain urine volume at 3 litres per 24 hours
   b. dietary moderation
      low-calcium and low-oxalate diet

4. Pharmacologic Therapy:
   proven metabolically active stone disease
   lifelong

   a. renal tubular acidosis
      alkalization with K^+ citrate
        ↑ renal citrate excretion
        ↓ Ca excretion

   b. cystinuria
      alkali therapy to increase urinary pH to 7.5-7.8
      D-penicillamine
        penicillamine - cysteine (more soluble)

   c. hypercalcemic disorders
      immobilized patient - oral orthophosphates

   d. uric acid lithiasis
      alkali therapy
        maintain urine pH at 6.5
      allopurinol (xanthine oxidase inhibitor)

   e. primary hyperoxaluria
      pyridoxine

   f. enteric hyperoxaluria
      low oxalate & fat diet
      cholestyramine
        ↓ oxalate absorption
        ↑ water absorption

   g. infection stones
      complete removal of stone
      post-operative irrigation with hemiacidrin
      antibacterial prophylaxis x 3 months
      acidification with ammonium chloride
      urease inhibitor (acetohydroxamic acid)

   h. idiopathic calcium urolithiasis
      thiazide + moderate sodium restriction for
        hypercalciuria
        ↓ urinary calcium
        ↓ urinary SS x Ca·oxalate & $Ca \cdot PO_4$
        S/E-fatigue, muscle weakness/cramping,
          ↓ libido, impotence
      orthophosphate (absorbable phosphate)
        ↓ urinary calcium & ↑ urinary inhibitor
        S/E diarrhoea
      cellulose phosphate + low oxalate diet
        ↓ G.I. absorption of calcium

URAEMIA AND RENAL TRANSPLANTATION

ROLE OF SURGEONS IN URAEMIA

1. Surgery for prevention of progressive renal failure.
2. Surgery for complications of uraemia.
3. Surgery for treatment of end-stage uraemia.

Surgical Correctable Causes of Chronic Renal Failure

1. Renal calculous disease

2. Surgical hypertension -
   renal vascular lesions
   endocrine lesions: Conn's syndrome
   phaeochromocytoma

3. Obstructive uropathy -
   pelvi-ureteric junction: congenital PUJ
   ureteric: stones, strictures, periureteric obstruction (retroperitoneal fibrosis)
   uretero-vesical junction:megaureter
   bladder: carcinoma
   bladder outlet: prostatic carcinoma
   benign prostatic hypertrophy
   urethral stricture

Complications of Chronic Renal Failure Requiring Surgery

1. Secondary or tertiary hyperparathyroidism
2. Uncontrollable renal parenchymal hypertension
3. Bleeding from polycystic kidney;
4. Constrictive pericarditis

The Risk Factors in Patients with Chronic Renal Failure Undergoing Surgery

1. Anaemia
2. Hypertension
3. Fluid and electrolyte imbalance, over-hydration, hyperkalaemia, acidosis
4. Clotting defects - platelet dysfunction, use of heparin
5. Impaired host defence mechanism
6. Hypoproteinaemia

Surgical Treatment of Uraemia

1. Vascular access surgery for haemodialysis
2. Renal homotransplantation

Vascular Access

1. Repeated and atraumatic
2. Provide high blood flow rate
3. Easily accessible part of body

Short-Term Access

1. Direct cannulation of central vein e.g., subclavian or femoral
2. External arteriovenous shunts

Long-Term Access

1. Internal arteriovenous fistula

2. Arteriovenous bridge graft
    - Saphenous vein
    - Polytetrafluorethylene (Gortex)
    - Bovine vessels
    - Other vein grafts

II. TRANSPLANTATION

Definition

Surgical procedure of transferring tissues or organs from one part to another of the same body or another individual.

Classification

1. Autograft - from the same individual
2. Homograft - from another individual of the same species
3. Isograft - from another individual of identical genetic structure.
4. Heterograft - from another animal of different species (xenograft)

RENAL HOMOTRANSPLANTATION

Donors - cadaveric
living-related

THE CADAVERIC DONORS

The major ethical problem is the definition of brain death. Brain death occurs when irreversible brain damage is so extensive that the organs enjoy no potential for recovery and can no longer maintain the body's internal homoeostasis i.e., respiration, cardiovascular function and temperature.

Pathologically in brain death both the cerebrum and brain stem are damaged whereas in vegetative state the brain stem is still functional.

Criteria for Brain Death (Set out by the Royal Colleges and their faculties in 1976).

Tests for confirming brain death. All brain stem reflexes should be absent.

1. Pupils fixed and no light reflex
2. Absence of corneal reflex
3. No gag reflex
4. No vestibulo-ocular reflex (caloric test)
5. No cranial nerves motor response to somatic stimulaton
6. No respiratory response despite adequate PCO_2 stimulation (PCO_2 - 50 mmHg)

Other Criteria to be fulfilled

1. There should be no suspicion that this state is due to depressant or paralysing drugs.
2. Exclude hypothermia
3. Exclude metabolic and endocrine disturbances

CADAVERIC KIDNEY DONOR SELECTION

General Criteria

1. 5-55 years of age
2. Normal renal function
3. No malignancy outside the central nervous system
4. No significant hypertension
5. Not diabetic
6. Australian Antigen status

PROCUREMENT OF CADAVERIC KIDNEYS

Aim

1. Minimal warm ischaemic time
2. Intact ureteral blood supply
3. Preservation of all anomalous renal vessels

THE LIVING DONOR

1. Justification
2. Motivation

THE RECIPIENT
- choice between dialysis and transplantation
- success rate of transplantation
- complication associated with transplantation

KIDNEY PRESERVATION - value of satisfactory organ preservation.

1. Ensure initial good organ function
2. Provide extra time allowing for
   (a) adequate tissue matching
   (b) semi-elective operation
   (c) adequate recipient preparation
   (d) organ sharing programme

Effect of Simple Cooling on Organ Preservation

>25°C	ineffective
25° - 15°C	2 hours of ischaemia
15° - 5°	6 hours
5° - 0°	10 hours

For Prolonged Protection

1. Ice storage (4°C) after initial cold flush with hypertonic intracellular solution
2. Machine perfusion - albumin

IMMUNOLOGY OF TRANSPLANTATION

Principle of Tissue Typing

Gorer (1937) stated:

"Tissues contain genetically determined antigenic factors and that if such tissues are transplanted to a recipient lacking the same factors then under normal circumstances an immune response is generated which usually results in destruction of the incompatible graft.

In human, these antigenic factors are composed of at least five series of antigen controlled by genetic loci on the sixth chromosome in a region known as the major histocompatibility complex (MHC). The series of antigen are named according to the locus controlling that series of antigen (A, B, C, D and DR) followed by numerical designation.

The HLA antigens are inherited in a codominant fashion and as a genetic unit or haplotype. Each parent contributes a haplotype consisting of the antigen from each of the five series, so that a fully typed individual would have a total of ten antigens. For clinical purpose most HLA typing will be expressed in term of A, B, sometimes C and normally DR antigen".

The Rejection Response

Burnet Clonal Theory:

"Lymphoid system is being made up of a large number of different clones or family of lymphocytes. All members of a clone are identical and have only one type of receptor for antigen. It follows that the immune response to any antigen is mediated by only a tiny fraction of the host lymphocyte pool".

Lymphocyte Subpopulation

T and B cells and further subdivision are different in their function; cell surface antigen, recirculation rate and site of residence.

Effector Mechanism

1. Cellular infiltrate - T cell component

2. Antibodies
   (a) pre-formed antibodies
   (b) antibodies formed in response to the graft

IMMUNOSUPPRESSION

Non-specific - general depression of the immune system (all clones of lymphocytes).

Standard Non-specific Immunosuppression

1. Steroid - lymphocytotoxic
2. Azathioprine - inhibits lymphoid differentiation
3. Cyclosporin A
4. Combination

Other Modalities

1. Antilymphocytic globulin
2. Total lymphoid irradiation
3. Thoracic duct drainage
4. Monoclonal antibodies
5. Transfusion effect

Specific Immunosuppression

One which directly or indirectly suppresses the action of the lymphocyte clones which are reactive to the donor histocompatibility antigen. The monoclonal antibody is the hope in this direction.

Complications of Transplantation

1. Rejection and loss of graft
   - hyperacute rejection - within 1 day
   - acute rejection - within 1 month
   - chronic rejection - 1 month to years

2. Surgical complication secondary to technical error or rejection
   - renal artery stenosis
   - urological complications - fistulae
     obstruction
   - lymphocele

3. Complications of long-term steroid administration
   - Cushingnoid appearance
   - impaired growth in children
   - diabetes
   - peptic ulceration
   - avascular necrosis of head of femur
   - impaired wound healing

4. Infections
   - major cause of death
   - may alter state of immunity and precipitate rejection
   - atypical site and presentaton

   Common infections
   - pulmonary - bacterial, tuberculosis and fungal (cryptococcosis)
   - urinary tract
   - viral infection - particularly CMV (cytomegalic virus), herpes

5. Malignant neoplasm
   - risk factor increased by 100 folds that of age match control
   - incidence increases with time after transplant
   - common neoplasms are cutaneous squamous carcinoma and lymphoma

SURGERY OF THE LUNG AND MEDIASTINUM

I. THE LUNG

The surgical diseases of the lung consist of:

1. Pulmonary neoplasms - benign and malignant
2. Spontaneous pneumothorax
3. Bronchopulmonary suppurations - empyema, bronchiectasis and lung abscess
4. Pulmonary tuberculosis

BRONCHIAL CARCINOMA

During the last few decades there has been a worldwide increase in deaths from lung cancer.

Aetiology

1. Smoking - Smoking of 30 cigarettes a day causes a 30-fold increase in lung cancer risk.
2. Atmospheric pollution - hydrocarbons like 3:4 benzpyrene
3. Occupational factors - Uranium mines and asbestos factory

Clinical Features

1. Respiratory symptoms -
   cough, haemoptysis, chest pain, dyspnoea
2. Acute respiratory infection -
   pneumonia, lung abscess
3. General symptoms -
   anorexia, weight loss, tiredness, ill health
4. Asymptomatic, abnormal chest X-ray

5. Features due to local extension of tumour or mediastinal metastases -

   Pleural effusion, rib involvement, nerve involvement
   S.V.C. obstruction, pericardial involvement,
   Oesophageal obstruction, tracheal obstruction,
   Pulmonary lymphangitis carcinomatosa

6. Features due to distant metastases

   Cervical lymphadenopathy, cerebral metastases,
   Bone metastases, liver metastases

7. Features due to non-metastatic syndromes

   Hypertrophic pulmonary osteo-arthropathy
   Migratory thrombophlebitis
   Neuromuscular syndromes
   Endocrine syndromes

Diagnosis

Finger clubbing (60%)

1. CXR: (a) dense hilar opacity
   (b) 'coin' lesion - a solitary nodule
   (c) ill-defined shadow - patch
   (d) cavitary lesion
   (e) collapse
   (f) lymphangitis carcinomatosa
2. Sputum cytology - Positive in over 80%
   False positive less than 1%

3. Bronchoscopy - Rigid or flexible

4. Mediastinoscopy or anterior mediastinotomy

5. Needle biopsy

6. Diagnostic thoracotomy

7. Pleural aspiration and biopsy

Prognostic Factors

1. Histological type - Squamous cancers
   Adenocarcinoma
   Anaplastic large - cell
   Anaplastic small - cell (oatcell cancer)

2. Staging - Stage I: tumour 3 cm or less
   (rough) ipsilateral hilar nodes - positive or negative

   Stage II: tumour more than 3 cm
   ipsilateral hilar nodes - positive

   Stage III: extensive tumour
   mediastinal nodes positive or distant metastasis

3. Presence of vascular invasion

4. Extent of immunologic reactivity in the resected specimen

A squamous cancer of Stage I without vascular invasion and showing a high immunologic reactivity in the specimen has the best prognosis.

Treatment

1. Surgery
2. Radiotherapy
3. Chemotherapy

Surgery - treatment of choice; only 30% of all patients are suitable for surgery. Surgery involves lobectomy or pneumonectomy.

Contraindications to Surgery

1. Inadequate pulmonary function

   FEV_1 , less than 1.01 (less than 60% of predicted value)

   Elevation of $PaCO_2$

2. Local extension of tumour or metastases

3. Surgery has a higher mortality in patients over 70

BRONCHIAL ADENOMAS (TUMOURS OF MUCUS GLAND ORIGIN)

1. Carcinoid tumour
2. Cylindroma (adenoid cystic carcinoma)
3. Muco-epidermoid tumour
4. Mixed tumour (low-grade malignancies)

Clinical Features

1. Cough
2. Haemoptysis
3. Pneumonitis
4. Wheeze
5. Fever
6. Carcinoid syndrome.

CXR

Hilar or peripheral mass
Pneumonitis, atelectasis

Treatment

Surgical resection

SOLITARY METASTATIC PULMONARY NODULES

Common Sites:

1. Colon
2. Breast
3. Rectum
4. Kidney
5. Cervix or uterus
6. Testis or ovary
7. Sarcomas.

BENIGN TUMOURS OF THE LUNG

1. Hamartoma
2. Benign fibrous mesothelioma
3. Xanthomas
4. Lipoma
5. Leiomyoma
6. Haemangioma

SPONTANEOUS PNEUMOTHORAX

Collection of air between the parietal and visceral pleurae.

2 Groups

1. 'Simple Pneumothorax' -

   Occurs in young and otherwise healthy people.
   Results from rupture of subpleural bullae, commonly located in the apical segments.

2. Pneumothorax associated with Chronic Obstructive Airway Disease (COAD)

Dangers of Pneumothorax

Tension pneumothorax
Simultaneous bilateral pneumothorax

Treatment

1. Intercostal drainage

2. Surgical treatment

   (a) Open pleurodesis - thoracotomy, ligation of bullae, pleurectomy or mechanical rub in fit patients

   (b) Closed Pleurodesis - Talc pleurodesis,
   older patients with COAD - patients unsuitable for G.A. and thoracotomy

BRONCHOPULMONARY SUPPURATIONS

Lung Abscess

A localised area of pulmonary suppuration and necrosis with a central cavity, caused by infection with pyogenic organisms.

The incidence of lung abscess has fallen as a result of:

1. Improved methods of anaesthesia
2. Use of antibiotics for treating acute respiratory infections
3. Improved oral hygiene

Causative Organisms

Staph. aureus, Str. pneumoniae, K. pneumoniae, H. influenza, Proteus, Pseudomonas, E.coli and Anaerobic bacteria

Routes of Infections

1. Through the bronchial tree
2. Via bloodstream
3. Through the chest wall or the diaphragm

Causes

1. Bronchial carcinoma
2. Inhalation of foreign material
3. Pneumonia
4. Septicaemia
5. Pulmonary infarction
6. Chest wounds

EMPYEMA

Purulent pleural effusion. The pus may lie in the general pleural space or may be loculated (encysted empyema).

Empyema may be acute or chronic and is usually unilateral.

Causes

Empyema is usually secondary to pneumonia, lung abscess, bronchiectasis or tuberculosis. Infection reaches the pleural space through the bronchial tree, the bloodstream or the chest wall (trauma). Postoperative empyema usually results from a bronchopleural fistula.

Since the widespread use of antibiotics, empyema has become a rare complication.

Diagnosis

Clinical features are:

1. Due to underlying cause of the empyema
2. Fluid in the pleural space
3. Systemic symptoms

Chest radiograph
Pleural aspiration

Treatment

1. Pleural aspiration - by repeated needle aspiration or by insertion of an intercostal drain with waterseal drainage

2. Antibiotics
   Surgery - is occasionally necessary to resect a chronic empyema and to carry out decortication of the lung in order to obtain re-expansion.

BRONCHIECTASIS

Pathological dilatation of the bronchi. Chronic infection leads to persistent cough and purulent sputum. Its prevalence has declined considerably since antibiotics have been available for treatment of acute respiratory infections.

Aetiology

1. Congenital causes - dextrocardia, cystic fibrosis, congenital hypogammaglobulinaemia.

2. Acquired bronchiectasis - bronchial obstruction and infection are responsible. Whooping cough, measles and pneumonia, foreign bodies, bronchial adenoma, tuberculosis, allergic bronchopulmonary aspergillosis.

Diagnosis

1. Cough, sputum, recurrent haemoptysis, recurrent pneumonia and pleurisy, breathlessness, chronic sinusitis, finger clubbing.

2. Chest radiograph

3. Sputum examination

4. Bronchography - can confirm the diagnosis and is always indicated when surgery is contemplated to localise the extent of the disease.

5. Bronchoscopy

Treatment

1. Postural drainage

2. Antibiotics
   Surgery - is indicated in patients with persistent troublebsome symptoms due to localised bronchiectasis. Results of surgery are excellent in patients with localised bronchiectasis.

PULMONARY TUBERCULOSIS

Considered to be a "surgical" disease 15-20 years ago.

Effective chemotherapeutic agents have

- dramatically altered all phases of management of the disease

- caused great contracture in the indications of surgery

Indications of Surgery

1. Residual lesions, open cavity, bronchiectasis
2. Destroyed lobe or lung
3. Complications e.g., empyema, bronchopleural fistula
4. Tuberculoma
5. Failed medical treatment - drug resistance, sensitivity or toxicity

Timing of Surgical Intervention

1. Indications for surgical treatment are established
2. Chemotherapeutic control has been achieved

Surgical Treatment

1. Collapse therapy (Pre-chemotherapy era)

   Scalenotomy, phrenic interruption, artificial pneumothorax, Pneumoperitoneum, thoracoplasty

2. Resection (Post-chemotherapy era)

   Segmental resection, lobectomy, pneumonectomy

THE MEDIASTINUM

ACUTE MEDIASTINITIS

Causes

1. Oesophageal perforation during oesophagoscopy or
2. Oesophageal rupture secondary to violent vomiting, lye ingestion, foreign body ingestion, external trauma
3. Postoperative oesophageal anastomotic leak
4. Mediastinitis following open heart surgery

Diagnosis

1. Fever
2. Respiratory distress
3. Pain
4. Dysphagia

CXR

Widened mediastinum, pneumomediastinum, pneumothorax, pleural effusion, hydropneumothorax

Gastrografin Swallow

Treatment

1. Antibiotics
2. I.V. fluid
3. Surgery (drainage, definitive operation)

MEDIASTINAL TUMOURS

Primary Tumours

Anterior Mediastinal Tumours

Thymomas
Lymphomas
Dermoids
Teratomas
Mediastinal thyroid
Mediastinal cysts (bronchial, pericardial)

Posterior Mediastinal Tumours

Neurogenic tumours

Rare Mediastinal Tumours

Fibromas
Sarcomas
Carcinomas (primary)
Enterogenous cysts
Lipomas
Xanthomas

Clinical Features

Usually due to pressure, and depend on the structures involved.

Diagnosis

1. CXR - PA and lateral, tomography, screening, ultrasound, CT scan
2. Mediastinoscopy
3. Thoracotomy

Treatment

Lymphatic tumours - non-surgical treatment

Others - should be excised to prevent or relieve pressure and to avoid malignant change.

PRINCIPLES OF CARDIAC SURGERY

The Normal Heart

Common Operable Heart

1. Septal defect
2. Ventricular outflow obstruction
3. Abnormal vascular connection
4. Complex anomaly
5. Valvular dysfunction
6. Coronary artery disease

The Normal Circulation

Haemodynamic Changes in Heart Disease

1. Volume overload ± shunting
2. Pressure overload ± shunting
3. Inadequate or inappropriate blood flow

Indications for Surgery

1. Uncontrollable symptoms e.g., exercise intolerance, angina pectoris
2. Intractable heart failure
3. Increasing hypoxaemia
4. Increasing severe reversible pulmonary hypertension
5. Growth failure or recurrent chest infection in the presence of significant haemodynamic derangement
6. Significant haemodynamic abnormality
   - shunting
   - pressure gradient
7. Unfavourable clinical course

Types of Surgery

1. Palliative
2. Corrective

Palliative Surgery

1. Augmentation of pulmonary blood flow
   Pulmonary artery banding
   Systemic-pulmonary artery shunt

2. Enhancement of interatrial mixing of blood

Corrective Surgery

Haemodynamic correction ± anatomical correction

Corrective surgery should be performed when

1. No palliation is available but satisfactory correction is possible
2. Morbidity and mortality of primary correction equal or lower than palliation ± secondary correction
3. Palliation is unsatisfactory but correction is a probability

Surgical Technique

1. Closed heart
2. Open heart

Cardiopulmonary Bypass (Extracorporeal Circulation)

Pump

- Pulsatile flow
- Non-pulsatile flow

Oxygenator

- Film
- Bubble
- Membrane

Extracorporeal Circulation Circuit

SVC + IVC return
Cardiotomy

Aorta
Femoral artery

Oxygenator
Heat exchanger

Pump

Intraoperative Myocardial Preservation

Cold cardioplegia

Complications of Extracorporeal Circulation

1. Embolisation - air, particle, blood
2. Toxins from extracorporeal apparatus
3. Haemolysis
4. Toxins due to protein denaturation caused by mechanical damage
5. Hypotension
6. Biochemical disturbances
7. Abnormal blood gases
8. Brain damage

MANAGEMENT OF BURNS

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° and 3° 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 - (1) 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)

(2) Resucitation with crystalloid in first 24 hours.

Baxter and Shives Formula

Fluid for 1st 24 hours =
4 ml Ringer x Body weight in Kg x % burn

Fluid for 2nd 24 hours =
ml colloid x 0.3 x Body weight in kg
x % burn + D5 (D5 amount is half amount of Ringer given in 1st 24 hours)

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

b. Maintenance

Amount = evaporative loss + maintenance + other loss (e.g. Ryle's tube output)

Evaporate loss = (25 + % burn) ml/hour

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 graft
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

PLASTIC AND RECONSTRUCTIVE SURGERY

Latin "plasticus"
Greek "plastikos" = moulded or formed

Plastic surgery refers to that branch of surgery that employs various techniques to mould or shape tissue, particularly for the renewal of destroyed or injured tissue.

In a practical sense, the speciality deals with the correction of congenital and acquired external visible defects. Plastic surgeons aim to improve function or the appearance or both. The boundary lines of the speciality are vague because deformities, either congenital or acquired can affect any part of the body.

In his work "On the Parts of Animals" Aristotle wrote, "Art, indeed, consists in the conception of the result to be produced before its realization in the material."

"An artist, therefore, must not only be able to conceive the end result to be produced, but he must also be able to visualize all the necessary steps leading to that end, and he must have the imagination, the intelligence and the dexterity to bring about that result. Is not, then, plastic surgery an art and the plastic surgeon an artist? The plastic surgeon works with living flesh as his clay, and his work of art is the attempted achievement of normalcy in appearance and function. He starts with a deformity, whether discovered at birth or acquired from disease, injury or from an operation performed by the surgeon himself to overcome infection or malignancy. He uses skin, fat, bone, cartilage, muscle, fascia and tendon in building up the parts. He must exert his imagination in order to see what can be used and in what way. He must know and be able to modify the mechanisms and techniques that will bring this material in to build up the part, and yet keep the tissue alive. The principles of handling living tissues must be known and observed. Living parts have a superabundance of vitality, but if too great a burden is put upon them, they cannot survive or be used. Death of tissue may be a temporary setback or even a final defeat. Imagination must be tempered by the limitations of practicality, for care must be exercised to avoid making the original deformity worse or creating a new unjustifiable deformity elsewhere in the attempt at reconstruction."

- Jerome P. Webster in
"The Principles and Art of Plastic Surgery"

Plastic surgery is concerned with the following main areas:

1. Cleft lip and palate
2. Haemangioma, lymphangioma, naevi
3. Urogenital abnormalities e.g., hypospadias
4. Maxillo-facial Trauma
5. Head and neck malignancy in particular reconstructive surgery of the face, oral cavity, jaws
6. Craniofacial surgery
7. Aesthetic/cosmetic surgery - the ageing face, eyelids, nose, breasts, trunk
8. Burns and their sequelae
9. Hand surgery
10. Cutaneous malignancy
11. Reconstruction of skin defects of the trunk, lower limbs
12. Microneuro-vascular surgery

The scope of plastic surgery will be illustrated with clinical examples. The aphorisms of Gillies and Millard will be used to illustrate basic principles:

1. Observation is the basis of surgical diagnosis.
2. Diagnose before you treat.
3. Make a plan, and a pattern for this plan.
4. Make a record.
5. Prepare a lifeboat.
6. A good style will get you through.
7. Replace what is normal in its normal position and retain it there.
8. Treat the primary defect first.
9. Losses must be replaced in kind.
10. Never throw anything away.
11. Never let routine methods become your master.
12. Consult other specialists.
13. Speed in surgery consists of not doing the same thing twice.
14. The after-care is as important as the planning.
15. Never do today what can honourably be put off until tomorrow

Treatment:
1. Anti inflammatory drugs
2. Surgery for copious & persistent discharge - excision of the major ducts
3. Any associated peri areolar lump must be biopsied

Galactorrhoea

1. Drugs - Phenothiazine, oral contraceptives, reserpine, methyl dopa, haloperidol, tricyclic antidepressants
2. Hypothalamic disease and tumours of pituitary stalk - tumours, granulomas, meningitis
3. Pituitary tumour - non-functioning tumours, acromegaly Cushing's disease
4. Ectopic prolactin secretion - lung tumours
5. Primary hypothyroidism
6. Chest wall injury - trauma, surgery, herpes zoster

Galactocoele

1. Occurs only during lactation
2. Solitary cyst containiing milk (liquid or inspissated)
3. Majority can be treated by aspiration

Haematoma of Breast

1. Spontaneously or after trauma
2. Anticoagulant therapy

Treatment: Support breast, stop anticoagulants, & occasionally aspirate

Benign Cyst of Montgomery's Gland

1. Blocked gland (excision)
2. Usually superadded infection (incision of abscess)

Gynaecomastia - Unilateral or bilateral

1. Idiopathic
2. Drugs - digitalis, spirono lactone, INAH, steroids phenothiazines, amphetamines, androgens
3. Puberty - increased pituitary secretion or increased sensitivity to normal levels of testicular or adrenal steroids
4. Hormones - oestrogen, teratoma or chorion epithelioma of testis, Sertoli cell tumour of testis, adrenal carcinoma
5. Cirrhosis of liver
6. Testicular failure - Klinefelter's syndrome, testicular agenesis, bilateral cryptorchidism, severe bilateral orchitis
7. "Re-feeding" - post-inanition, treated C.H.F., treated pulm. T.B., chronic renal failure on dialysis
8. Non-endocrine tumours - bronchogenic CA, renal ca, Hodgkin's disease
9. Endocrine disorders - hyperthyroidism, hypothyroidism, acromegaly, diabetes mellitus, Addison's disease, pituitary tumours

Treatment: Treat cause; if idiopathic give danocrine 200 mg daily or tamoxifen 10 mg bid.
Excision of hypertrophied tissue may be also done

Traumatic Fat Necrosis

Can be confused with breast cancer. Usually seen in fat pendulous breasts with a history of trauma and bruising. Mammographic appearance can resemble carcinoma. Occusionally can be caused by injections into breast. Excisional biopsy will confirm diagnosis.

Paraffinoma of Breast

This is a sequel to injection of paraffin into a breast. Several years later a hard mass indistinguishable from breast cancer develops. The bilateral nature of the condition is a clue to its diagnosis.

SURGERY OF C.N.S.

Neurological diseases are very common and account for nearly 10% of all patients seen in general medical and surgical outpatients. Apart from head injuries, the commonest neurosurgical problem is the brain tumour, which may be primary or secondary.

A. GENERAL SURVEY OF S.O.L.

Classification of tumours depends upon the tissue of their origin. The best statistics that are so far available are those of Zulch (1965) who analysed 6,000 cases. The following percentages were worked out:

Glioma	42.0%
Meningioma	18.0%
Pituitary adenoma	8.0%
Acoustic neuroma	7.6%
Blood vessel tumours	3.8%
Congenital S.O.L.	5.5%
Metastatic	4.0%
Granulomas	0.7%
Miscellaneous	10.4%

Diagnosis

As the skull is a rigid box, when the tumour starts to grow it compresses the brain, thus producing signs of raised intracranial pressure (I.C.P.). Clinical examination therefore lends considerable help towards the diagnosis. The symptoms and signs may be classified as:

1. Increased I.C.P. Due to increased I.C.P. there are symptoms/signs that are well known to all. These are:-

   - Headaches, usually worse first thing in the morning, increasing by straining, coughing, defaecating, etc.

   - Vomiting, usually projectile and comes without warning. During later stages patient may refuse to eat.

   - Papilloedema. This is more marked with posterior fossa tumours and may be associated with haemorrhages in the fundus. It may lead to visual impairment.

   - Dizziness, nausea, etc.

2. Abnormal Neuronal Activity. The tumour may irritate the neurones and cause them to discharge in abnormal way giving rise to epilepsy. Depending upon the site and the lobe of the brain involved, epilepsy may be :

   - Grand mal type
   - Temporal lobe, with typical aura
   - Psychomotor
   - Jacksonian

3. Progressive Neuronal Paralysis. As the tumour increases in size, it may damage the part of the brain in its vicinity producing paralysis of the opposite side of the body. Depending upon the brain damage or compression, the patient may have:

   - Monoparesis or plegia
   - Hemiparesis or plegia

   The cranial nerves commonly involved are the II (pap.) VI, VII, VIII and for posterior fossa tumours the IX - XII. During late stages when brain herniation starts the III nerve gets involved. If a patient has an olfactory groove meningioma he classically has anosmia. For pituitary tumours remember the Foster-Kennedy syndrome.

4. Systemic Disturbances. One example is a pituitary tumour producing excessive growth hormone leading to gigantism in the young or to acromegaly. Similarly, if there is hypopituitarism the patient may have loss of body hair (pubic and axillary), thin shining skin etc.

   Besides these four groups, the patient may present with various syndromes that go with specific parts of the brain e.g., frontal lobe - personality change; posterior frontal lobe - hemiparesis; occipital lobes - hemianopia; pituitary SOL - bitemporal hemianopia; left temporal lobe - dysphasia; left parietal lobe - Gertsmann syndrome; basal ganglia - tremor and rigidity; cerebellum - ataxia, etc., etc.

Diagnostic Measures

1. X-ray-Skull. Usually obtained as A-P, lateral, and Towne's view may show beaten silver appearance (normal in children), erosion of lamina dura, calcification or erosion of skull, etc.

2. E.E.G.. In these days its role lies in diagnosing epilepsy only.

3. Echoencephalography. Again this is being performed less frequently. Also, in the best hands, its efficacy in diagnosing a lesion is only up to 80%. It has nearly 20% false positive or false negative rate i.e., it is not reliable.

4. Isotope Brain Scan. Usually technetium isotope is used. It gives positive result up to 90% in brain abscess and meningiomas but only 60% or so in glioma and still less in cystic, low-grade gliomas.

5. Angiogram. This is still one of the important investigations and will remain so for cerebral vascular lesions e.g., aneurysms, angiomas, blocked blood vessels etc.

6. Ventriculogram. Now rarely performed because it is a traumatic investigation. Some surgeons still use it for posterior fossa tumours.

7. Lumbar Air Encephalogram (L.A.E.G.). Now rarely performed. Its place has been taken over by CT scan.

8. CT Scan. This is now the investigation of choice.

VARIOUS BRAIN TUMOURS

1. Gliomas

Gliomas are classified according to their cell of origin i.e.,

- Astrocytoma
- Oligodendroglioma
- Ependymoma

Others

- Medulloblastoma

(a) Astrocytomas. These are usually divided into 4 grades (KERNOHAN). Grade 1 and 2 are relatively slow growing and grade 3 and 4 are very malignant and may be called glioblastoma.

Astrocytoma may originate from:

- Cerebrum
- Cerebellum
- Optic nerves
- Spinal cord

Grade 1 and 2 astrocytomas may originate at any of these sites and occur usually in the young i.e., 30-40 years (cerebrum) and 5-10 years (cerebellum). These are relatively avascular, there is no capsule and these infiltrate the brain. Consistency is usually firm to rubbery and some 16% of the cases have some calcification. Nearly 50% of the cases have fairly large cyst containing high protein, and xanthochromatic fluid.

Grade 3 and 4 gliomas are very malignant and few patients survive more than 2 years; mostly they die during the first year. They occur commonly between 40 and 60 years of age and the symptoms have been present only for a few months. These tumours have microcysts, many of them with thrombosed blood vessels. These can never be totally excised, in contrast to some slow growing grade 1 astrocytomas seen in children involving the cerebellum or occasionally the optic nerves.

(b) Oligodendrogliomas. These occur in adults between 30 and 50 years, usually in frontal lobes, symptoms may be present for months to years. Calcification in the tumour is seen in 40% of the cases and often appear, erroneously encapsulated. Appear greyish-pink on cut section and consistency varies. Spontaneous haemorrhages in the tumour are common. Secondary changes may be mucoid, calcification or the tumour may become mixed in character.

(c) Ependymomas. These originate from the ependymal lining. These are quite common in children under the age of 5 years; nearly 50% of the cases are seen under the age of 15 years and are more often seen in the cerebellum. In adults, however, the cerebrum is more often involved.

(d) Medulloblastomas. These are highly malignant and are usually seen in children involving the 4th ventricle of the cerebellum. Peak age incidence is 5 years, usually in the midline and fills the 4th ventricle. Consistency is soft, very poorly defined, very vascular, difficult to control bleeding during surgery. Highly cellular with abundant mitotic figures and Rosetts. Also, it spreads along the CSF pathways i.e., it may gravitate and start growing at the sacral end of the subarachnoid space. Prognosis is poor.

Treatment of Gliomas

Since these are space-occupying lesions they must be removed. The principle is to excise, as much as you can, safely without producing neurological deficit. Then give radiation and chemotherapy (CCNU/BCNU). Grade 1 gliomas in childhood, especially of the cerebellum can be totally excised, the child may be cured. Glioblastoma carries bad prognosis despite any form of treatment.

2. Meningiomas

This name was coined by Cushing. These originate from the endothelial cell lining the leptomeningeal spaces or the lumps of rest cells. They occur in middle age, > in females, forms rounded, lobulated mass, well demarcated and encapsulated, attached to the dura, may have calcification, very vascular and blood supply comes from the external carotid system, and classically are seen along the site of arachnoid granulations. These are classified as : parasagittal, falcine, convexity, sphenoid ridge, olfactory groove, suprasellar or posterior fossa meningiomas. Microscopically these may be:-

Syncytial
Transitional
Fibrous
Angioblastic
Malignant (only 5%)

Once diagnosed, these must be excised. If totally removed the patient is cured.

BRAIN ABSCESS

Brain abscess is usually secondary to infection somewhere and the commonest sites are:-

Otogenic
Sinusitis
Haematogenous

Common bacteria seen are:-

Staph. aureus
Streptococci
Pneumococci
Proteus
Haemophilus
E. coli

Common sites are (in order):-

Frontal lobe
Temporal lobe
Parietal lobe
Cerebellum

The abscess usually starts as a localised area of encephalitis in the white matter which in a few days leads to the formation of frank pus, and in about a week's time becomes encapsulated.

Signs and Symptoms

Would be S/S of raised ICP, focal neuronal stimulation and deficit.

Investigations

X-ray skull, CT scan; isotope scan.

Treatment

Heavy doses of antibiotics e.g.,

Ampicillin 4 G Q 4 - 6 H
+Cloxacillin 4 G Q 4 - 6 H I.V.

When capsule has formed the treatment is burr hole and aspiration of pus under antibiotic cover or excision of abscess wall as primary or secondary stage. The prognosis is good if diagnosed and treated early.

CEREBRAL ANEURYSMS

These are usually berry aneurysms and their incidence is 15.7 per 100,000 people (HELSINKI) and occur at the circle of Willis:

Anterior communicating artery	(ACA)	28%
Posterior communicating artery	(PCA)	25%
Middle communicating artery	(MCA)	20%
Anterior cerebral Internal carotid]		17%
Basilar system		10%

Usually occur between 40 and 60 years of age, 10% never rupture. Unruptured aneurysms are almost twice common in females, so are the internal carotid aneurysms. ACA are > common in males. These arise mainly at circle of Willis and at bifurcation of an artery. These may be saccular, fusiform, mycotic or fistulous in type.

Aetiology is unknown. These may be congenital or atheromatous. May be associated with local stress, hypertension or physical activity. Symptoms and signs are:-

Sudden onset of headache
Vomiting
Unconsciousness
Fever +
Meningism +
B.P. elevated
Decerebrate rigidity
Focal signs
Fundal haemorrhages

If untreated, 50% patients die within 2 weeks, 55% die within 6 weeks; only 34% survive 3 years.

Investigations

Lumbar puncture (L.P.)
Urine - glycosuria
Carotid angiogram
Vertebral angiogram
CT scan

Treatment

(a) Conservative, if the patient is

Old (> 70 years)
Debilitated
Cardiac disease
Severe atherosclerosis
Severe disease e.g., hypertension, diabetes mellitus

(b) Operative

Carotid ligation
Direct attack

Usually the patient is first stabilised, investigated and started on steroids and antifibrinolytic agent therapy to prevent bleeding. Patient is very unstable during the first week and they have lots of B.V. spasm and thus not a good risk patient. However, the spasm wears off in about 1-2 week and then you perform a direct attack. Carotid ligation is good for P.C.A. or aneurysms of I.C.A.

B. PRINCIPLES OF POSTOPERATIVE MANAGEMENT IN NEUROSURGERY

1. Clinical observations. The so-called vital signs are recorded every 15 minutes, 30 minutes or every hour. These signs must be observed and recorded carefully because they can alter the line of management or the outcome. These signs are:-

- Level of consciousness
- Pulse rate
- B.P.
- Temperature
- Pupils

Along with these also observe any early signs of deterioration and limb movements.

The commonly employed method to assess level of consciousness in these days is the GLASGOW coma scale which comprises:

Eyes Open

Spontaneously
To speech
To pain
None

Best Verbal Response

Orientated
Confused
Inappropriate words
Incomprehensible sounds
None

Best Motor Response

Obeys commands
Localises pain
Flexion to pain
Extension to pain
None

2. Headache. It is common following operations on the skull. Usually no analgesics are given as they may mask the symptoms. If pain is very severe then the best drug is probably Codeine Phos. 60 mg i/m Q6H.

3. Cerebral Oedema. It is very common following operations on the brain. The treatment is as discussed before. Steroids are usually given with cimetidine or antacids.

4. Fluid and Electrolytes. It is very important not to overhydrate the patient because that will only increase cerebral oedema. Also a meticulous intake/output chart is maintained, and vomitus, gastric aspirate, temperature, CSF leak and drainage etc. are considered. Usually we give 2L of dextrosaline/24 hours.

5. Nutrition. This becomes a problem only if the patient is comatose and remains so for longer than 48 hours. Usual requirements are 2000-2500 Cals/day but following operations or trauma it is increased by 10-50%. Normal daily protein requirements are 65G/day and a minimum of 1000 Cals/day are required to prevent negative nitrogen balance. The feeds must be properly balanced and contain vitamins C, K and B12.

6. Coma Management. These patients require 'total care', i.e.:

    Respiration
        position
        tracheostomy/airway
    Observations
    Fluid and electrolyte balance
    Nutrition
    Elimination
        bladder
        rectum
    Personal hygiene, etc.

7. Postoperative Complications.

    The commonest ones are:

    Postoperative cerebral oedema
    Intracranial haematoma
    Hydrocephalus
    Epilepsy
    Infections
    CSF fistulas
    Aseptic meningitis

AMBULATORY SURGERY

Ambulatory Surgery or Day Surgery is an elective surgery, performed on a fit patient who is admitted and discharged on the same day.

Recent advances in anaesthetic agents with rapid recovery from their use, enables the provision of general anaesthesia in a wide range of general surgical, gynaecological, urological, ophthalmological and plastic surgical procedures, performed as a day surgery patients.

I. List of General Surgical Procedures :
(excluding gynaecological, ophthalmological and plastic)

* Inguinal/femoral hernia repair
* Circumcision
* Excision of medium to large-size lumps (e.g. lipoma, bursa)
* Diagnostic cystoscopy, laparoscopy
* Varicose veins - ligation
* Excision of breast lump
* Anal fissure - excision or anal dilatation
* Laparoscopic cholecystectomy (in selected patients)

II. Advantages :

1. Minimal disruption to patient's normal activities
2. Less separation from home and family - an important consideration for children
3. Ability to return to work early
4. Reduction in incidence of hospital-acquired infection
5. Reduced cost in comparison with inpatient surgery

III. Selection Criterion :

* Patient should be within defined age limits (usually from 6 months to 65 years)
* The patient must be fit and healthy, ASA 1 or 2
* The patient must live within 1 hour's drive from hospital
* The patient must have access to a responsible adult who will assist him/her at least until 24 hours after operation

IV. Guidelines for the Surgery :

* Exclude procedures where severe post-operative pain and significant bleeding is likely
* The procedure should not be expected to exceed 30-60 minutes in length
* Exclude patients who are obese, diabetics, or have chronic cardiovascular and respiratory disease

V. Facilities :

1. A Hospital-Integrated Unit or
2. An Independent Day Surgery Unit

VI. Pre-operative Preparation :

* Written consent
* Clear instructions to patient about
  - fasting
  - time of arrival at the day ward
  - being accompanied
  - transport to and from hospital
  - drug requirements

VII. Anaesthesia :

* Local
* Regional
* General

In most cases, routine premedication is not required.
Epidural or intrathecal analgesia is usually considered inappropriate for day cases.

VIII. Post-operative Care :

* Post-operative recovery should be rapid
* Immediate recovery can be assessed on five factors - Activity, Respiration, Circulation, Consciousness and Colour

IX. Discharge :

* Adequate post-operative analgesia must be provided for all patients
* Information about analgesia must be given to the patient and to whoever is caring for them in the post-operative period
* The facility for overnight admission of any patient not considered well enough for discharge must exist. This may be required in about 2-4% of patients