UNIVERSITY OF HONG KONG

Department of Surgery

LECTURES FOR MEDICAL STUDENTS

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CONTENTS

PAEDIATRIC SURGERY

NEONATAL ABDOMINAL EMERGENCIES

GASTROINTESTINAL PERFORATIONS

Gastric Perfortion

Aetiology of G.I. Perforations

- Ischaemia secondary to asphyxia, stress, shock, hypoxia
- Second stage labour and associated stress

(No history of stress, hypoxia or shock in 20% of infants)

Incidence

1/10,000-15,000 live births

Sites of G.I. Perforations

Gastric
Duodenal
Jejuno-ileal
Colonic

History and Findings

Prematurity	50%
Asphyxia, shock	80%
Single:Multiple	3:1
Average duration, after stress	2.4 days

Diagnosis

1. History
2. Physical examination
3. X-ray abdomen - supine
   - upright, or lateral decubitus

Treatment

1. Active resuscitation with fluids, electrolytes, antibiotics
2. Surgical exploration for resection/respair/anastomosis/ enterostomies
   (For gastric perforation, conserve as much of stomach as possible)

Necrotising Enterocolitis (NEC)

The following features and findings should alert the clinican to the diagnosis of NEC :

Premature infants
Abdominal distension
Bilious vomiting
Gastrointestinal bleeding
Ischaemic gangrene of intestine
Perforation
Peritonitis
X-ray: Pneumatosis intestinalis

Aetiology

(a) Ischaemic Damage to Intestine

Vasospasm
- ischaemia and shock ——> "diving reflex"
- catheterisation of umbilical artery
- infusion of calcium containing solutions

Thrombosis
- indwelling catheters in aorta
- hyperviscosity states

Low flow states
- congenital heart disease
- deep hypothermia and circulatory arrest
- shock

(b) Bacterial Colonisation

E.coli, Klebsiella, Salmonella, Clostridium, Staphylococcus

(c) Substrate - Formula Feeding

Hyperosmolar feeds to small infants ——> NEC
Hypertonic goat milk to newborn goats ——> NEC
Breast milk contain IgA - protects intestinal mucosa

Diagnosis

Clinical : Distension
G.I. bleeding
Lethargy
Gastric retention
Vomiting and regurgitation
Temperature instability
Apnoeic spells
Pneumatosis intestinalis (palpable)

Radiological : Pneumatosis intestinalis (radiological)
Portal venous gas
Pneumoperitoneum

4HM5

Indications for Surgery

Absolute : free perforation
gangrene

Good : persistent abdominal tenderness
erythema
inflammatory mass
persistent dilated loop

Controversial : severe G.I. haemorrhage
clinical deterioration
- acidosis
- shock
- apnoea
- hyponatraemia
- gasless abdomen

persistent thrombocytopenia
disseminated intravascular coagulation (DIC)

Contraindications : Pneumatosis intestinalis per se
Portal venous gas per se

Treatment

Conservative : Nil by mouth
N/G suction hourly and free drainage
N/G instillation of aminoglycosides (controversial)
I.V. fluids
I.V. antibiotics
Repeated abdominal examination by same person
X-ray abdomen at least 3 times in 24 hours
Blood gases, Na^+, K^+, electrolytes
Septic work-up
CBP, Platelets

Surgical : (a) Resection of gangrenous bowel
Faecal diversion

(b) Re-anastomosis later ± resection
of stenotic bowel

GASTROINTESTINAL BLEEDING

Causes : Swallowed maternal blood
Duodenal ulcer
Intussusception
Meckel's diverticulum
Tubular duplications
Volvulus neonatorum
Necrotising enterocolitis

Bleeding from the Alimentary Tract

1. Presenting symptom : haematemesis

Age of Patients	Amount of Bleed	Possible Cause	Colour
Neonates	Small	Pyloric stenosis	Dark
		Reflux oesophagitis	Dark
	Large	Peptic ulcer (rare)	Bright
Older children	Large	Oesophageal varices	Bright
		Acute erosions	Bright
		Stress ulcers	Bright
		Peptic ulcer	Bright

2. Presenting symptom : rectal bleeding or melaena

Age of Patients	Amount of Bleed	Possible Cause	Colour
Infants	Small	Intussusception	Bright
		Volvulus	Dark
	Large	Tubular duplications	Bright
Toddlers	Small	Anal fissure or prolapse	Bright
Older children	Small	Polyps	Bright or Dark
	Large	Ulcerated Meckel's	Bright
		Oesophageal varices	or Dark
		Stress ulcers	

Volvulus Neonatorum

The return of foetal alimentary canal from the extra-embryonic coelome into the abdomen occurs during the fourth week of intra-uterine life, and the bowel then undergoes rotation and fixation at certain points by the attachment of its mesentery to the posterior abdominal wall.

When this process is incomplete or deviates from normal, it results in malfixation or malrotation.

Obstruction occurs in two ways:

1. Narrow base of attachment of small intestinal mesentery allows volvulus around the axis of the "universal mesentery", ——> strangulation, obstruction.

2. As caecum is wound tight, the Ladd's peritoneal bands obstruct second part of the duodenum.

Features

- No obstruction in first few days possible
- Meconium may be passed
- Then sudden obstruction occurs
- Obstruction may subside
- May recur in some days or weeks

Signs

- Shock, pallor
- Blood or blood-stained stools
- Vague central mass
- Distension variable

Investigations

1. Plain X-ray - double bubble; some fluid levels
2. Barium enema - subhepatic caecum
3. Contrast meal - duodenal obstruction and abnormal position of D-J junction

Treatment

Surgical

(a) Untwist volvulus, divide Ladd's bands and bring caecum to LIF
(b) Resection and anastomosis when necessary

"NEONATAL ASCITES"

1. **Urinary "ascites"** : obstructive uropathies e.g., pelvi-ureteric junction obstruction posterior urethral valves

2. **Bile "ascites"** : duct abnormalities

Diagnosis

Awareness
Physical examination
Paracentesis
Intravenous urography
Cystourethrogram

Treatment

Surgical

(a) Urinary diversion and treatment of cause later for urinary ascites
(b) Cholecystostomy, peritoneal drainage for bile ascites

SURGERY OF CONGENITAL ANOMALIES

OESOPHAGEAL ATRESIA

Oesophageal atresia is a congenital anomaly in which there is complete interruption of the lumen of the oesophagus in the form of a blind upper pouch, generally associated with a tracheo-oesophageal fistula. Though there are many variations, only the commonest type will be discussed.

Early Diagnosis

The most important aspect of oesophageal atresia is that it should be recognised as soon after birth as possible, for any delay inevitably leads to progressive pulmonary complications.

The chances of successful surgical treatment are to a large extent directly related to the length of time between birth and diagnosis.

Important Points for Early Diagnosis

1. Maternal hydramnios - present in about 60% of cases

2. Features after birth before feeding
   - Coughing)
   - Choking) = "3 Cs" plus Froth
   - Cyanosis)

3. On feeding - aggravation of "3 Cs"

Diagnosis

1. Firm catheter passed down oesophagus. If arrested, diagnosis is established.
2. Percuss abdomen. If resonant, tracheo-oesophageal fistula (TOF) is present. Dullness suggests absence of TOF.
3. X-ray chest and abdomen with catheter in upper pouch to see
   - level of atresia
   - state of lungs
   - presence of thoracic skeletal anomalies
   - air in stomach confirms presence of TOF

Treatment

1. Primary anastomosis
2. Delayed primary anastomosis
3. Staged repair
   (a) oesophagostomy, thoracotomy, division of fistula, gastrostomy
   (b) colonic or stomach tube reconstruction

DIAPHRAGMATIC HERNIA

1. Congenital - posterolateral hernias (Bochdalek type)
   - anterolateral hernias (Morgagni type)

2. Acquired - traumatic rupture
   - operative damage

Development of Diaphragm

1. The pleuroperitoneal membrane
2. The septum transversum
3. Marginal ingrowths from the muscle wall.

Congenital diaphragmatic hernias - result from failure of formation of part of the diaphragm, failure of fusion of one part with another, or failure of its muscular components to form.

Whereas failures of formation or fusion result in a defect and a hernia, failure of "muscularisation" produces a thin, weak diaphragm with an upward bulge of part or all of one or other leaf. This latter form is referred to as eventration.

The common left-sided Bochdalek hernia will be discussed.

Presentation

Respiratory distress in the newborn

Physical Findings

1. Respiratory distress or cyanosis
2. Apparent dextrocardia
3. Small and somewhat scaphoid abdomen
4. Diminished air entry on affected chest
5. Intrathoracic borborygmi
6. "Ballooned" chest

Diagnosis

1. Plain X-ray of chest and abdomen
2. Contrast upper G.I. study (very occasionally)

Treatment

1. Trans-abdominal reduction of hernia contents and repair of Bochdalek hernia
2. Ladd's procedure for malrotation
3. Abdominoplasty if necessary with silastic sheet
4. Postoperative ventilation is usually required.

ANTERIOR ABDOMINAL WALL DEFECTS

Exomphalos

Rare, serious abnormality of the umbilicus.
Large congenital hernia into the base of the umbilical cord.

Covering

Translucent membrane formed by fused layers of amniotic membrane and peritoneum

Contents

Loops of small and large bowel
Liver - quite commonly in defects > 5 cm diameter

Diagnosis

Obvious on inspection

Coexistent Malformation

Cardiac anomalies
Malrotation of gut

Investigations

X-ray chest to
- detect cardiac anomalies
- atelectasis of lungs

Aims of Treatment

To provide a cover of skin as soon as possible
Not to embarrass respiration by above procedure

Treatment

Depends upon
- size and condition of the infant
- presence of other anomalies
- size of defect and capacity of sac
- presence or absence of rupture
- presence or absence of intestinal obstruction

1. Immediate operation and complete repair
2. Immediate operation to cover the sac with skin
3. Immediate replacement of the sac with silon -
4. Non-operative treatment with 2% aqueous mercurochrome

Gastroschisis

Small defect in anterior abdominal wall, usually just below, to the right of and completely separated from the umbilicus. There is no covering.

Treatment

Operation is done as soon after birth as possible, employing a modification of methods 2 or 3 described for exomphalos.

Extrophy of Cloaca

An anterior abdominal wall defect together with the failure of the formation of the uro-rectal septum results in this severe anomaly, which is extremely difficult to manage.

Treatment

1. Complex surgical procedures, which are usually staged, are employed
2. Primary repair may be attempted on occasions

NEONATAL INTESTINAL OBSTRUCTION

Duodenal Obstruction

1. Extrinsic obstruction - Peritoneal bands of Ladd
   - Volvulus neonatorum
   - Preduodenal portal vein

2. Intrinsic obstruction - Atresia
   - Stenosis
   - Membrane
   - Annular pancreas

Site

Most commonly second part of the duodenum

Chromosomal Disorder

Special correlation between Down's syndrome and atresia proximal to the ampulla of Vater is present

Clinical Features

Atresia - Signs of acute, gastric outlet obstruction

Stenosis - Slightly less severe features than above

Membrane - Signs of incomplete obstruction because the small hole in the septum initially permits the passage of air and some fluid. The diagnosis may be missed if symptoms are mild or transient in the early stages. The membrane is pushed onwards by peristalsis so that it may bulge far along the duodenum, stretching its mucosal attachment.

X-ray - Plain ——> "double bubble" is diagnostic of atresia
- Contrast meal and fluoroscopy may be needed for obstruction by membrane

Treatment

Atresia - Duodeno-duodenostomy or Duodeno-jejunostomy
Membrane - Duodenotomy and excision of membrane

Intestinal Obstruction

Aetiology

Interruption of mesenteric arcades by a vascular accident in utero

Sites

Ileum - most frequent
Colon - less frequent

Number

Usually one, sometimes multiple

Types I : Atresia with bowel in continuity

II : Atresia - Proximal bowel is connected to the distal bowel by a fibrous strand

III : Atresia - Discontinuity between two ends with an associated gap in the mesentery

IV : Multiple intestinal atresias

Gross Pathology

Proximal bowel
- distended
- hypertrophied
- balloned terminal bowel
- abnormal vascular pattern

Distal bowel
- unused and undistended
- micro-colon on contrast study

Clinical Features

Features of intestinal obstruction
Distension varies directly with distal obstruction
Vomiting varies directly with proximal obstruction
History of having passed meconium does not rule out atresia

Diagnosis

Plain X-rays of the abdomen
Barium enema p.r.n.
Contrast meal occasionally

Treatment

1. Resection and anastomosis for jejuno-ileal atresias
2. Proximal colostomy and reanastomosis later for colonic atresia.

Anorectal Anomalies

A perineum without an anal opening is traditionally described as "imperforate", a term which embraces a number of anomalies, and is incidentally inappropriate.

Classification

Development of the distal bowel is arrested at one of two levels, each with its own subtypes.

The principal distinction is in the relation of the end of bowel to the chief muscle of continence, the puborectalis component of levator ani.

Arrested development at or above this sling (the supralevator lesions) produces rectal deformities; arrested development below the sling (the translevator lesions) produces anal deformities. In each group the bowel may end blindly, or communicate by a fistula with a neighbouring viscus or the perineal skin.

Types

1. Rectal deformities (supralevator lesions)
2. Anal deformities (infralevator lesions)

Incidence

1:5,000 live births

Sex

Males - slight preponderance
- higher incidence of more difficult rectal deformities

Females - most are of anal type

Aetiology

Quite unknown
No exogenous factors in pregnancy have been identified
Evidence of genetic determinant is meagre
Rarely a subsequent sibling affected

Clinical Features

Intestinal obstruction is the presenting feature in most cases. However, in females the fistula to the genital tract is usually wide enough to decompress the bowel adequately. In males, fistula to the urinary tract may lead to the appearance of meconium in the urine, an important diagnostic observation.

The discovery of a fistula to the skin or even a minute orifice is proof that it is an anal type of anomaly, whereas a completely "blind" perineum may be due to either a rectal or anal anomaly, usually the former.

Diagnosis

1. Physical examination
2. X-rays
   (a) Spine for vertebral anomalies, especially sacral agenesis
   (b) Pelvis with child held upside down, in the exact lateral position i.e., an invertogram. "P.C. line" (pubo-coccygeal line) is the important landmark for supralevator or infra-levator types.
   (c) MCU for recto-urinary communications
   (d) IVP later to assess renal anomalies

Treatment

1. Anal deformities : Anoplasty

2. Rectal deformities:

   (a) Colostomy in neonatal age, followed by
   (b) Sacroperineal rectoplasty for intermediate lesions
   (c) Sacro-abdominoperineal rectoplasty for high lesions

(N.B.: Procedures (b) and (c) are performed at age of 10-15 mths)

Associated Anomalies

These are common and are present in 50-60% of cases. Anomalies include genitourinary, vertebral, alimentary, cardiac and the miscellaneous group of which CNS anomalies are the commonest.

HIRSCHSPRUNG'S DISEASE

Hirschsprung's disease is the commonest cause of intestinal obstruction in the newborn.

Incidence

1:5,000 births

Genetic Types

1. 'Short' segment - Commoner
   Involves sigmoid, rectum and anal canal
   Males:Females = 5:1

2. 'Long' segment - Higher degree of "penetrance"
   Males:Females = 1:1

Age

Mostly in infancy (70-80% in first few days)
Few in childhood
Rarely in adulthood

Clinical Features

Delayed passage of meconium beyond 24 hrs after birth
Picture of low intestinal obstruction with bilious vomiting
Abdominal distension
Constipation or diarrhoea

Rectal Examination

Empty rectum
Tight anal sphincter

Diagnosis

1. Clinical features
2. Barium enema without bowel preparation
3. Rectal biopsy
4. Manometry
5. Electromyography
6. Serum and erythrocyte acetyl cholinesterase activity

(Latter three investigations are adjuncts)

Treatment

1. An initial colostomy placed in normal bowel above the cone of transition, confirmed by frozen section biopsies
2. Rectosigmoidectomy at 9-20 months when the patient is in optimal condition
3. Closure of colostomy in about 4 weeks

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 30^0
- 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))	

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

SURGERY OF THE THYROID GLAND

Conditions presenting to the Surgeon

Non-toxic Nodular Goitre

Toxic Nodular Goitre

Non-toxic Diffuse Goitre

Graves Disease

Viral Thyroiditis (Sub acute thyroiditis)

Hashimoto's Disease (auto immune thyroiditis)

Neoplasms

Benign - non-toxic adenoma
- toxic adenoma

Malignant

Thyroid Carcinoma

(a) Well differentiated - papillary
- follicular

(b) Anaplastic

Medullary Carcinoma (from parafollicular C cell)

Lymphoma

Metastatic

Clinical Presentation

Symptoms

Local - neck swelling
pain
pressure symptoms
voice change

General

Features of toxicity
- palpitations
- heat intolerance
- tremor
- loss of weight with normal or increased appetite.

Eye symptoms

Signs

Local

Thyroid enlargement :
- diffuse
- multinodular
- clinically solitary nodule

Tenderness

Tracheal deviation

Retrosternal extension

Abnormal voice

General

Features of toxicity

(tachycardia, arrhythmias, high pulse pressure, sweating, tremor, hyper-reflexia)

Eye signs

(a) Sympathetic overaction
(lid lag and lid retraction)

(b) Infiltrative "auto-immune"
(proptosis, chemosis, periorbital swelling, occuloparesis)

Special Investigations

- Thyroid Function Tests
  (T_4, T_3 uptake, Free thyroxine index, TSH)
- Serum Calcium
- Calcitonin
- Antibodies
- Fine Needle Aspiration Cytology
- Isotope Scan
- X-ray Thoracic Inlet
- Ultrasound

Management

- Surgery
- Medication
- (T_4, Anti-thyroid drugs, Beta-blockers)
- Radioative iodine
- External source irradiation

Approach to the Common Surgical Problems

(1) Multinodular Goitre

Very common in females over 40
Frequently asymptomatic

Investigations

- Thyroid function tests
- X-ray thoracic inlet

Surgery

- Large glands with pressure symptoms retrosternal enlargement or trachael deviation
- Toxicity
- Cosmetic complaint

Observation only

- when the above indications are absent T_4 medication not often helpful in reducing gland size and therefore long term treatment with T_4 not indicated.

(2) Clinically Solitary or Dominant Nodule (C.S.N. or D.N.)

Occur in any age group.

Underlying pathology is:-

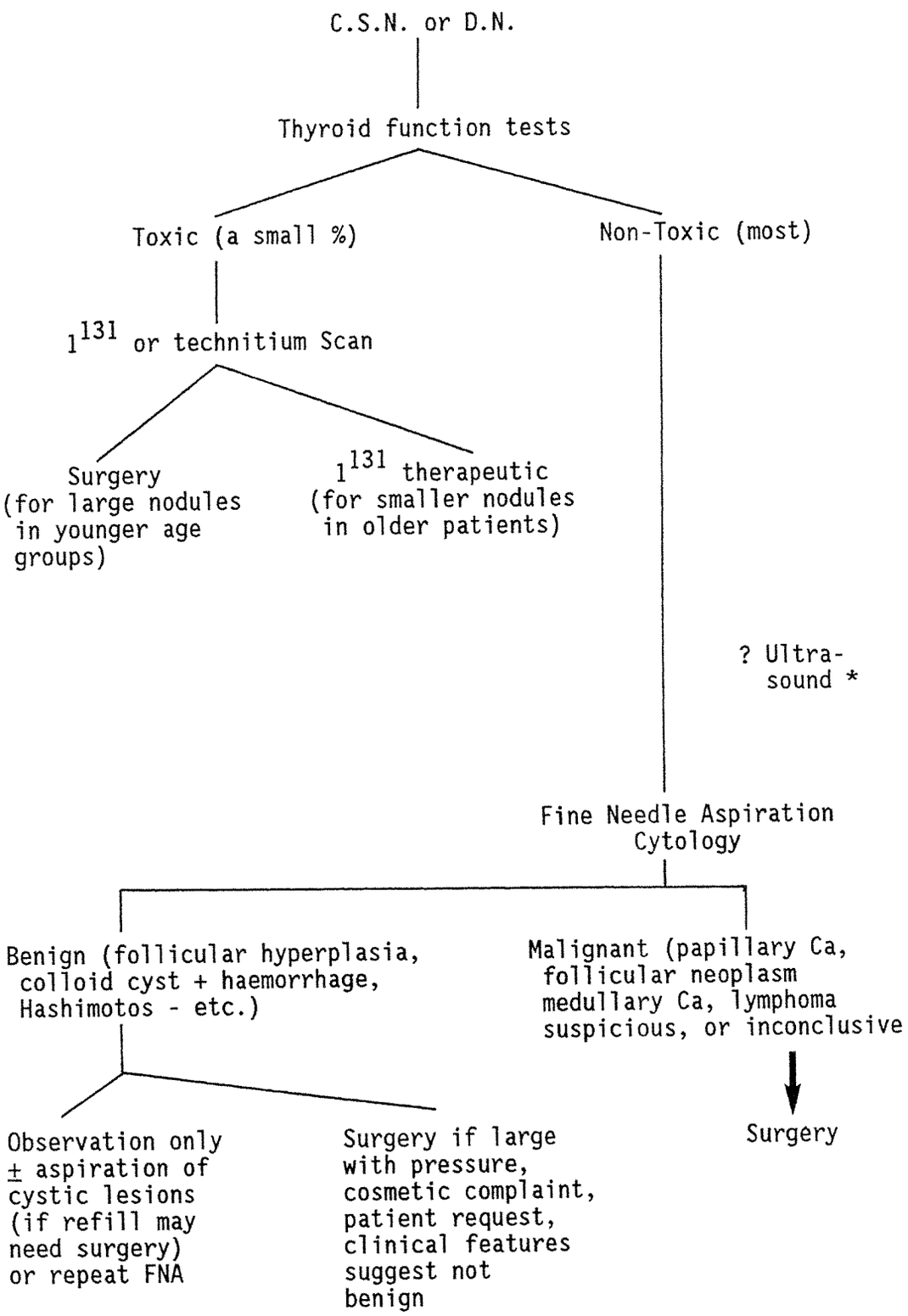
(a) An area of colloid cystic degeneration and haemorrhage as part of nodular goitre (± 70%)

(b) Benign follicular adenoma - mainly non-toxic with small number toxic (± 17%)

(c) Well differentiated thyroid carcinoma (± 8%)

(d) Miscellaneous - true congenital thyroid cyst, other malignancies, Hashimoto thyroiditis. (± 5%)

Management plan



* Ultra sound may be helpful in treating cystic lesions but is not essential.

(3) Graves Disease

Diffuse Goitre + hyperthyroidism of auto-immune origin due to Thyroid Stimulating Immune globulins and sometimes associated with infiltrative occulopathy and pre-tibial myxoedema.

Investigations

Thyroid Function Tests
Isotope scan used in planning treatment by I^{131}

Management

Depends on natural history

Type I - Often with less marked thyroid enlargement and characterised by return to euthyroidism spontaneously after 6-18 months. May recur after variable period. Best managed with antithyroid drugs unless persistent recurrences.

Type II - Often with large gland in younger age group (15-40 yrs) and florid hyperthyroidism which is persistent or progressive. Best managed by surgery for the larger glands in younger patients or radio-active iodine in smaller glands in older age group.

Type III - Gland size variable and progressing to hypothyroidism owing to replacement of thyroid functioning tissue by lymphocytes. Best managed by antithyroid drugs initially and later by T_4 replacement.

Because the natural history only becomes clear with time most patients are managed initially with antithyroid drugs for a period of 6-12 months.

(4) Thyroid Cancer

Papillary ($\pm$ 60%) and follicular ($\pm$ 20%) account for most thyroid malignancy seen clinically.

Less commonly seen

Anaplastic $\pm$ 5%

Medullary 2-5% - may be associated with M.E.N. II syndrome

Miscellaneous - lymphoma metastatic etc.

Investigation

Fine Needle Aspiration Cytology is the single most important investigation.

Management

For well diffentiated thyroid cancer (papillary and follicular) and medullary cancer the treatment will usually be total or modified total thyroidectomy.

In some this will be combined with lymph node surgery and with adjuvant radio-active iodine therapy.

For anaplastic carcinoma the main treatment is by external source irradiation and surgery is used only for bulk reduction or tracheostomy. The prognosis is very poor.

SURGERY OF OTHER ENDOCRINE GLANDS

I. THE ENDOCRINE SYSTEM

Types of Hormones

1. Polypeptides and proteins — e.g., trophic hormones, insulin
2. Steroids — e.g., cortisol, sex hormones
3. Low M.W. peptides and amines — e.g., thyroxine, catecholamines
4. 'Candidate' hormones (role not yet clearly defined) — e.g., histamine, prostaglandins, alimentary polypeptides.

Source of Hormones

1. Specialised cells grouped to form major constituent of a gland — e.g., thyroid, adrenal, parathyroid
2. Discrete clumps of cells in organs with other major functions — e.g., Islets of Langerhans in pancreas; Leydig cells in testis
3. Scattered singly among other types of cells — e.g., gut hormones
4. Formed in blood from precursors — e.g., kinin, angiotensin

The Neuroendocrine Cells (APUD System)

1. High AMINE content
2. Capacity for amine PRECURSOR UPTAKE
3. Presence of DECARBOXYLASE

Hypothalamus, pituitary, thyroid (parafollicular cells), adrenal medulla, mucosa of alimentary tract, pancreatic islets.

Endocrine Disorders

	Functional State	Treatment
Hyperfunction	Neoplasm Hyperplasia	Surgical removal Pharmacological manipulation
Hypofunction	Haemorrhage Infarction Infection Neoplasm Iatrogenic Congenital	Substitution therapy Injection of trophic hormones

II. THE PARATHYROID GLANDS

Anatomy of the Parathyroid Glands

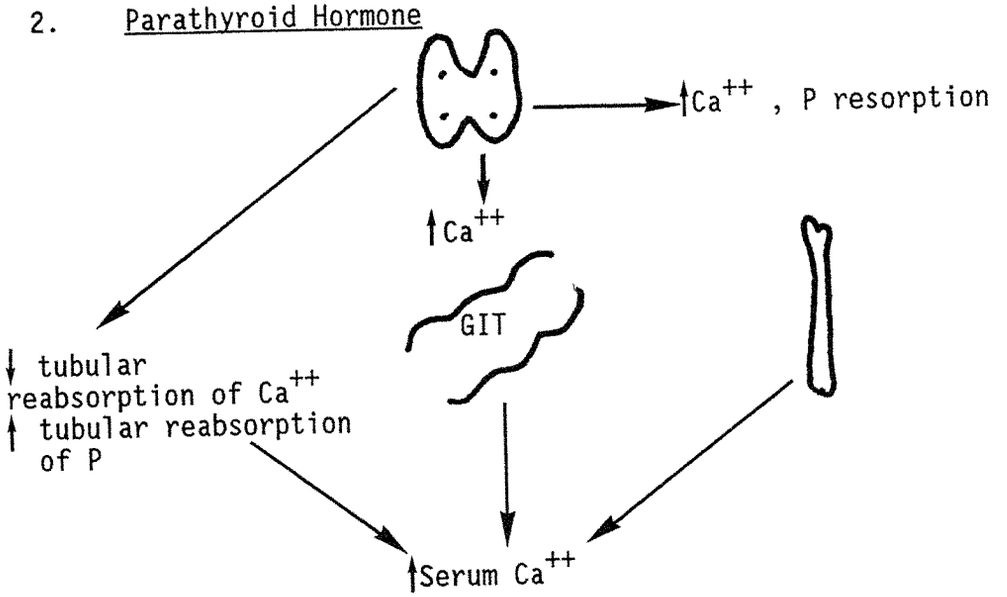
1. Ovoid, flattened, smooth, tan-coloured, 15-40 mg each
2. Superior parathyroids - 4th pharyngeal pouch
3. Inferior parathyroids - 3rd pharyngeal pouch

Regulations of Calcium Homeostasis

1. Vitamin D — 25.OH cholecalciferol (liver) — 1, 25 diOH cholecalciferol (kidney)

(Facilitate calcium absorption from GIT)

2. Parathyroid Hormone



3. Calcitonin - Inhibit Ca^{++} resorption from bone
4. Glucagon - Promote renal excretion of Ca^{++}
5. Sex steroids - Oestrogens, androgens partially reverse the effect of PTH on bone

Differential Diagnosis of Hypercalcaemia

1. Primary hyperparathyroidism
2. Vitamin D intoxication
3. Milk-alkali syndrome
4. Sarcoidosis
5. Multiple myeloma
6. Lymphoma
7. Paget's disease
8. Secondary carcinomatosis (breast, bronchus, thyroid, prostate)
9. Cushing's syndrome
10. Thyrotoxicosis
11. Ectopic PTH production - bronchogenic carcinoma, hypernephroma of kidney.
12. Prolonged immobilisation
13. Familial hypocalciuric hypercalcaemia

Primary Hyperparathyroidism

Clinical Features

1. Renal - recurrent renal stones
   nephrocalcinosis
2. Bone - osteopenia
   subperiosteal resorption
   osteitis fibrosa cystica
3. Symptoms of hypercalcaemia
   - fatigue, muscle weakness, constipation, thirst, polyuria.
4. Peptic ulcer
5. Psychiatric disorders
6. Incidental finding of hypercalcaemia.

Diagnosis

1. Clinical suspicion
2. Biochemical abnormality :
   serum Ca^{++} , p
   24 hr urine calcium
   mild hyperchloraemic metabolic acidosis
   A.P.
3. Radiological evidence
   - skull, long bones, clavicles, dental
4. Circulating PTH level

Pathology

1. Adenoma 80-85%
2. Hyperplasia 10-15%
3. Carcinoma 1-2%
4. M.E.N. I - PPP (parathyroid, pancreas, pituitary)
   II - TAP (thyroid, adrenal, parathyroid)

Management

1. Preoperative

   (a) Adequate hydration
   (b) Assess vocal cord function
   (c) Anatomical localization
       CT scan
       Ultrasound
       Thallium-technetium scan
       Venous sampling of neck veins

2. Operative

   (a) Gross identification
   (b) Blushing test
   (c) Methylene blue infusion
   (d) Density flotation test
   (e) Intracellular fat staining
   (f) Biopsy

3. Postoperative

   (a) Monitor serum calcium level
   (b) Transient hypocalcaemic phase

Problems in Parathyroid Surgery

1. Re-exploration after parathyroidectomy
2. Parathyroid autotransplantation

III. THE ADRENAL GLAND

Superior medial pole of each kidney at 11-12th rib 4-7 gm each

Hormone Production

1. Cortisol
2. Aldosterone
3. Catecholamines

A. CUSHING'S SYNDROME

Clinical Features

1. Truncal obesity
2. Muscle weakness
3. Abdominal striae
4. Easy bruising
5. Hypertension
6. Acne
7. Hirsutism
8. Diabetes mellitus
9. Psychosis

Causes

1. Adrenal tumour - adenoma
   carcinoma
2. Adrenal hyperplasia (Cushing's disease)
3. Ectopic ACTH syndrome - malignant tumours
   e.g., bronchogenic, thymic, pancreatic

Diagnosis

1. Clinical manifestations
2. Biochemical - high plasma cortisol level with loss of diurnal rhythm
   high 24 hour urine 17-hydroxycorticosterone
   17-ketosteroids
3. Pharmacological - dexamethasone suppression test
   low dose
   high dose
   metyrapone test

Treatment

Depends on cause:
1. Adrenal tumours - unilateral adrenalectomy
2. Adrenal hyperplasia - bilateral adrenalectomy
   pituitary irradiation
   transphenoidal pituitary surgery
3. Ectopic ACTH syndrome - excision of malignant source
   medical therapy e.g., metyrapone,
   aminoglutethimide, o.p.'D.D.D.

Pre- and Post-operative Treatment

1. Steroid cover - hydrocortisone 100 mg q6h ivi
   Steroid replacement - cortisone acetate 25 mg a.m.
   12.5 mg p.m.
   9 -fluorohydrocortisone
   0.05-.1 mg Q.D.
2. Correct electrolyte imbalance

B. HYPERALDOSTERONISM

Clinical Features

1. Hypertension
2. Hypokalaemia
3. Muscle weakness
4. Polydipsia, polyuria
5. Hypokalaemic alkalosis

Causes

1. Primary - adrenal adenoma 70%
   adrenal hyperplasia

2. Secondary - liver cirrhosis with ascites
   nephrotic syndrome
   congestive heart failure
   renal artery stenosis

Diagnosis

1. Clinical suspicion
2. 24 hours urine aldosterone
3. plasma aldosterone
4. plasma renin level

Treatment

1. Operative
2. Medical - spironolactone (aldosterone antagonist)

C. PHAEOCHROMOCYTOMA

Characteristics

1. Derived from adrenal medulla, sympathetic nerve endings
2. Secrete - epinephrine, non-epinephrine
3. 10% tumour - 10% bilateral
   10% extra-adrenal
   10% malignant

Clinical Manifestations

1. Persistent hypertension 70%
2. Paroxysmal hypertension 30%
   - headache, tachycardia, palpitation
     facial flushing, sweating, diarrhoea

Diagnosis

1. Clinical suspicion
2. Biochemical - urinary amines, VMA, ME and MNE, catecholamines
   plasma catecholamines
3. Pharmacological test - phentolamine 5-10 mg ivi
   BP 35/25 mmHg

4. 131-I-MIBG scan

Treatment

1. Excision of tumour
2. Preoperative - medical treatment - α or β blockers
   e.g., phenoxybenzamine
   propranolol
3. Intraoperative - hypertensive crisis - phentolamine
   - nitroprusside
   - explore both adrenals and para-aortic areas
4. Postoperative - hypovolaemia
   adrenal insufficiency
   hypotension and hypovolaemia - fluid, plasma

Anatomical Localisation

1. 131-I-MIBG scan + superimposed scanning of organs
   e.g. kidney, bone, liver
2. CT scan
3. Adrenal arteriogram
4. Adrenal venography + venous sampling

IV. ISLET CELL TUMOURS OF THE PANCREAS

Types

All Types	Secretion	Syndrome
	Glucagon	Hyperglycaemia
	Insulin	Hypoglycaemia
	Gastrin	Zollinger-Ellison
Non-beta		WDHA

Pathology

1. Solitary adenomas 90%
2. Multiple adenomatosis
3. Diffuse hyperplasia
4. Malignant

A. INSULINOMA

Diagnosis

1. Fasting hypoglycaemia
2. Plasma glucose < 2 mmol/L
3. Symptomatic relief by intravenous glucose (Whipple's triad)
4. Inappropriate elevation of circulating insulin

Anatomical Localisation

1. CT scan
2. Coeliac angiogram (Ditigal subtraction angiography, DSA)
3. Percutaneous venous sampling
4. Intraoperative ultrasound

Treatment

1. Surgical excision of tumour - enucleation, partial pancreatectomy

   (a) Preoperative - frequent feeds to reduce number of hypoglycaemic attacks
   (b) Intraoperative - monitor blood sugar level rise in blood glucose 30 mins after complete removal of hyperfunctioning tissue

2. Non-operative

   (a) Diazoxide (thiazide compound) - inhibit insulin secretion
   (b) Streptozotocin, Other chemotherapeutic agents e.g., 5-FU

B. GASTRINOMAS (ZOLLINGER-ELLISON SYNDROME)

Clinical Manifestations

1. Recurrent or atypically located peptic ulcer
2. Marked gastric acid hypersecretion
3. Watery diarrhoea

Diagnosis

1. Circulating basal gastrin > 200 pg/ml, secretin-stimulated gastrin
2. Intravenous calcium infusion test
   - marked increase in serum gastrin and gastric acid production

Treatment

1. Resection of tumour
2. Unresectable tumours
   - Vagotomy
     Total gastrectomy
     H2-receptor antagonist

V. PITUITARY

Types of Pituitary Tumours

1. Anterior
   - chromophobe adenomas (prolactinomas, M.E.N. I)
   - acidophil adenomas (acromegaly, prolactinomas)
   - basophil adenomas (Cushing's disease)
2. Posterior - rare
3. Craniopharyngiomas

Clinical Features

1. Local pressure - headache
   visual disturbance V.A. V.F.
   diabetes insipidus
   obesity
   sleep disturbance

2. Hormonal changes - trophic hormones
   prolactin
   GH

Diagnosis

1. Clinical picture and hormonal abnormality
2. X-ray pituitary fossa
3. CT scan of pituitary

Treatment

Choice:

1. Hypophysectomy - transphenoidal
   transfrontal
2. Radiotherapy
3. Drugs

Types of Tumours

(a) Prolactinomas

Hypophysectomy + radiotherapy
Bromocryptine (microadenomas)

(b) Acromegaly

Bromocryptine
Radiotherapy

(c) Cushing's Disease

Bilateral total adrenalectomy + pituitary irradiation
Hypophysectomy

HERNIA

GENERAL DEFINITION

Protrusion of whole or part of a viscus from its normal position through an opening in the wall of its containing cavity.

Anatomical Classification

1. External hernia
   - herniation through a defect with the contents going out of the cavity so that the hernia may be detectable.

2. Internal hernia
   - herniation of viscus through openings within the cavity. The contents do not appear outside the cavity.

Sites

1. Common hernias
   - inguinal, femoral, umbilical, paraumbilical, epigastric, incisional, paracolostomy.

2. Uncommon hernias
   - obturator, spigelian, lumbar, gluteal, sciatic, perineal.

Aetiology

1. Congenital defect
   - Patent processus vaginalis
     Patent canal of Nuck
     Non-obliterated umbilicus

2. Acquired defect
   - Surgical incisions
     Muscle weakness - obesity, pregnancy,

3. Raised intra-abdominal pressure
   - Chronic cough
     Constipation
     Urinary obstruction
     Parturition
     Vomiting
     Muscular effort
     Ascites

Surgical Pathology

1. Coverings
   - Skin
     Stretched muscles and fascia
     Sac - peritoneum

2. Contents
   - Omentum
     Bowel
     Part of bowel circumference - Richter's
     Meckel's diverticulum - Littre's
     Two loops of bowel - Maydl's
     Bladder

3. Pathological types
   - Reducible, irreducible, obstructed,
     Strangulated, sliding.

Clinical Presentation

1. Lump - gradually enlarging, reducible, discomfort
2. Feeling of content
   - omentum - soft
     bowel - gurgling
3. Complications
   - intestinal obstruction
     strangulation - peritonitis

I. INGUINAL AND FEMORAL HERNIAS

Surgical Anatomy

Differential Diagnosis of Lump at the Groin

1. Hernia
2. Lymph node
3. Saphenous varix
4. Femoral aneurysm
5. Cord - hydrocele, lipoma
6. Testis - incomplete descent
   ectopic
7. Psoas abscess, bursa
8. Ruptured adductor longus

Distinction between Inguinal and Femoral Hernias

1. Appearance
2. Position

Distinction between Direct and Indirect Inguinal Hernias

1. Inspection
2. Appearance at reduction
3. Ring obliteration test

Types of Indirect Inguinal Hernias

1. Bubonocele
2. Funicular
3. Complete scrotal

Treatment of Inguinal Hernia

1. Truss
2. Operations - herniotomy
   herniorrhaphy - Bassini
3. Hernioplasty - rectus sheath
   fascia lata
   tantalum mesh
   mersilene mesh
   nylon

Treatment of Femoral Hernia

Steps of Operations
- excision of sac
  obliteration of defect

Approaches
- supra-inguinal
  inguinal
  subinguinal

II. UMBILICAL HERNIA

Weak umbilical scar
Treatment - conservative for 2 years
90% success

III. PARAUMBILICAL HERNIA

Middle age, obese, multiparous women
Large, irreducible
Strangulation likely
Treatment - operative

IV. EPIGASTRIC HERNIA

Upper midline
Extraperitoneal fat + sac
Pain
Treatment - excision

V. OBTURATOR HERNIA

Women above 50
Presentation - intestinal obstruction
Pain radiating to knee
Vaginal examination
Lump at medial aspect of thigh
Treatment for intestinal obstruction

VI. LUMBAR HERNIA

VII. SPIGELIAN HERNIA

Through linear semilunars
Strangulation common
Treatment - operative

VIII. GLUTEAL HERNIA

Greater sciatic notch
Bowel obstruction

IX. SCIATIC HERNIA

Lesser sciatic notch
Bowel obstruction

SCROTAL SWELLING

Anatomical Classification

1. Testis and Epididymis
   - inflammation
     tumours
     cysts of epididymis
     spermatocele

2. Spermatic Cord
   - torsion
     varicocele

3. Tunica Vaginalis
   - hydrocele
     haematocele

Clinical Classification

1. Acute painful swelling

   (a) Acute viral orchitis
   - Mumps
     Young age
     Danger of infertility

   (b) Acute epididymo-orchitis
   - Associated urogenital infection
     Retrograde infection
     Painful testis and epididymis
     Scrotum swollen
     Treatment - rest, elevation, antibiotics

   (c) Torsion of testis
   - Young age
     Abnormal opposite testis - long mesorchium, horizontal, ectopic, capacious tunica vaginalis
     Scrotum swollen
     Treatment - excision/fixation

2. Solid Swellings

(a) Testicular neoplasm

(b) TB epididymo-orchitis
- Haematogenous
  Epididymis first, cord
  Prostate and seminal vesicles
  Discharging sinus
  Treatment - chemotherapy
  surgery for residual disease

(c) Haematocele
- Acute trauma - sudden onset of haematoma
  rupture of testis

  Old injury - enlarged testis
  exploration to exclude tumour

3. Cystic Swellings

(a) Cysts of epididymis
- Cystic degeneration of appendices
  Unilateral/bilateral
  Single/multiple
  Separate from testis
  Small
  Clear fluid - translucent

(b) Spermatocele
- Retention cyst of epididymis
  Spermatozoa
  Small
  Opalescent fluid - translucent

(c) Hydrocele
- Translucent bag of fluid round testis
  Aspiration
  Operation - excision, Jaboulay's operation

DISEASES OF VEINS AND LYMPH VESSELS

I. SURGICAL ANATOMY OF THE VEINS OF THE LOWER LIMB

1. The peripheral veins consist of three layers of venous networks:

   (a) Subcuticular venules.
   (b) Network of subcutaneous veins.
   (c) Long and short saphenous veins which lie on the deep fascia.

   (Note: i. Relationship of saphenous nerve, medial femoral cutaneous nerve and the sural nerve to the veins.
   ii. Branches of the long saphenous vein which include:

   Three groups of tributaries at knee region - the calf group, the anterior vein of the leg and the posterior arch vein.

   Two large tributaries at thigh - the posteromedial and anterolateral veins.

   Four branches near the sapheno-femoral junction - the superficial epigastric, superficial circumflex iliac, superficial external pudendal and the deep external pudendal.)

2. The deep veins - consist of paired venae comitantes of leg arteries i.e., the anterior and posterior tibial and peroneal; but single popliteal and femoral veins.

3. The valves and the perforating veins - the valves of the deep veins are profuse and important in the pump mechanism, ensuring blood flow from superficial to deep venous system. The number of valves becomes progressively less from distal to proximal.

   The valves of the superficial veins are important in preventing varicosities. They consist of major valves with strong white cusps near the sapheno-femoral and sapheno-popliteal junctions and more numerous minor valves which are delicate transparent cusps lower down the veins.

The perforating veins are valved to permit flow from superficial to deep system.

Along the long saphenous system, there are three main groups of perforating veins:

1. A constant long perforator vein from middle to lower third of thigh and end in the femoral vein in the Hunter's canal.

2. Knee perforator, at just below knee level, close to posterior border of tibia connecting either the long saphenous or the posterior arch vein to the posterior tibial vein.

3. Ankle perforating veins, there are three veins:

   (a) Upper, in middle of leg at posterior margin of tibia.
   (b) Middle, one hand's breadth above tip of internal malleolus.
   (c) Lower, just behind and below internal malleolus.

   These communicate by tributaries with long saphenous veins, penetrate deep fascia and drain into the posterior tibial venae comitantes.

Along the short saphenous system, there is a constant ankle perforating vein between the middle and lower third of calf at the outer border of the tendo-Achillis and an inconstant (present in about 25% of cases) perforating vein in the mid-calf region.

II. VARICOSE VEINS OF THE LOWER LIMBS

Classification :

1. Primary familial varicose veins - can be of the long saphenous system (the commonest), the short saphenous system (less common) and the primary ankle perforator imcompetence (very uncommon) or a combination of any of the above.

2. Secondary varicose veins - occur after a deep vein thrombosis, resulting either in permanent blockage of a major iliac vein and with generalised venous hypertension in the limb or in destruction of the valves in the ankle perforators.

3. Varicose veins secondary to arteriovenous fistulae (traumatic or congenital)

4. Capillary veins (venous stars, telangiectasia, or burst veins) little clusters of dilated capillaries appear often during pregnancy. Main effect is cosmetic, cause unknown, may be related to level of oestrogen.

5. Athletes hypertrophied veins, not actually due to valvular incompetence, usually unsightly and sometimes aching.

Primary Familial Varicose Veins

Major causes:

1. Heredity - Family history present in about 70% of cases, probably due to inherited absence of one or more strategic valves.

2. Race - Essentially a disease of the European race, far less common in the pure black Africans, the Indians and the Asiatics.

3. Other causes - Probably affects the rate of progression of the disease, these included:

    - pregnancy
    - prolonged standing
    - overweight
    - diet

Symptoms

1. Uncomplicated varicose veins.

2. Disfigurement, especially in females.

3. Aching and pain, comfortable when moving or walking but ache on standing.

    (Note: Always look for other causes of pain e.g., osteoarthritis of knee and hip or disc lesions, must exclude arterial causes.)

4. Swelling, variable complaint, usually absent if the ankle perforators are competent.

5. May complain of fullness and heaviness without actual swelling.

6. If gross swelling occurs, look for deep vein thrombosis, general cause of lower limb oedema, and lymphoedema.

7. Rarer symptoms - discolouration,
cramps (nocturnal),
pain over the veins.

Complications

1. Haemorrhage - spontaneous, traumatic or subcutaneous. The bleeding can be profuse and sometimes fatal, difficult to stop without surgical intervention.

2. Thrombophlebitis - occurs spontaneously or as a complication of prolonged bed rest e.g., after operation. A group of varicose veins become tender, hot, inflamed and hard or solid, with surrounding oedema. Sometimes complicated by secondary bacterial infection with spreading cellulitis.

   (Note: This may trigger off a deep vein thrombosis.)

   Treatment: Pressure bandaging and early ambulation.

3. Eczema - early form as slightly pigmented scaly patch over the enlarged group of varices, or over the internal malleolar area, this is a precursor of venous ulceration.

   Treatment:

   Early surgical intervention of the varicose veins. Do not apply lotions and ointments to ameliorate the irritation or itching as many cases will develop drug sensitivity.

   Late form or complicated by drug sensitivity or secondary infection are difficult to treat and may persist as lifelong problem.

4. Ulceration - nearly all venous ulcers occur in the lower third of the leg, especially around the malleoli. This area is drained mainly by the ankle perforating veins, and the venous ulcers are associated with valve incompetence in the perforating veins. The ulcer is due to a slow tissue necrosis caused by the high venous pressure in the capillary loops of the skin and subcutaneous tissues, causing cellular oedema and then necrosis.

Treatment of venous ulcers:

To ascertain the diagnosis, venous ulcers are to be differentiated from the following conditions:

(a) Ischaemic ulcer, due to impaired arterial blood supply, the peripheral pulses must always be examined.

(b) Traumatic ulcers, this occur around bony prominences.

(c) Other causes include:
- Infective e.g., syphilitic, pyogenic.
- Neuropathic, diabetic and alcoholic peripheral neuritis, tabes dorsalis and syringomyelia. Theses ulcers commonly occur on the sole of the foot or the heel where they may penetrate to bone or joint levels. The toes and feet are commonly affected too.
- Neoplastic e.g., squamous carcinoma, or malignancy developing in the edge of a long-standing ulcer or osteomyelitic sinus (Marjolin's ulcer).
- Cryopathic, from cold injury, self-inflicted.

Treatment - three main ways, namely:

i. Posture

Bed rest with elevation of the legs above heart level.

ii. Elastic compression bandaging

Firm and even pressure high enough to counteract the high venous pressure efficiently (80-100 mmHg). This may be applied by the continuous method or intermittently where the bandaging may be removed when the patients sleep with the legs elevated.

Systemic and not local antibiotics are necessary to control the infection.

iii. Surgery

This is only applicable to incompetence in the ankle perforating veins and not for valve loss in the deep veins or a persistent obstruction of the large iliac veins. It is, however the only way of aiming at cure for incompetent perforators. The methods include surgical ligation of the perforators (either extrafascial approach, the Cockett's operation or subfascial approach, the Linton's operation or Rob's procedure) or injection sclerotherapy with compression bandaging. Skin grafting may be needed.

III. GENERAL MANAGEMENT OF VARICOSE VEINS

1. Conservative Measure

(a) No treatment e.g., for trivial capillary veins, venous stars, for minor long saphenous incompetence in elderly patients.

(b) Supportive treatment and posture

Elastic bandaging or elastic stockings e.g., in patients waiting for surgery, and elderly patients reluctant for surgery.

Bed rest with elevation of legs e.g., initial healing of ulcer, must watch for risks of prolonged immobilisation especially in old patients.

2. Injection Treatment (Compression Sclerotherapy) - Aims at inducing aseptic thrombosis which organises and closes the vein.

Practical Points of Injection Treatment

(a) Accurate injection of a small dose (0.5-1 ml) of sclerosant, e.g., Ethanolamine oleate, sodium tetradecyl sulphate 3% (S.T.D. or Thrombovar) into a short segment of vein, and its retention there for a minute or more to act on the vein wall.

(b) The maintenance of steady pressure over the injected segment for at least 6 weeks - to prevent the formation of a bulky thrombus which will recanalise.

(c) Elastic compression and active movement of the whole leg, starting within minutes of the injection.

Injection treatment may take more than one session and with injection of more than one site for each session. Failure to sclerose the vein is usuallly due to poor technique in injection and failure to continue with at least 6 weeks of compression.

Complication of Injection Treatment

(a) Production of a painful thrombus.

(b) Extravenous injection with local inflammatory focus, or injection ulcer.

(c) Permanent brown staining of skin.

(d) Anaphylactic reaction with the sclerosant.

(e) Deep vein thrombosis and pulmonary embolism.

(f) Peri-arterial injection or intra-arterial injection with post injection gangrene.

3. Operative Treatment - Aims at flush ligation which cuts off accurately the source of the high pressure leak from the deep veins, and stripping which removes the dilated veins. The three operations advised for varicose veins are:

(a) Ligation and division of the sapheno-femoral junction, with saphenectomy of the long saphenous vein from the groin to the ankle by stripping.

(b) Ligation and division of the sapheno-popliteal union, saphenectomy of the varicose short saphenous vein from the popliteal space to the ankle by stripping.

(c) The ligation and division of a faulty communication vein or veins located usually in the lower third of the leg or thigh.

(Note: The act of stripping does not necessarily destroy incompetent perforating veins)

Complications of Operation

(a) Haematoma and bruising, this is usually present along the stripper track and is a normal course of event. Usually this is absorbed within 3 to 4 weeks.

(b) Lymphocele, due to a small collection of lymph in the groin wound causing a painless egg-sized swelling, this arises from tearing of some of the groin lymphatics during a more extensive dissection e.g., at re-exploration for recurrent varicose veins.

(c) Wound sepsis

(d) Saphenous neuritis, due to temporary or permanent nerve damage with hypersensitivity to touch occurring 2 to 3 weeks after the operation or giving an area of anaesthesia with an uncomfortable zone of hypersensitivity around it.

(e) Lymphoedema of leg, this is usually minor and occurs more with ligation of the perforators than with stripping. They tend to subside spontaneously in 1 to 2 months' time.

(f) Induration of the stripper track.

(g) Deep vein thrombosis and embolism.

IV. DEEP VENOUS THROMBOSIS

This affects most commonly the lower limbs, it may result in significant complications which include pulmonary embolism, perforator vein incompetence and varicose veins.

Sites

1. Upper limb, in superior vena cava, in axillary vein.
2. Lower limb.
   (a) In soleal sinuse (calf vein thrombosis)
   (b) In Iliofemoral vein (usually giving a white swollen limb called phlegmasia alba dolens).
   (c) In entire venous system (with venous gangrene and sometimes called phlegmasia caerulea dolens).

Predisposing Factors (Virchow's triad)

1. Stasis e.g., heart failure, prolonged bed rest, pelvic obstruction.
2. Endothelial trauma e.g., in rough handling of unconscious patients, pressure on unprotected calf muscles, intravenous therapy and spreading infection from the surrounding structures.
3. Altered constituents of the blood e.g., in dehydration, in polycythaemia, leukaemia and malignancy, increased stickiness of platelets after operation and parturition.

Clinical Features

1. The predisposing cause, this is usually present if carefully looked for. If absent, one must always suspect a hidden malignancy.
2. The stage of phlebothrombosis, because the clot is propagative and not attached to the vein wall, there are no local signs to indicate its presence. Various tests are available to confirm and locate the venous thrombosis before overt clinical features are manifested. These tests are:

(a) Venography, probably the most accurate test and should be used if pulmonary embolism has occurred.

(b) Labelled fibrinogen uptake, radioactive iodine-labelled fibrinogen is taken up and incorporated as fibrin into any new thrombus and this uptake can be detected with a scintillation counter. The test is reliable when compared with venography but it is of doubtful value in the upper thigh and of no value at levels above the inguinal ligament. It is of practical value in high risk patients.

(c) Ultrasonics, a venous hum can be heard over the femoral vein and it can be augmented by compressing the calf, absence of this augmentation implies occlusion.

3. The stage of thrombophlebitis - There is often calf tenderness, elevation of temperature, and swelling of the limb with pitting oedema. In cases of massive deep vein thrombosis, severe shock may accompany oedema of the entire limb and the lower abdominal wall. There is usually agonising pain and the limb has a dusky purple colour which persists on elevation. The subcutaneous veins are turgid and peripheral arterial pulses may be impalpable.

Treatment

1. Prevention is better than cure e.g., early mobilisation, mechanical means of intermittent stimulation of the calf muscles.

   Antithrombotic agents such as small dose of subcutaneous heparin (5000 units every 12 hours) or dextran 70 during and after operation.

2. Definitive treatment

   (a) Limb support with bandaging or elastic stocking.
   (b) Elevation
   (c) Anticoagulation, helps to reduce the extent of the consecutive thrombus and the incidence of pulmonary embolism. Usually given intravenously with 5000 units as a loading dose followed by continuous infusion of 5000 units in 500 ml of 5% dextrose solution every 6 hourly. The dosage is adjusted to maintain the clotting time between 20 and 30 mins. This should be continued for about 10 days when oral anticoagulants may be introduced and continued for about 6 months. Dosages are adjusted to keep the prothrombin time between 2 to 2.5 times normal. The patients should be kept on leg bandaging during this period.

(d) Fibrinolytic drugs e.g., streptokinase, a plasminogen activator may be tried. This is especially useful in recent thrombosis less than 3 days and in the absence of a wound.

(e) Surgery - This is seldom necessary, most of the cases are for prevention of pulmonary embolism e.g., caval plication and caval umbrella.

Outcome Of Deep Vein Thrombosis

When the clot is confined to the paraxial veins, (principally the soleal sinuses) little harm ensues. When the axial vein becomes blocked, it can be complicated by:

1. Pulmonary embolism, occurs between the 7th and 10th days after operation. With minor emboli, this may be symptomless, with massive embolism, instant or rapid death may occur. Repeated smaller emboli may give rise to pulmonary hypertension.

2. Damage to the valve in the deep veins and at the deep and superficial junction at a later stage, resulting in varicose veins and ulcers.

Acute Lymphangitis

Characteristic red blushes and streaks in the skin, corresponding to the inflamed lymphatic. Streptococcus is the common organism. Toxaemia is severe. Permanent lymphatic obstruction may follow leading to persistent oedema.

Treatment

Bed rest, elevation, antibiotics.

Lymphoedema

Caused by accumulation of fluid in the lymphatics.

Differentiate from other causes of lower limb oedema:

1. Central causes - cardiac,
renal,
hepatic,
nutritional,
hormonal.

2. Venous causes - deep vein thrombosis,
varicose vein,
fistula.

3. Local causes - injuries,
fracture,
muscle contusion,
cellulitis.

Primary Lymphoedema

Result of obstruction to lymphatic flow due to subcutaneous lymphatic channel developmental defects.

Three main groups:

1. Aplasia - usually apparent at birth (lymphoedema congenita)

2. Hypoplasia - few and underdeveloped channels, majority of the cases.

3. Varicose lymphatics - may be associated with congenital arteriovenous fistula and also with 'chylous reflux'.

   Depending on the time of presentation, they are also known as lymphoedema congenita, lymphoedema praecox (at puberty, or lymphoedema tarda (at adult life).

Secondary Lymphoedema

Result from

1. Trauma e.g., surgical removal
2. Repeated acute infections
3. Chronic infection e.g., tuberculosis, filariasis and fungus infection.
4. Malignant obstruction

Treatment

1. Conservative - limb massage, limb elevation, elastic stockings or bandages, bed rest and antibiotics for attacks of acute infection, intermittent diuretics.

2. Surgery - reserved for severe disabilities or disfigurement.

   Aims at removal of all the abnormal subcutaneous tissues and either skin grafting or rolling the excess skin like a swiss roll cake along the leg, hoping that the subdermal lymphatics may assist drainage.

   Microsurgery makes it possible to anastomose dilated lymphatics to veins (lymphovenous anastomosis) to establish drainage.

PERIPHERAL ARTERIAL DISEASES

Causes

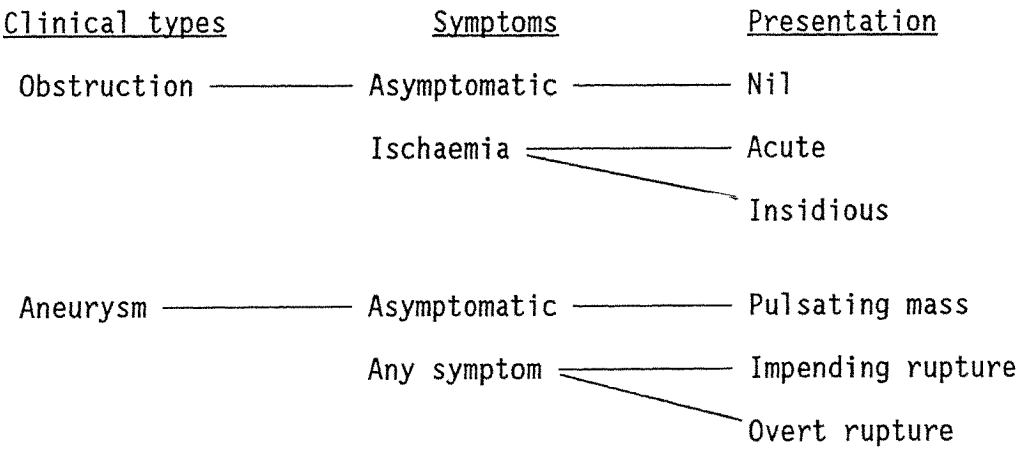
Atheromatous* - most common

Buerger's disease*
Other arteritides*

Embolism*
Trauma

Arterio-venous malformations (AVM)

* Local manifestation of systemic disease



I. ARTERIAL OBSTRUCTION

1. Ischaemia of Insidious Onset

(a) Symptoms

Intermittent claudication - onset
- site
- distance
- progression

Rest pain

(b) Signs

Trophic changes - hair
- nails
- skin
- muscle

Pulses - volume
- bruit

Temperature
Ulceration
Gangrene

Detect clinical features of disease of other systems, especially
cardiovascular
cerebrovascular
respiratory
renal

(c) Clinical Evaluation of Peripheral Ischaemia

Intermittent claudication only

Femoral pulse ±	Aorto-iliac occlusive disease Usually younger age group (50-60) Progression rapid Good prognosis with revascularisation
Femoral pulse ++ Popliteal pulse ±	Femoral-popliteal occlusive disease Elderly patients (70-80) Slow progression May not need intervention

Rest pain or gangrene

Femoral pulse ++ Popliteal pulse ++	Small vessel disease, e.g. Buerger's disease, Diabetes mellitus. Poor prognosis
Femoral pulses ±	Multiple level occlusion Fair prognosis

(d) Investigations

General "work-up" for any elderly hospitalised patient
- haematological
- biochemical
- serological
- microbiological
- CXR
- ECG
- respiratory function test
- cardiological consultation

Specific: Anatomic location and functional evaluation

Arterial investigations

Non-invasive - Segmental blood pressure measurement and waveform analysis by Doppler ultrasound
- Exercise test
- Pulse volume recording

Invasive - arteriography*
- digital subtraction angiography

* Arteriography - only for patients in whom surgery is indicated
- type of examination depends on pulse level and expertise of radiologist

(e) Management (see algorithm 1)

Claudicants

i. Generally conservative
weight reduction
stop smoking
exercise
foot care
control coexisting disease
- anaemia
- diabetes
- hypertension

ii. Surgery - for symptoms which interfere with patients' enjoyment of life or ability to work

Rest pain/gangrene
All in need of urgent surgery

(f) Operations

Depends on arteriographic findings and condition of patients :

i. Endarterectomy/profundoplasty
ii. Bypass graft
e.g., aorto-iliac
femoro-popliteal
axillo-femoral
femoro-femoral
iii. Sympathectomy
- increase skin flow
- diminish pain
- limit extent of amputation
iv. Amputation

2. Acute Arterial Obstruction

This is a surgical emergency

Delay results in loss of limb or life

Blood flow must be established within 4-6 hours if irreversible changes/amputation is to be avoided

Favourable outcome depends on prompt diagnosis

(a) Causes of Acute Ischaemia

Embolus*

Thrombosis

Trauma

* "Saddle" embolus is one which is lodged in the distal aorta across the bifurcation (saddle).

i. Sources of Arterial Emboli

90% from heart
- atrial fibrillation
- mitral valve disease
- postmyocardial infarction

Others
- atheromatous
- myxoma
- subacute bacterial endocarditis
- paradoxical

ii. Arterial Emboli

Tend to lodge at bifurcations
70% - lower extremities
20-25% - brain
4-10% - visceral arteries

(b) Clinical Features of Acute Ischaemia

Pain
Pallor
Paraesthesia
Paralysis
Pulseless
Perishing cold

Colour change is a late sign

(c) Management (see algorithm 2 & 3)

Heparinise and operate
Arteriography is rarely needed and must not be the cause of delay
Site of obstruction can be established by palpation
Operation is performed under local anaesthesia in most patients

(d) Operations

Embolectomy with Fogarty balloon catheters
Fasciotomy
Bypass in rare instances
Continue anticoagulation in some

II. ANEURYSMS

Causes

Degenerative e.g. atheromatous - most common
Traumatic e.g. false aneurysm
Inflammatory e.g. subacute bacterial endocarditis
Congenital e.g. berry aneurysm

Complications

Rupture - abdominal aortic aneurysm

Thrombosis) popliteal
) - femoral
Embolism) carotid

Infection - Salmonella

Pressure effect on adjacent organ

Abdominal Aortic Aneurysm

97% infrarenal
Usually extend to the left side
Rarely thrombose or give rise to embolism
Occasionally cause pressure effects
All (> 5 cms) at risk of rupture
- 20% within 1 year of diagnosis
- additional 10% for each year thereafter

(a) Symptoms

Most asymptomatic except for a pulsating mass; incidentally discovered by patient or doctor

Any symptom = IMPENDING RUPTURE
- Low back pain/sciatica
- Renal colic type pain
- Any acute abdominal condition

Association with peripheral ischaemia uncommon

(b) Signs

Pulsating mass
If infrarenal, can get above upper border
If above bifurcation, lower border is above umbilicus

(c) Triad of Rupture

Mass - pulsation may be masked
Pain - abdomen or back
Shock - transient or profound

(d) Investigation

Plain X-ray abdomen - AP and lateral
- calcification

Ultrasound) - confirm diagnosis
CT scan) - estimate size

Arteriography - not essential for diagnosis
- indicated for
1. clinically "high" aneurysm (suprarenal)
2. associated peripheral ischaemia
3. renal failure
4. uncertainty of diagnosis

(e) Management (see algorithm 4)

All should be operated on unless life expectancy is less than 1 year or aneurysm is less than 5 cm or medically unfit for surgery.

All untreated ruptured abdominal aortic aneurysm is fatal

Mortality rate of operation for intact abdominal aortic aneurysm < 5%

Mortality rate of operation for ruptured abdominal aortic aneurysm > 50%

(f) Operations

"Aneurysmectomy" and inlay graft (Endoaneurysmectomy)
- straight tube
- bifurcated graft

Procedures to cause thrombosis or to produce isolation of aneurysm
- not usual practice for aortic aneurysm
- more commonly applicable to peripheral artery aneurysm, e.g. popliteal artery aneurysm

III. ARTERIAL INJURIES

1. Penetrating Trauma

(a) 20% have normal pulse distal to injury
(b) 30% have diminished pulse distal to injury

2. Non-penetrating Injuries

(a) Adjacent to fracture fragments
(b) Intimal tear with infolding without gross external damage
(c) Often associated with delayed diagnosis
(d) "Spasm" should be diagnosed at operation

Arteriography is indicated when in doubt of the injury.
(see algorithm 5)
Close follow-up by non-invasive tests allows early diagnosis

IV. ARTERIOVENOUS MALFORMATION

1. Arteriovenous Fistula

(a) Signs

i. Thrill
ii. Dilated pulsating veins
iii. Continuous murmur

(b) Complications

i. Skin ulceration
ii. Limb hypertrophy (in children)
iii. Heart failure (rare)
iv. Subacute bacterial endocarditis (rare)

(c) Management

i. Excision
ii. Ligation
iii. Embolisation

2. Cavernous Haemangioma

Localised or diffuse
Commonly in the limbs
Discolouration of skin
Presentation - disfigurement
- phlebitis
- bleeding
- loss of function
- skin ulceration
Signs of emptying
Surgery curative for localised lesions
Many recur after apparently complete excision

V. BUERGER'S DISEASE

Young male smokers

Medium and small arteries and veins affected

Feet affected more than hands

Present with rest pain, gangrene and ulceration

Femoral and popliteal pulses usually intact

Arteriography shows cut-off in distal femoral downwards with "tree trunk" appearance

Diagnosis made by clinical features

Mainstay of treatment is total abstinence from smoking

Arterial reconstruction rarely possible or effective

Sympathectomy and amputation as last resort

Life expectancy not reduced by disease

VI. RAYNAUD'S PHENOMENON

Pallor - cyanosis - rubor
Precipitated by cold or emotion

Primary Raynaud's
- no underlying disease

Secondary Raynaud's
- Buerger's
- scleroderma
- cervical ribs
- blood disorders

Management
- avoid cold
- sympathectomy (severe cases)
- close follow-up for underlying disease

VII. COMPLICATIONS OF ARTERIAL SURGERY

Early

Local	Haemorrhage	Colonic necrosis
	Thrombosis	Paraplegia
	Wound problems	Embolisation

Systemic
- myocardial infarction
- cerebrovascular accident
- respiratory problems
- renal failures

Late
- false aneurysm
- graft-enteric fistula
- graft occlusion
- graft infection

VIII. FACTORS PROMOTING DEVELOPMENT OF DIRECT ARTERIAL SURGERY

1. Blood transfusion
2. Anticoagulation and reversal
3. Technological advances
   - (a) evaluation of patients
   - (b) materials of surgery

(a) Evaluation of Patients

i. Non-invasive diagnostic modalities
ii. Arteriography
iii. Digital subtraction angiography

(b) Materials of Surgery

i. Instruments
ii. Grafts
iii. Sutures
iv. Catheters

i. Instruments
- atraumatic
- fine control
- durable
- reliable
- light weight
- biomechanically efficient
- antiglare

ii. Grafts

(1) Biological
- autografts (saphenous vein)
- allografts (umbilical vein, saphenous vein)
- xenografts (calf carotid)
- silk

(2) Synthetic - plastic
- teflon
- dacron
- PTFE

(3) Synthetic grafts

a. Size (4mm to 40mm)

b. Configuration - straight
- bifurcated
- stepped
- tapered
- cuffed

c. Weave - knitted
- woven
- velour
- PTFE

d. Surface - crimpled
- smooth
- externally supported

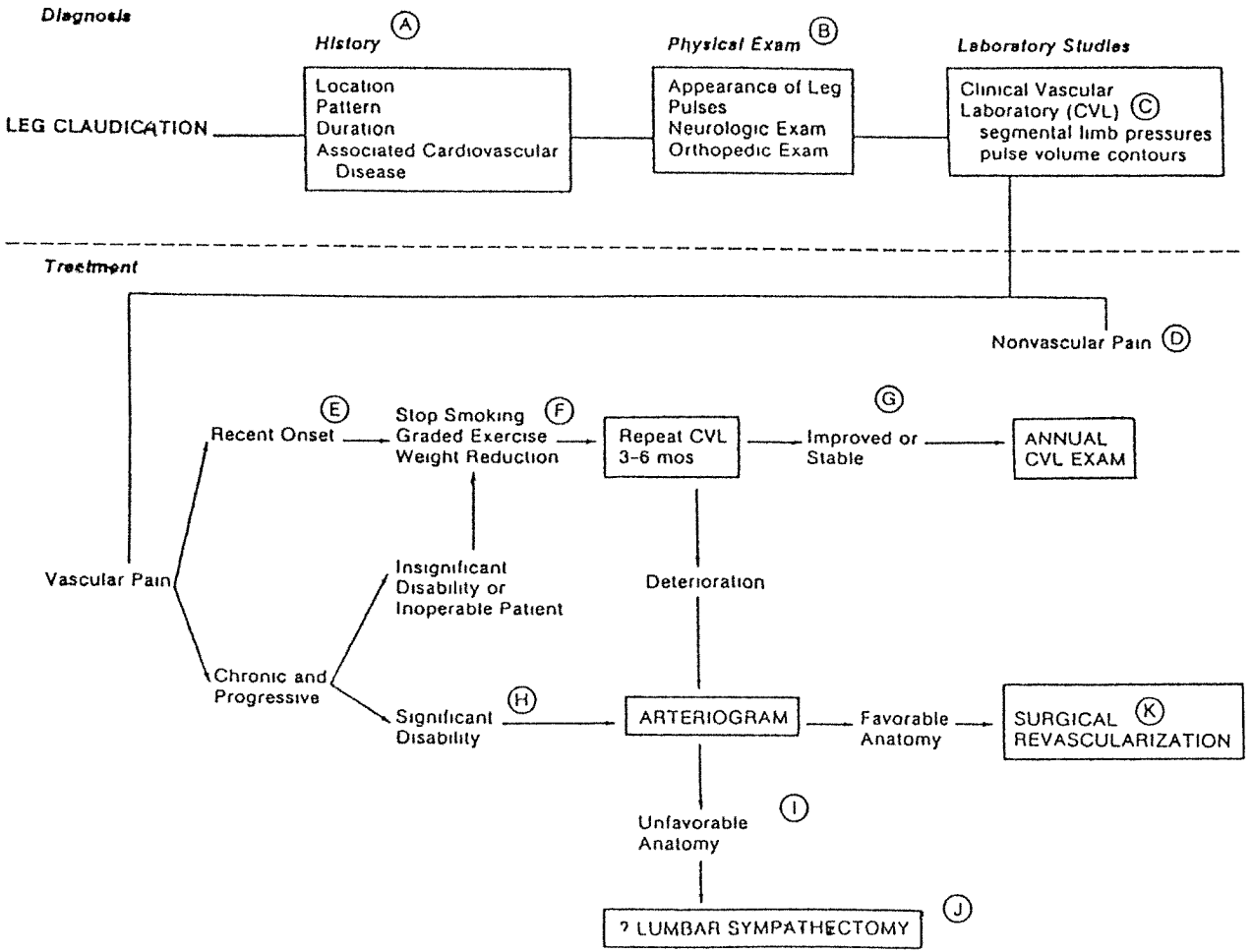
e. Reference line

f. Impregnation - antibiotics
(amikacin in collagen matrix)
- antithrombogenics
(endothelial cell seeding)

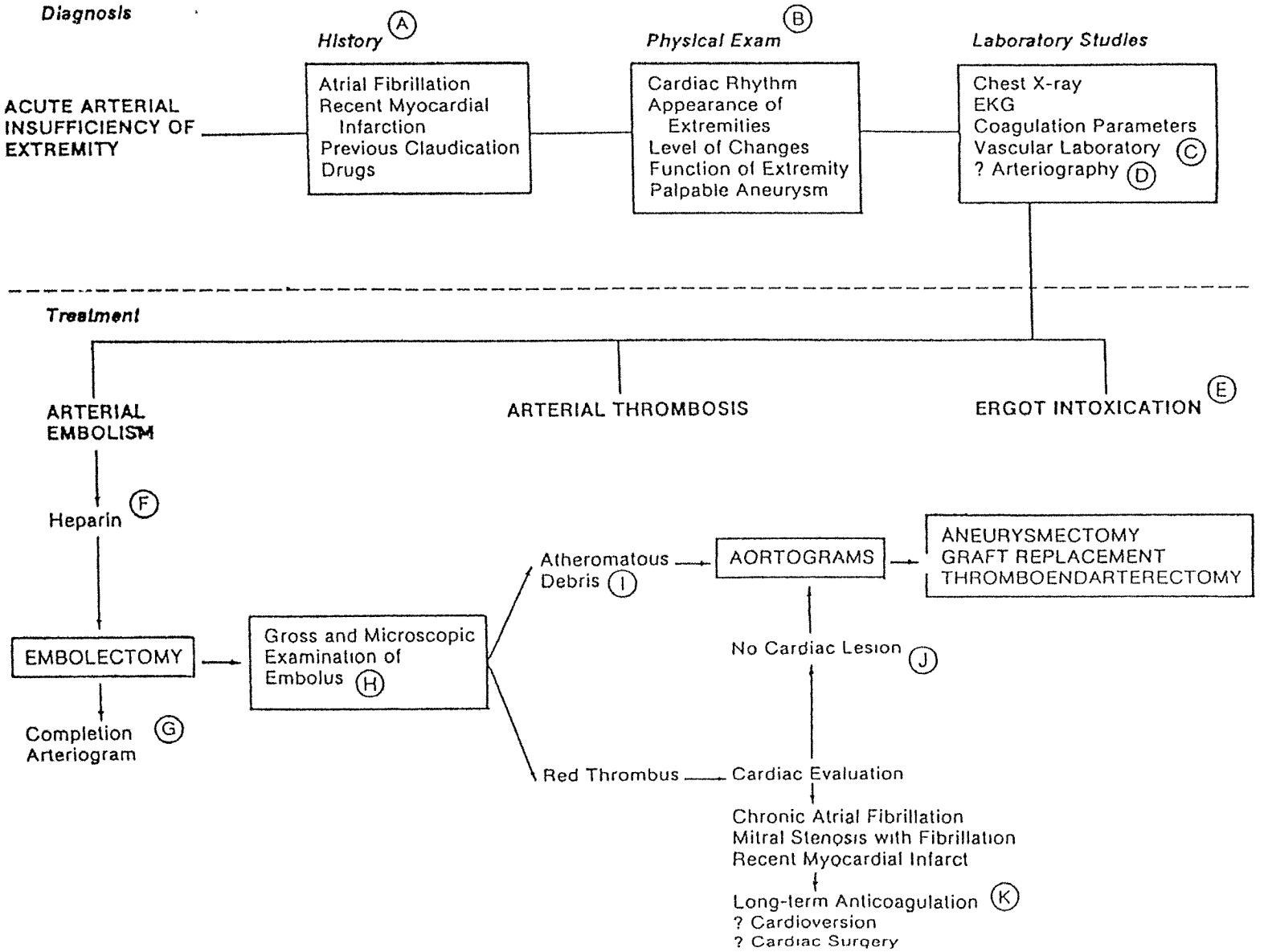
IX. HOW DOES ARTERIAL SURGERY DIFFER FROM OTHER BRANCHES OF SURGERY ?

1. More careful evaluation of patients
2. More haemodynamic disturbance at operation
3. Greater technical care required
4. Results immediately evident
5. Failures more catastrophic
6. Greater stress and vigilance for all

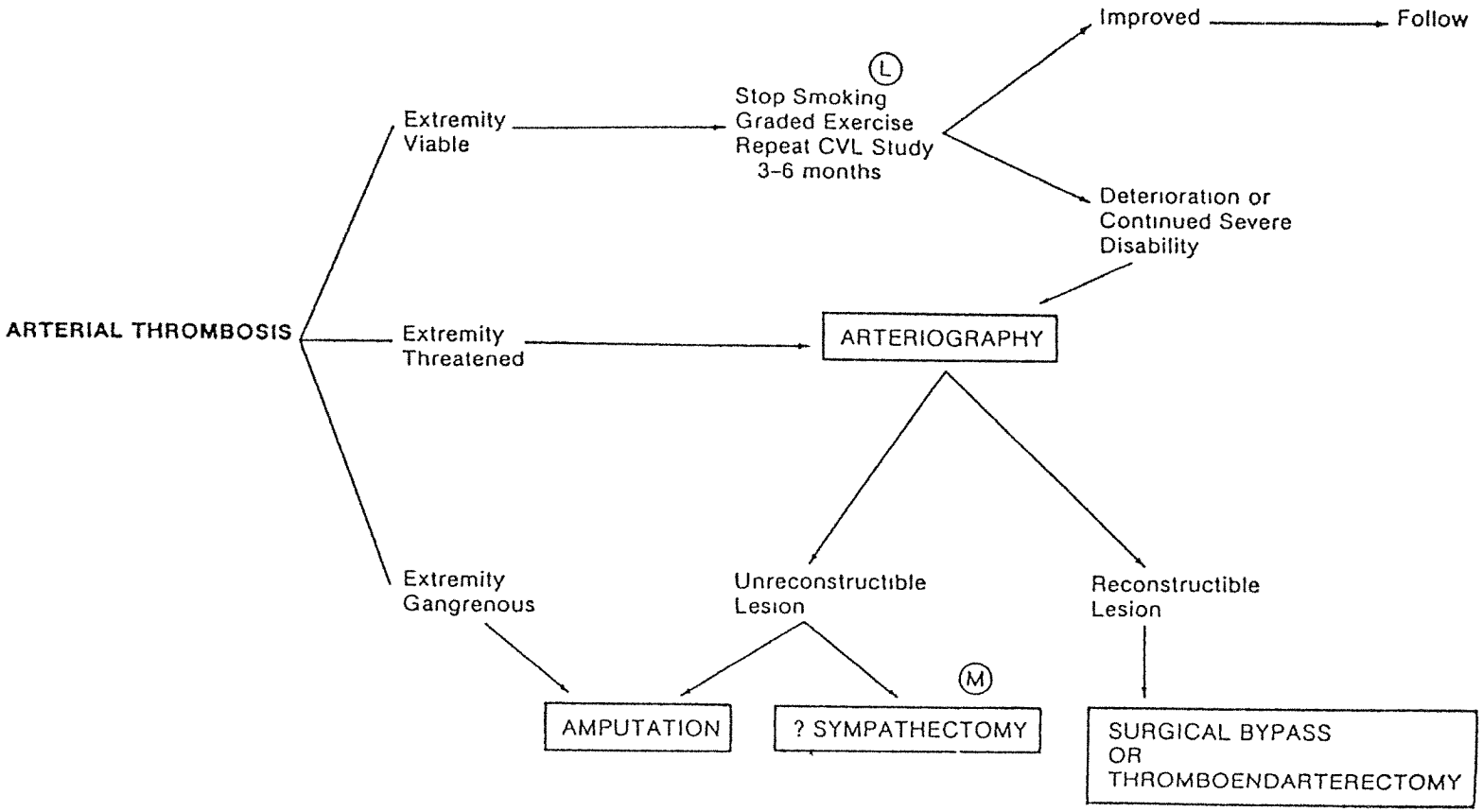
LEG CLAUDICATION



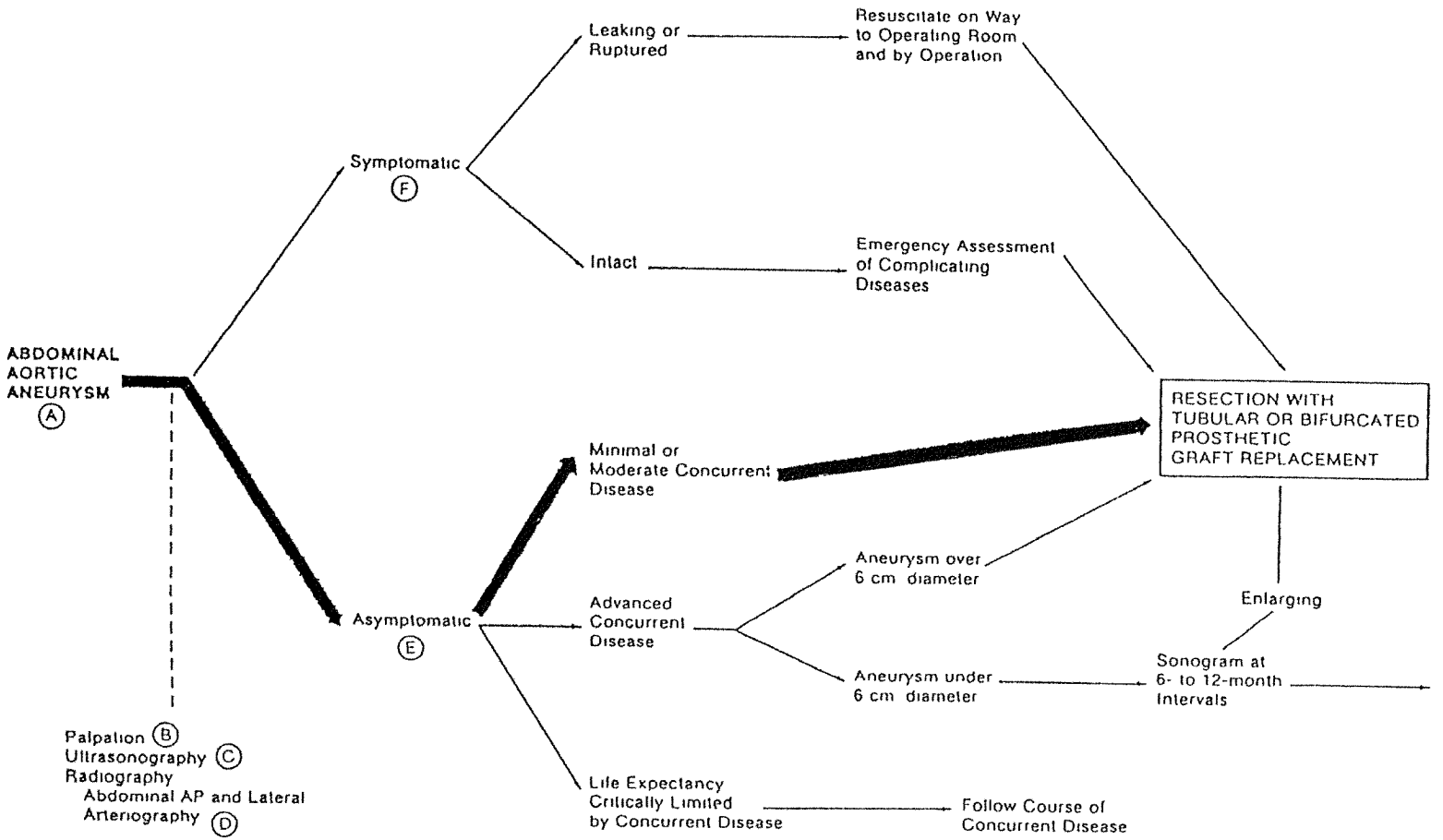
ACUTE PERIPHERAL ARTERIAL INSUFFICIENCY



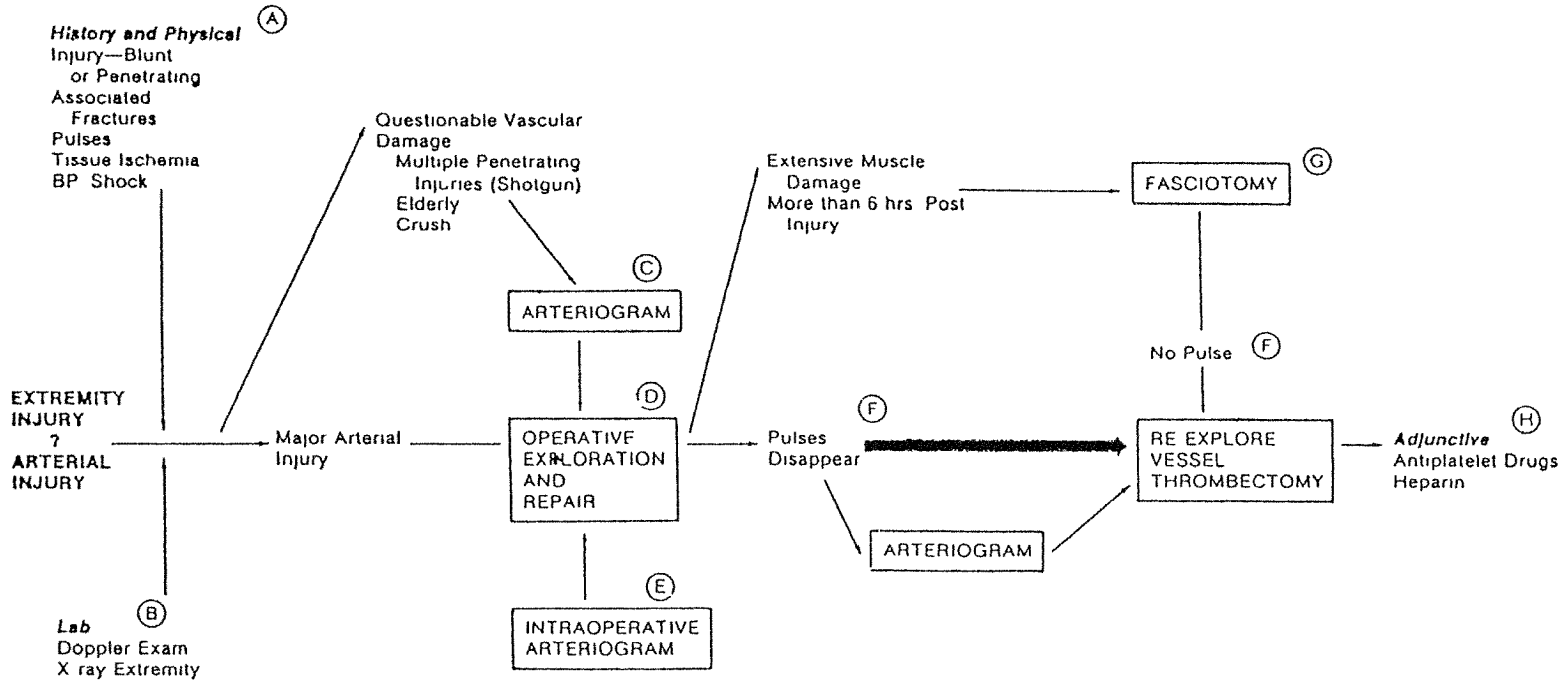
ACUTE PERIPHERAL ARTERIAL INSUFFICIENCY



INFRARENAL ABDOMINAL AORTIC ANEURYSM



ARTERIAL INJURY OF EXTREMITY



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

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 (paradoxic) incontinence
   enuresis
3. Act of urination - hesitancy
   interruption of urinary stream
   weak stream
   terminal dribbling
   retention
   dysuria

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.

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).

Genital Symptoms

1. Urethral discharge
2. Haemospermia - blood in seminal fluid
3. Sex difficulty
4. Infertility

Systemic Symptoms

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

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
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 fruitful of all laboratory screening tests and yet 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

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
6. Special test of differential functions of parts of renal tubules.

IV. UROLOGICAL INVESTIGATION

Renal Function Tests

1. Serum electrolytes
2. Blood urea and creatinine
3. Serum acid/base status

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

   Contrast agents (Iodine containing) given intravenously are eliminated by glomerular filtration and are not reabsorbed by the tubules. Water reabsorption causes a progressive increase in concentration of contrast (30-50x that of plasma).

   Application

   (a) Crude test of renal function

   (b) Demonstration of anatomy of urinary tract, particularly of upper urinary tract.

   (c) Crude test of bladder function

3. Retrograde and antegrade urogram

Principle

Introduction of contrast agents 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-functioning kidney or poorly functioning kidney.

4. Micturiting Cystourethrography (Voiding Cystourethrogram)

Principle

Contrast medium is introduced into bladder via catheter. Serial X-rays are then taken when the patient urinates.

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 into urethra with the X-ray being taken while the fluid is being injected.

Application

Anatomy of urethra e.g., before and after urethroplasty.

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 necessary.

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 permeation into IVC.

7. Lymphography

Principle

Injection of contrast material after cannulation of superficial lymphatic vessel (on the foot) with cranial 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 cycles per second which cannot be appreciated by human ear (medical ultrasound 1.5×10^6 cycles per second). 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 probe).
(f) Scrotal mass differentiation

VIII. RADIOISOTOPIC UROLOGICAL STUDIES

Principle

The radiopharmaceuticals 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 or clearance study.

Applications

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

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

- urethrocystoscopes
- uretero-renoscopes
- percutaneous nephroscopes

Applications

(a) Direct inspection
(b) Biopsy potential
(c) Retrograde and antegrade - radiology
(d) Split renal function test

X. URODYNAMIC STUDY

Principle

Field of study which encompasses the study of hydrodynamic of urine transport. The various methods of objective measurement of bladder and sphincter function include:

1. Cystometry - volume/pressure change intravesically
2. Uroflowmetry
3. Urethral pressure profile
4. Electromyography
5. Cineradiological study of micturition

TUMOURS OF THE GENITO-URINARY TRACT

I. KIDNEY

Parenchymal

1. Benign tumours - angomyolipoma
   renin-secreting juxtaglomerular tumour

2. Malignant tumours -

   (a) Renal cell carcinoma
   - arises from proximal convoluted tubules
   - M:F = 3:1, usually in their 60's
   - presentation variable
   - triad : haematuria , pain, palpable mass (9%)
     constitutional symptoms
     endocrine/paraneoplastic syndromes
   - radiological diagnosis: IVP
     arteriogram
     venacavogram
     ultrasound
     CT scan
   - Treatment: radical nephrectomy for localised disease
     radiotherapy/chemotherapy ineffective

   (b) Sacromas: leiomyosarcomas, liposarcomas

   (c) Wilm's tumour
   - most common abdominal neoplasm in children
   - associated with congenital anomalies
   - present with a palpable mass
   - differential diagnosis: hydronephrosis,
     multicystic kidney
   - treatment: chemotherapy
     nephrectomy
     radiotherapy

Renal Pelvis (see Urothelial Tumours)

II. UROTHELIAL NEOPLASMS

1. The concept of "urothelium"
2. Renal pelvis, ureter, bladder and prostatic urethra
3. Co-existing tumours

Bladder Tumours

1. Chemical carcinogens; cigarette smoking (?)
2. Transitional cell carcinoma (TCC) most common, squamous cell carcinoma, adenocarcinoma
3. Papillary vs non-papillary tumours
4. Symptoms and signs
   - 75-80% painless, graoss haematuria
   - irritable bladder symptoms
   - Urinalysis - haematuria
   - IVP - bladder filling defect
   - cystoscopy - bladder tumour
5. Clinical staging for extent of disease
6. Treatment
   - local - non-invasive - transurethral resection
     intravesical chemotherapy
   - locally invasive - cystectomy and/or radiation
     ileal/gastric/colonic conduit
   - disseminated - palliative resection ± chemotherapy

Renal Pelvis and Ureter

1. Population at risk: phenacetin abusers, Balkan nephropathy, dye exposure, schistosomiasis and stones
2. Symptoms and signs
   - gross haematuria
   - colic due to clots
3. Treatment : nephroureterectomy
   segmental resection

III. PROSTATE GLAND

Surgical Anatomy

1. True prostate (surgical capsule)
2. Periurethral adenoma

Benign: Benign Prostatic Hyperplasia (BPH)

1. Hormonal milieu

2. Pathogenesis: outflow obstruction
   upper tract dilatation
   detrusor instablility

3. Sypmtoms
   Obstructive: hesistancy, intermittency, retention
   Irritative : frequency, nocturia, urgency, incontinence
   Infection
   Uraemia

4. Signs
   enlarged prostate on rectal examiration
   palpable bladder - acute or chronic

5. Management - relief of obstruction; when active, uroflow measurement
   prostatectomy: transurethral prostatectomy and bladder neck incision
   retropubic
   suprapubic
   perineal
   alpha-adrenergic blockers - temporary relief

Prostatic Carcinoma

1. Prevalence - less in Chinese

2. Clinical expression
   anatomy
   presentation - palpable nodule
   coincidental in prostatectomy
   bone pain

3. Management - staging and grading
   skeletal survey, bone scan, acid phosphatase
   (external and implants)
   locally advanced - XRT
   disseminated - hormonal (orchidectomy or DES)
   chemotherapy
   XRT for bone pain

Carcinoma of the Penis

1. Squamous cell carcinoma
2. Circumcision
3. Viral theory: cervical cancer
4. Surgical treatment: amputation
   lymph node dissection

Carcinoma of the Testis (Germ Cell Tumours)

1. Young adult males
2. Association with cryptorchidism
3. Arises from primordial germ cells
4. Seminoma vs non-seminomas (teratoma, choriocarcinoma, embryonal carcinoma)
5. Differential diagnosis - scrotal mass
6. Treatment - inguinal orchidectomy
   - surgery - retroperitoneal lymph node dissection
   - XRT - aortic lymph nodes
   - combination chemotherapy
7. Tumour markers - α FP, β HCG, CEA

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1. Johnson, D.E. and Boileau, M.A. 'Genitourinary Tumors', Grune and Stratton, 1982.

2. Smith, D.R. 'General Urology', Large Series, Chapter 18, 1981.

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
     VB1, VB2, EPS, VB3
     suprapubic aspiration
     catheterisation
2. Microscopy - pyuria/bacteriuria
3. Culture - 10^5 organisms/ml: significance
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 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

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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.

URAEMIA AND RENAL TRANSPLANTATION

I. 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 kidneys
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°C - 15°C	2 hours of ischaemia
15°C - 5°C	6 hours
5°C - 0°C	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

CyA
Co worker

Other Modalities

1. Antilymphocytic globulin
2. Total lymphoid irradiation
3. Thoracic duct drainage
4. Cyclosporin A
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

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 (20%) - gun shot
   stabbed by knife
   iatrogenic - percutaneous renal biopsy

2. Blunt (80%) - traffic accident
   contact sport
   falling from height

Pathology and Classification (Blunt Injury)

1. Renal Parenchymal
   (a) Contusion
   (b) Laceration - minor)
   major) 85%
   (c) Shattered kidney

2. Renal vascular (pedicle injury)

Clinical Features

1. History of injury
2. Haematuria (85%) 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

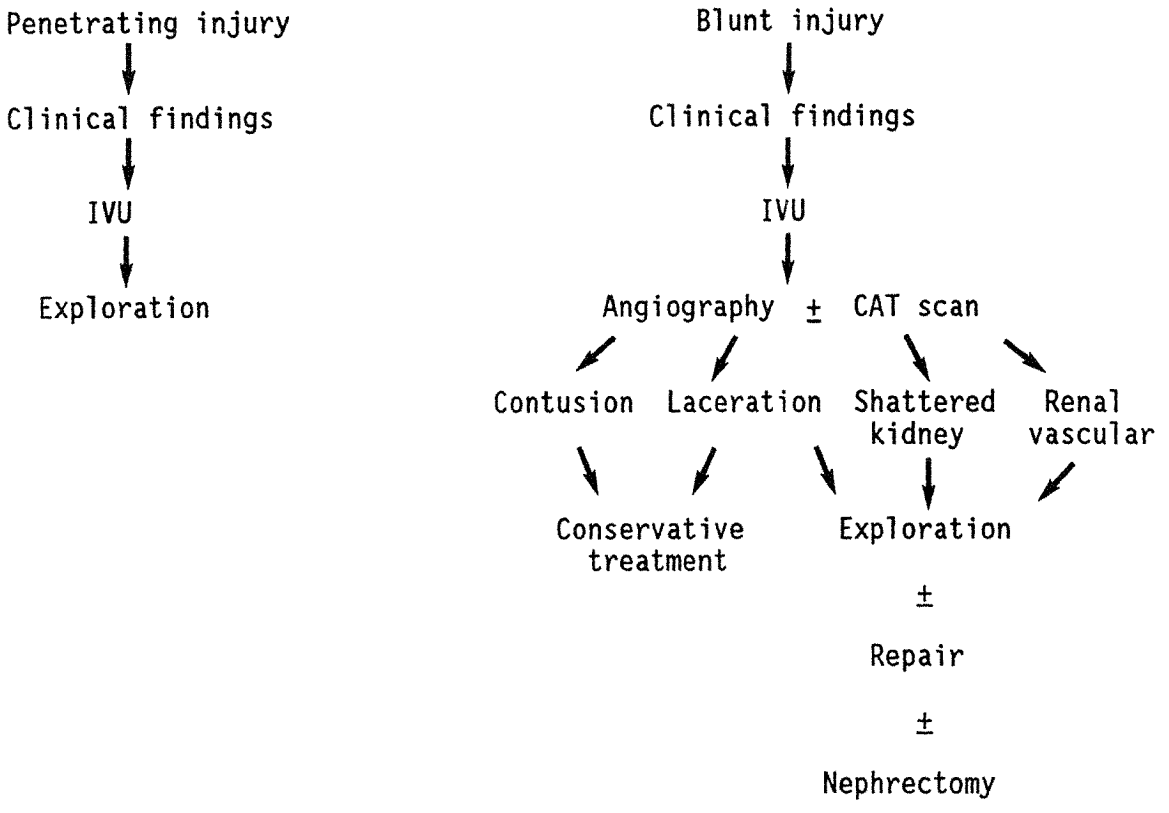
1. Urinalysis
2. haematocrit
3. Role of radiology - primary role in diagnosis and staging
   Plain film - KUB
   Intravenous urogram + tomography
   Renal angiography - equivocal IVU findings
   prolonged bleeding with known trauma
   suspected renal vascular injury
   evaluation of complications - A-V fistula
   arterial stenosis
   CAT scan

Treatment

Three governing factors:

1. Clinical parameters
2. Associated major injuries
3. Accurate diagnosis of extent of injury

Management Flow Chart



Conservative Treatment

1. Bed rest
2. Antibiotics
3. Blood transfusion
4. Serial monitor

Complications

1. Early (< 6 weeks)

   (a) Persistent or recurrent bleeding
   (b) Urinary extravasation (urinoma)
   (c) Abscess
   (d) fistula formation to bowel or skin

2. Late

   (a) Hypertension
   (b) Hydronephrosis
   (c) Traumatic A-V fistula

II. INJURIES TO URETER

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

Aetiology

1. Iatrogenic (70%) - operative trauma
   - pelvic surgery - hysterectomy
   - abdominal aneurysmectomy
   - spinal operations
   - endoscopic manipulation

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) extraperitoneal cellulitis
   (b) peritonitis
   (c) anuria or deterioration in renal function
   (d) urinary fistula to incision or vagina (75%) - after 1-2 weeks
   (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 technique

Surgical Options

1. Direct end-to-end anastomosis
2. Psoas hitch technique
3. Boari bladder flap
4. Transuretero ureterostomy
5. Renal vascular relocation (autotransplantation)
6. 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
   - direct blow to abdomen - intraperitoneal
   - pelvic fracture - incidence 10% (20% in pubic arch)
   80% extraperitoneal - antero-lateral wall
2. Penetrating injuries
3. Iatrogenic - pelvic surgery
   transurethral surgery

Clinical Features

1. History of injury
2. Haematuria
3. Lower abdominal pain
4. Abdominal distension ileus
5. Acute retention of urine
6. Hypovolaemia
7. Associated injury - musculo-skeletal

Investigations

1. Ascending urethrocystogram, if urethral injury is suspected.

2. Cystogram

   (a) "Tear drop" bladder - bladder compressed by haematoma but no perforation.
   (b) Intraperitoneal perforation with contrast in and around the gut.
   (c) Extraperitoneal perforation with pelvic extravasation.

Treatment

1. Bladder contusion by urethral catheter drainage

2. Surgical exploration for perforation
   debridement
   repair
   drainage of bladder and perivesical space
   deal with other organ damage

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

1. Fractured pelvic - injury to posterior urethra
2. Direct trauma to perineum
   blow from kick)
   straddle injury) injury to anterior urethra
   go-cart injury)
3. Iatrogenic - transurethral operation

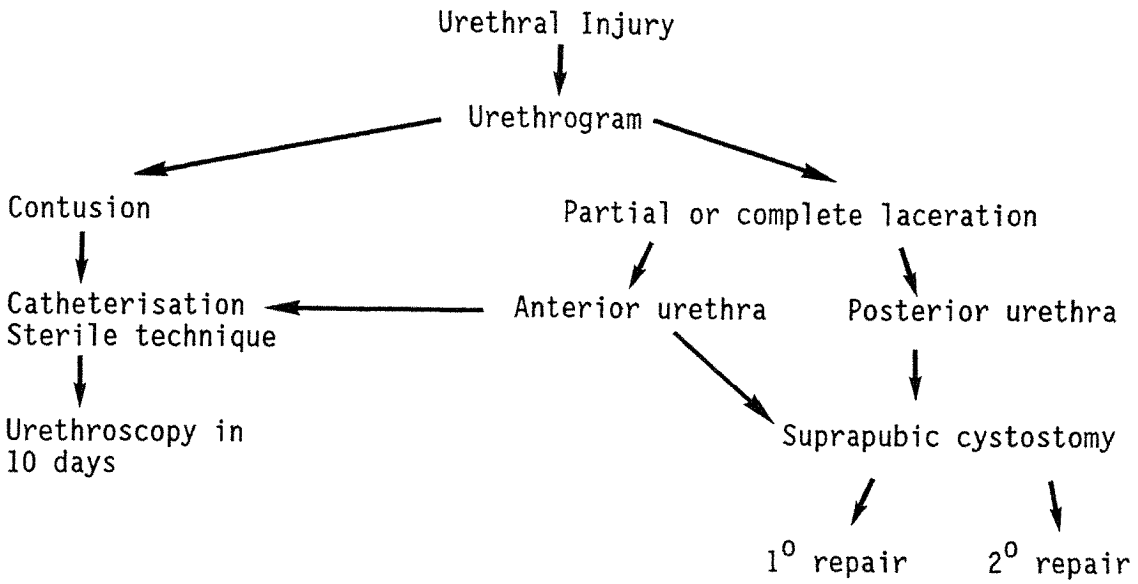
Clinical Features

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

Investigations

1. KUB - fractured pelvis
2. Urethrogram (ascending) - water soluble contrast
3. Diagnostic catheter - controversial

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.

CALCULOUS DISEASE OF THE GENITO-URINARY TRACT

Aetiology

1. Idiopathic calcium stones (calcium oxalate)
2. Urinary infection and stasis (triple phosphate)
3. Hyperparathyroidism (calcium phosphate)
4. Gout (uric acid)
5. Inborn errors of metabolism - cystinuria, oxalosis
6. Bladder stones of children - endemic (triple phosphate)

Composition - analysis by qualitative or quantitative methods

1. Calcium oxalate - commonest in Hong Kong
2. Calcium magnesium ammonium phosphate (triple phosphate)
3. Calcium phosphate
4. Calcium mixed stones from above
5. Cystine stones

Theories of Formation

1. Supersaturation of the calcium salts affected by other solutes and acid macromolecules
2. Dehydration and concentrated urine especially overnight
3. Hyper-excretion of urinary constituents in metabolic disorders and hypercalciuria
4. Pre-existing renal damage, stasis or damage due to refined carbohydrates leading to fixed particles of tissue debris which forms the nidus of crystal aggregation
5. Crystal growth - epitaxis and aggregation
6. Inhibitors of crystallization - ionic inhibitors and macromolecules
7. Infection with urea-splitting organisms - Proteus
8. Metabolic defects of the renal parachyma - medullary sponge kidneys and renal tubular acidosis
9. Malabsorption and ileal/colonic conduits

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

Managements - 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. Conservative measures
   (a) hydration and diuresis
   (b) analgesics and anti-spasmodics

3. Treatment for renal stones
   (a) extra-corporeal shockwave lithotripsy
   (b) percutaneous nephrolithotripsy
   (c) pyelolithotomy and nephrolithotomy as reserve

4. Treatment for ureteric stones
   (a) extra-corporeal shockwave lithotripsy
   (b) ureteroscopic lithotripsy
   (c) endourological pushing and basketing
   (d) ureterolithotomy as reserve

5. Treatment for bladder stones
   (a) mechanical endoscopic litholapaxy
   (b) ultrasonic and electrohydraulic lithotripsy
   (c) suprapubic lithotomy
   (d) treatment of obstruction such as TUR prostate

Follow-up of Stone Patients

1. Dietary advice to minimize recurrence
   (a) Drink sufficient water to maintain urine volume at 3 litres (6 pints) per 24 hours
   (b) Restrict calcium-rich items
      - milk and its products including custard, milk puddings, yogourt, cheese. Tea and coffee may however be taken with small quantities of milk
      - medications which contain calcium or raise urinary calcium must be avoided. These include calcium aspirin, many insoluble indigestion mixtures and antacids. Magnesium preparations are useful alternatives and are good
   (c) Restrict oxalate-rich foods
      - completely forbidden foods are : rhubarb, spinach, beetroot, strawberries, nuts and chocolate
      - tea is allowed in moderation, say two cups per day. Coffee made with beans is satisfactory, but instant coffee is to be avoided
      - large doses of vitamin C are forbidden, but normal quantities in natural foods are allowed
   (d) Restriction of refine carbohydrates
      - sugar in tea or coffee, soft drinks
      - sweets
      - natural sugar is allowed e.g. fruits

2. Drug treatment for recurrent stone formers
   (a) thiazides for hypercalciuria
   (b) mandelamine to acidify urine
   (c) allopurinol
   (d) magnesium salts
   (e) antibiotics for repeated infections
   (f) cellulose phosphates
   (g) long-term antibiotics or antiseptics
   (h) high dose pyridoxine for oxalosis

3. Regular 24-hour urine analysis for risk factors and monitor of control of calcium output, MSU cultures

4. Regular KUB and IVU for stone recurrence.

NEUROGENIC BLADDER

Definition

Disorder due to functional disturbance of micturition which may result in anatomical and renal damage.

Classification I

1. Pure sensory
   diabetes mellitus, tabes dorsalis
2. Pure motor
   amyotrophic lateral sclerosis
3. Mixed
   acquired (spinal trauma and tumours)
   congenital
   idiopathic (commonest in urological practice)

Classification II

1. Upper motor neuron
   - no voluntary control
   - reflex emptying at high pressure
   - reduced capacity
   - hypertrophied wall with ureteric obstruction
   - spastic bladder
   - residual urine variable
   - upper tract dilatation

2. Lower motor neuron
   - no voluntary control
   - overflow incontinence
   - large capacity at low pressure
   - thin bladder wall
   - atonic bladder
   - large volume of residual urine

Aetiology

1. Idiopathic unstable bladders
2. Post-pelvic operation e.g. AP resection, hysterectomy
3. Spinal trauma
4. Spinal tumour with compression
5. Degenerative nervous disorders - neruopathies
6. Prolapsed intervertebral disc
7. Myelomeningocele
8. Drug induced instability e.g. hypertension, Parkisonism

Presentation - most variable from asymptomatic to total loss of renal function

1. Recurrent urinary infection
2. Frequency and nocturia, enuresis
3. Urgency incontinence
4. Difficult micturition and retention
5. Overflow incontinence and dribbling
6. Features of end stage renal failure
7. History of trauma/operation to spine

Presentation after Spinal Injury

1. Phase of spinal shock - initial 3 months
   - retention of urine
   - catheterization-intermittent or continuous
   - prevent infection
   - wait for recovery

2. Phase of recovery - 3 months after injury
   - assessment of bladder function can be done
   - full investigations
   - treatment to provide "normal" function

Investigations

1. Urine culture, renal function test
2. Intravenous urogram for hydronephrosis, pyelonephritis and stone formation
3. Voiding cystogram for reflux in ureters, bladder diverticuli and emptying
4. Complete urodynamic workup to look for
   (a) activity and pressure of detrusors with respect to volume
   (b) sphincter function, control and co-ordination to detrusor contraction

Management - tailored to individual patient
- a combination of treatment is usually required

1. Detrusor hyperactivity detected
   - atropine-like agents
     - propantheline
     - hyoscyamine
     - isopropamide
   - anti-cholinergic agents
     - oxybutynin (Ditropan)
     - probanthin
   - tricyclic anti-depressants
     - imipramine
   - cystodistension
   - cystoplasties (gastric, colonic, ileal)

2. Detrusor hypoactivity/dysreflexia detected
   - decrease outflow resistance - phenoxybenzamine
     - prazosine
     - TUR bladder neck
   - parasympathomimetic - urocholine
   - catheter drainage - intermittent
     - continuous
   - manual compression/reflex emptying
   - implanted electrodes
   - replacement of bladder outflow by artificial sphincters after resection

3. Results of treatment aimed at
   - good storage
   - effective emptying
   - reduce residual urine
   - minimize infection

4. When all measures failed and with increasing hydronephrosis
   - urinary diversion - conduits
   - bladder replacement with artificial sphincters

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.

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 not 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.)

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)

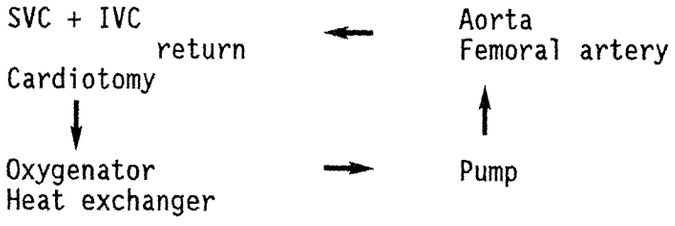
Pump

- Pulsatile flow
- Non-pulsatile flow

Oxygenator

- Film
- Bubble
- Membrane

Extracorporeal Circulation Circuit



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

NON-MALIGNANT DISEASES OF THE BREAST

Congenital Anomalies

1. Accessory nipples
2. Accessory breast
3. Absence of breast

Acute Mastitis

1. Pubertal mastitis
2. Mastitis of mumps
3. Mastitis from local irritation
4. Mastitis from milk engorgement
5. Bacterial mastitis

Breast Abscess

1. Intramammary abscess
2. Subareolar abscess
3. Chronic intramammary abscess
4. Chronic subareolar abscess (mammillary fistula)
5. Tuberculous breast abscess
6. Actinomycosis of breast
7. Retromammary abscess

Fibroadenosis

May occur at any age after puberty.

Pain is felt usually in both breasts just before period time; the breasts feel nodular especially in the upper outer quadrants. The pathological changes are:

1. Adenosis
2. Epitheliosis
3. Fibrosis
4. Papillomatosis
5. Microcyst formation

There is controversy as to whether fibroadenosis is a pre-cancerous condition.

Benign Tumours of the Breast

1. Fibroadenoma
   (a) Pericanalicular type in the younger patient
   (b) Intracanalicular type in the older patient.
   Treatment is simple excision

2. Giant fibroadenoma - this is really a large fibroadenoma in a young patient. It is not to be confused with cystosarcoma phyllodes.

3. Cystosarcoma phyllodes - occurs in middle aged women. These are large bulky tumours. Occasionally some turn malignant. Treatment is excision through a submammary incision.

4. Duct papilloma - These arise from the ducts of the breast and usually from larger ducts beneath the areola. A bloody discharge from the nipple is usually present. A sub areolar lump may also be present. Treatment is microdochectomy. If papillomatosis is suspected, excise major duct system.

5. Adenoma of the nipple - This is a rare benign tumour.

Traumatic Fat Necrosis

Can be confused with breast cancer. Usally seen in fat pendulous breasts with a history of trauma and bruising. Mammographic appearance can resemble carcinoma.

Mammary Duct Ectasia (Plasma Cell Mastitis)

The clinical appearance may resemble carcinoma (nipple retraction, hard mass, etc.). It usually presents with nipple discharge and a periareolar area of pain, tenderness redness.

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.

CARCINOMA OF THE BREAST

Mortality from breast cancer in the local population is low compared to that of Western countries. It has been recognised (from clinical studies) that there are certain factors which make some women more prone to develop breast cancer. These are:

1. Increasing age
2. A history of breast cancer in the family
3. Nulliparous women
4. Women whose first pregnancy is after the age of 30
5. Those who have early menarche
6. Those who have late menopause
7. A woman with cancer in one breast is at a higher risk in developing a second cancer in the opposite breast
8. Other associations with irradiation to the breast, hypothyroidism.

There is some evidence that those who start using the contraceptive pill at a young age (teen-age) may have a higher risk of developing breast cancer.

There are three types of breast cancer

1. Carcinoma arising from the nipple (Paget's disease)
2. Carcinoma arising from the ducts
3. Carcinoma arising from the lobules

Clinical modes of presentation of breast cancer

1. Lump in the breast
2. Pain in the breast or a painful lump in the breast
3. Blood-stained nipple discharge
4. Ulceration of the nipple or an eczema-like lesion
5. An axillary mass
6. Unusually as a pathological fracture, pleural effusion, lymphoedema of arm, back pain or girdle-type pain
7. May present as recurrent breast cancer following earlier treatment
8. Rarely the male breast may develop cancer

Some of the histological types of breast cancer are

1. Atrophic scirrhous
2. Papillary type
3. Paget's Disease
4. Mastitis carcinomatosa
5. Encephaloid type
6. Scirrhous type

Spread of breast cancer

1. Locally in breast to skin, pectoral muscle and chest wall
2. Lymphatic spread to the internal mammary and axillary lymph nodes; later to supraclavicular lymph nodes.
3. Blood stream spread to lungs, liver, brain and bones

Investigations in breast cancer

1. Mammography - irregular mass, ill defined margins, microcalciaction and increased venous markings around tumour
2. Fine-needle aspiration cytology
3. Tru-cut needle biopsy
4. Radiology of skeleton or bone scans
5. Ductograms
6. Thermography
7. Xeroradiography
8. Oestrogen receptor assay - 40-60% of tumours positive for tumours respond to hormonal manipulation

Once the diagnosis of breast cancer has been confirmed by histology, it is necessary to stage the disease. The TNM staging system is currently in favour.

Treatment is controversial. Recent trends have been:

1. To do less radical surgery on the breast

2. To recognise that breast cancer is a systemic illness in which early dissemination occurs. This has led to the concept of adjuvant chemotherapy in place of adjuvant radiotherapy.

Stage I
1. Simple mastectomy and biopsy of axillary gland
2. Partial mastectomy and axillary clearance

Stage II
1. Modified radical mastectomy. If axillary glands are positive, then adjuvant therapy is indicated. In pre-menopausal women, this will be adjuvant chemotherapy. In post menopausal women it will be tamoxifen 20 mg. bid.
2. Partial mastectomy and axillary clearance and RT

Stage III & IV Palliative surgery on the breast followed by regional radiotherapy. Some form of systemic treatment such as hormonal manipulation or chemotherapy depending on oestrogen receptor status. Local radiotherapy is good palliation for bone metastases.

CHEMOTHERAPY OF TUMOURS

I. HISTORIC PERSPECTIVES

Paul Ehrlich - father of chemotherapy.

Three major pieces of work in cancer treatment research in the early 1900's:

1. Halsted - en-bloc resection as part of cancer operation.
2. Roentgen - discovery of X-rays.
3. Clowles - development of inbred rodent models carrying transplanted tumours.

1943: Alkylating agents used in lymphomas

II. BIOLOGICAL PRINCIPLES

Principles developed with rodent leukaemia L1210

- 100% growth fraction
- 60% synthesising DNA
- life cycle consistent and predictable

These principles not entirely applicable to human tumours

- growth heterogenous
- life cycle prolonged
- low percentage synthesising DNA
- many cells are resting

Growth fraction - the fraction of cells in a tumour mass actively dividing.

Principles of Chemotherapy and Implications

1. Resting cells are resistant to most chemotherapeutic agents, only the growth fraction respond.
   Implication - a fraction of cells are consistently not killed.

2. The larger the tumour bulk, the lower the growth fraction.
   Implication - the larger the tumour, the lower the response

3. Micrometastases have a higher growth fraction.
   Implication - response to chemotherapy is improved if the primary growth (bulk) is removed (by surgery or irradiation).

4. Resting cells may resume DNA synthesis and growth.
   Implication - cancer is difficult to eradicate, mechanism of relapse.

5. Minimal cytotoxic concentration of most drugs for neoplastic and normal cells are the same when their growth fraction is similar.
   Implication - fine line between fatal toxicity and response for sensitive tumours, toxicity without benefit in non-responsive tumours.

6. A small fraction of cancer cells are specifically resistant to a given drug.
   Implication - the larger the bulk, the greater the chance of resistance.

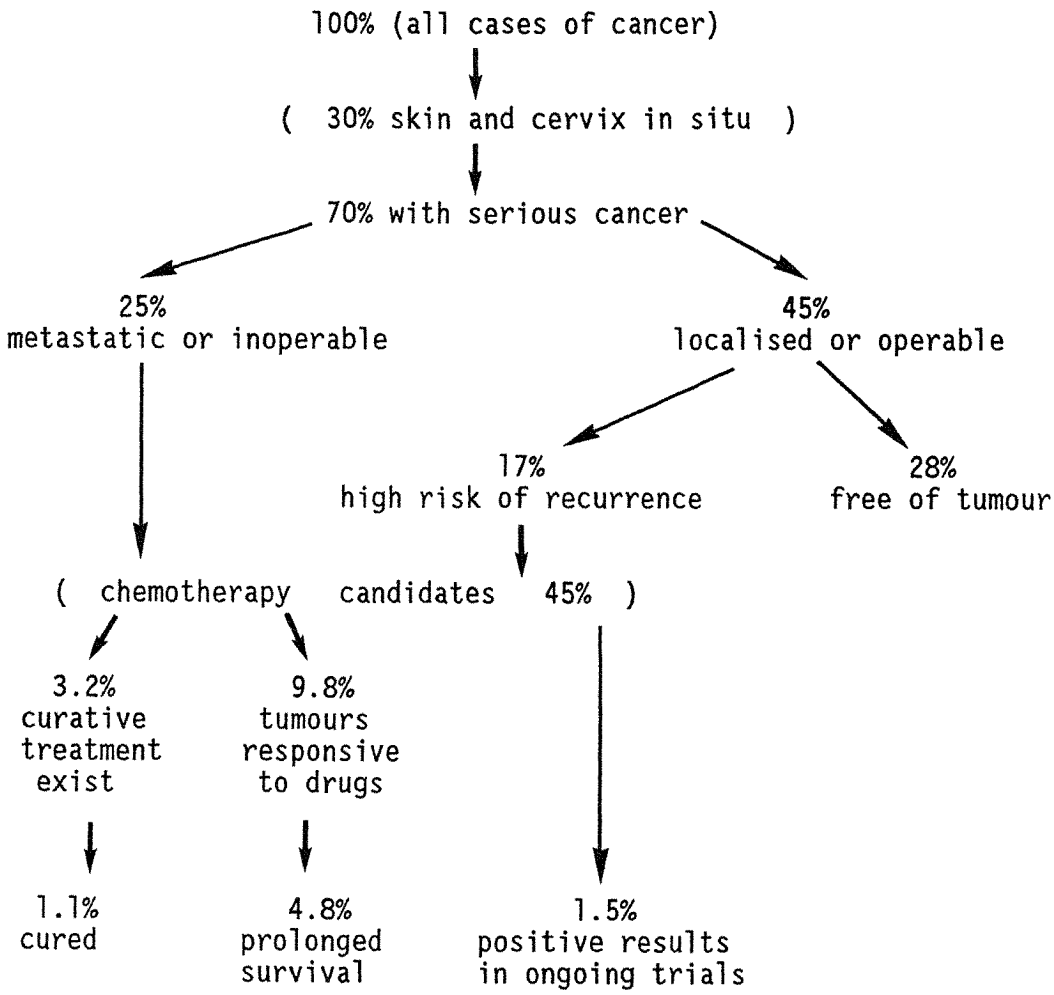
7. Neoplastic cells mutate spontaneously to a state of specific resistance to a wide variety of cancer drugs.
   Implication - mechanism of relapse, relapsed tumour mostly resistant.

III. CLINICAL APPLICATIONS OF CHEMOTHERAPY

Chemotherapy is used in:

1. Patients with cancers where drugs are the mainstay of treatment.
2. Patients presented with metastasis or inoperable cancer
3. Patients with a high risk if recurrence after local therapy.
4. Combination with other modalities of treatment for specific cancers.

Cancer Data



Cancers Curable by Chemotherapy (Overall 25%)

Choriocarcinoma
ALL
Hodgkin's disease
Diffuse histiocytic lymphoma
*Nodular mixed lymphoma
Testicular carcinoma
*Ovarian carcinoma
*AML
Wilms' tumour
*Burkitt's lymphoma
Embryonal rhabdomyosarcoma
Ewing's sarcoma

*combined with other forms of treatment

Cancers with Low Response to Chemotherapy
(Fraction of Patients with Increased Survival)

Breast carcinoma
CML
CLL
Nodular poorly differentiated
Lymphocytic lymphoma
Multiple myeloma
Oat cell carcinoma of lung
Sarcomas
Gastric carcinoma
Insulinoma
Endometrial carcinoma
Adrenal cortical carcinoma
Medulloblastoma
Neuroblastoma
Prostatic carcinoma
Glioblastoma

Cancers Marginally Responsive to Chemotherapy
(No Demonstrable Improvement in Survival)

Hepatocellular carcinoma
Head and neck carcinoma
Oesophageal carcinoma
Colorectal carcinoma
Hypernephroma
Malignant melanoma
Thyroid carcinoma
Penile carcinoma
Bronchogenic carcinoma
Malignant carcinoid

Chemotherapeutic Agents

1. Drugs
2. Hormones
3. Immune modulators

Drugs

Structural analogues
- Methotrexate
- Fluorouracil
- Mercaptopurine

Alkylating Agents

- Nitrogen mustard
- Cyclophosphamide
- Chlorambucil
- Phenylalanine mustard

Thiotepa

- Vinca alkaloids:
- Vincristine
- Vinblastine

Cytotoxic Antibiotics

Dactinomycin (Actinomycin)
Doxorubicin
Bleomycin
Mitomycin C

Inorganic Metal Salt

Cisplatinum diaminodichloride

Nitro-ureas

BCNU
CCNU
Methyl CCNU
Streptozotocin

Miscellaneous

Procarbazine
Hydroxyurea

Combination Chemotherapy

1. Avoid overlapping of toxicity
2. Blocking multiple biosynthetic pathways - synergistic effect

Immunotherapy

Three types of agents:

1. Generalised immunological stimulation
   - BCG, conA, levamisole
2. Causes intense local inflammatory reaction
   - intralesional BCG
3. Specific immunotherapy
   - killed tumour cells, tumour antigens

Hormones

Oestrogens
Androgens
Progesterone
Corticosteroids
Thyroxine

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.