THE UNIVERSITY OF HONG KONG

Department of Surgery

LECTURES FOR MEDICAL STUDENTS

VOLUME 1 August 1994

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SURGERY AND SURGICAL METHODS

WHAT IS SURGERY?

Branch of medicine employing manual and operative skills.

WHO ARE SURGEONS?

Doctors with such skills.

WHAT DO SURGEONS DO?

Treat patients, with surgery if necessary.

WHAT BROAD GROUPS OF OPERATIVE PROCEDURES DO SURGEONS DO?

Remove tissues/organs.
Rejoin tissues/organs.
Replace tissues/organs.

WHAT ARE THE BASIC OPERATIVE PROCESSES?

To cut or separate (incise).
To repair or join (suture).

WHAT DO WE USE TO CUT?

Scissors
Knife
Shears
Chisel and hammer
Saw
Drill
Diathermy
Laser

WHAT DO WE USE TO REPAIR?

Needle and thread
Clips
Staples
Glue

HOW DO WE STOP BLEEDING?

With ligature
With diathermy
With pressure

HISTORY TAKING

1. Aim

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

2. Principles

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

3. Format

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

a. Presenting Complaint

Define the main complaint and its duration.

b. History of the Present Illness

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

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

c. Past Health

i. All important previous illnesses should be noted

ii. Previous drug treatment, surgery, radiotherapy and psychotherapy

iii. Adverse reactions to drugs - hypersensitivity

d. Menstrual History

i. Menarche Duration Cycle Menopause

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

iii. Oral contraceptive

e. Family History

i. Patient's position in the family

ii. Ages of the children

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

f. Social and Occupational History

i. Exact nature of his/her occupation

ii. His/her home surroundings

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

iv. Whether or not he/she has lived abroad

TUMOURS OF THE ORAL CAVITY AND PHARYNX

PREMALIGNANT LESIONS

1. PRECANCEROUS LESIONS

- Leucoplakia
- Erythroplakia

2. PRECANCEROUS CONDITIONS

- Syphilis
- Sideropenic dysphagia
- Oral submucous fibrosis

3. LEUCOPLAKIA

Aetiology

- Tobacco
- Candida
- Virus
- Alcohol

Histology

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

Common Sites

- Buccal mucosa
- Labial commissure
- Labial mucosa

Factors Influencing Malignant Potential

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

Treatment

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

4. COMPARISON OF MALIGNANT POTENTIAL

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

5. ERYTHROPLAKIA

Features

- Bright red
- Velvety

Common Sites

- Floor of mouth
- Tongue

ORAL CANCER

1. TYPES OF ORAL CANCER

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

2. AETIOLOGY

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

3. FEATURES

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

4. TENDENCY TO METASTASISE TO REGIONAL LYMPH NODE

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

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

5. CLINICAL DETECTION OF LYMPH NODE METASTASIS

- Reliability - 70-80%
- Tongue - worse

6. EFFECT OF NODE METASTASIS ON SURVIVAL

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

7. T CLASSIFICATIONS

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

8. N CLASSIFICATION

- N0 - not palpable
- N1 - mobile - homolateral
- N2 - mobile - contralateral
- N3 - fixed

NEW CLASSIFICATION

- N0 - not palpable
- N1 - up to 3 cm
  single
  unilateral
- N2 - 3 to 6 cm
  multiple
- N3 - more than 6 cm
  bilateral

9. M CLASSIFICATION

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

10. STAGE-GROUPING

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

11. MANAGEMENT OF ORAL CANCER

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

12. OTHER METHODS OF TREATMENT

- Chemotherapy
- Cryosurgery

13. RECURRENCE PATTERN

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

14. CARCINOMA OF LIP

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

15. CARCINOMA OF FLOOR OF MOUTH

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

16. CARCINOMA OF ALVEOLUS

- Mandible 4 times
- Loose teeth
- Bony destruction

17. CARCINOMA OF BUCCAL MUCOSA

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

18. CARCINOMA OF TONGUE

Site - Anterior lesion : posterior lesion = 4:1
- Edge 50%
- Dorsum 20%
- Undersurface 15%
- Extensive 15%

Presenting symptoms - mass or ulcer of tongue 90%
- difficulty in swallowing 5%
- cervical lymphadenopathy 5%
- ear pain
- bleeding
- hoarseness

TUMOURS OF ORO- AND HYPOPHARYNX

1. OROPHARYNGEAL CANCER

Anatomy

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

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

Clinical Features - dysphagia, hoarseness, pain
indirect laryngoscopy

2. HYPOPHARYNGEAL CARCINOMA

Anatomy and Relative Site Incidence

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

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

Pathology - squamous carcinoma

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

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

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

3. TREATMENT OF ORO- AND HYPOPHARYNGEAL CANCER

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

DISEASES OF THE SALIVARY GLANDS

Major salivary glands - parotid, submandibular, sublingual

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

Diseases
- Inflammatory diseases and stones
- Tumours

I. NON-OBSTRUCTIVE SIALADENITIS

1. Acute suppurative parotitis

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

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

Pathogen - staph. aureus

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

2. Recurrent Sialadenitis

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

Sialogram - chronic distension of peripheral duct system (sialectasis)

Treatment
- treat superimposed infection
- surgical excision if frequent suppuration

3. Acute viral parotitis (mumps)

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

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

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

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

Treatment - no specific treatment

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

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

Associated with rheumatoid arthritis
polyarteritis nodosa
SLE
Hashimoto's thyroiditis

An autoimmune disease
Affects middle aged women

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

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

II. OBSTRUCTIVE SIALADENITIS

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

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

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

2. Ductal strictures

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

   Treatment - dilatation with lacrimal dilators
   - surgical excision of gland

III. Tumours

Common benign tumours
- pleomorphic adenoma
- Warthin's tumour

Malignant tumours
- adenoid cystic carcinoma
- muco-epidermoid tumour
- adenocarcinoma
- squamous carcinoma
- malignant pleomorphic adenoma

Tumours of variable malignancy
- acinic cell tumour
- oncocytoma

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

1. Benign tumours

a. Pleomorphic adenoma (benign mixed cell tumour)

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

Usual site - parotid just below ear lobe
Smooth, superficial, round and mobile
Encapsulated, but small excrescences may project from capsule
Sometimes multilobulated
Whitish yellow

Histologically, both epithelial and mesenchymal elements present. Epithelial element consists of small basophilic cells arranged in trabecular pattern, mesenchymal stroma may be myxoid, fibroid, chondroid or combination

The cartilaginous stroma is believed to be mucin secreted by the myoepithelial cells.

Treatment - superficial parotidectomy

Recurrence after primary surgery 5%

b. Warthin's tumour (papillary cystadenoma lymphomatosum, adenolymphoma)

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

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

Histologically composed of proliferating salivary gland cells in lymphoid tissue

Treatment - superficial parotidectomy

Recurrence rate - very low

2. Malignant Tumours

a. Adenoid cystic carcinoma

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

Signs of malignancy
- pain
- rapid enlargement
- fixation
- facial nerve paralysis

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

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

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

b. Muco-epidermoid carcinoma

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

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

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

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

c. Adenocarcinoma

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

d. Squamous cell carcinoma

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

e. Malignant pleomorphicadenoma

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

3. Tumours of variable malignancy

a. Acinic cell tumour

Uncommon
Exclusively affects parotid
Tumour cells resemble normal acinar cells with variable lymphoid tissue
Benign and malignant varieties

b. Oncocytoma

Uncommon
Also affects parotid only
Oncocytes are derived from intralobular ducts or acini
Benign and malignant varieties

4. Approach to parotid swellings

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

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

Bimanual palpation - for stones and deep lobe tumours

Duct orifice - stones
discharge

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

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

Stage I : T_1 N_0

Stage II : T_2 N_0

Stage III : T_3 or any tumour size with N_1

6. Biopsy

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

b. Fine needle aspiration cytology sometimes helpful.

7. Treatment of parotid tumours

Benign parotid tumours -

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

Total conservative parotidectomy -
Deep lobe tumour
Conservation of facial nerve

Malignant parotid tumour -

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

Malignant parotid tumour

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

Submandibular gland tumour

More likely to be malignant but usually encapsulated
Remove entire gland

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

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

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

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

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

DISEASES OF THE OESOPHAGUS

I. SURGICAL ANATOMY OF THE OESOPHAGUS

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

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

II. SYMPTOMS AND SIGNS

Main symptoms - dysphagia (distinguish from oropharyngeal cause)
Other symptoms - pain, hoarseness, regurgitation, haematemesis and constitutional upset.
Minimal local physical signs except in advanced carcinoma when lymphadenopathy, hepatomegaly and ascites may be present.

III. INVESTIGATIONS

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

IV. CAUSES OF DYSPHAGIA

(2 types : oropharyngeal and oesophageal)

Oesophageal causes:

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

V. SOME COMMON DISORDERS OF THE OESOPHAGUS

1. Foreign body e.g., Fish bone, chicken bone, coins and denture

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

Treatment - oesophagoscopic removal.

2. Perforation

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

Treatment: Nil by mouth
Drainage of abscesses
Surgical repair

3. Caustic stricture

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

4. Achalasia (cardiospasm)

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

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

VI. OTHER DISORDERS OF THE OESOPHAGUS

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

VII. TUMOURS OF THE OESOPHAGUS

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

VIII. CANCER OF THE OESOPHAGUS

Level of oesophageal tumour

Cervical	C6 - T1	
Thoracic	upper third	T2 - T4
	mid-third	T4 - T8
	lower-third	T8 - oesophageal hiatus
Abdominal	oesophageal hiatus to gastroesophageal junction	

Distribution

Mid-third thoracic	(50%)
Lower-third + abdominal	(30%)
Upper-third + cervical	(20%)

Macroscopic

Mucosal irregularities, ulcer, stricture or fungating mass

Microscopic

Main: Squamous cell carcinoma, adenocarcinoma, or anaplastic small cell carcinoma
Others: Mucoepidermoid, adenoid cystic, adenosquamous or pseudosarcoma

Spread

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

Clinical features of carcinoma of oesophagus:

a. Usually elderly males (50-80 years of age) with rapidly progressing dysphagia
b. Pain, hoarseness, lymphadenopathy, irritating cough, weight loss
c. Features of metastasis to lung, bone and liver

Investigations

Aim:
a. Establish a firm diagnosis
b. Assess the extent of the tumour growth
c. Assess the general state of the patient

Method:
a. Barium swallow
b. Oesophagoscopy and bronchoscopy with biopsy
c. CXR, CT scan
d. Cardiopulmonary and nutritional work-up

Treatment

Aim:
a. Curative for early tumour by complete removal
b. Palliative for more advanced tumour to allow patient to eat

Method:
a. Resectable lesions - oesophagectomy and reconstruction
b. Non-resectable lesions - bypass operation, intubation or laser therapy
c. Radiotherapy for those refusing surgery or as adjunct to surgery
d. Chemotherapy - of questionaller benefit

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
- Thyroglobulin
- Antibodies
- Fine Needle Aspiration Cytology
- Isotope Scan
- X-ray Thoracic Inlet
- Ultrasound
- Preoperative direct laryngoscopy

Management

- Surgery
- Medication
- (T_4, Anti-thyroid drugs, Beta-blockers)
- Radioactive 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 tracheal 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 :-

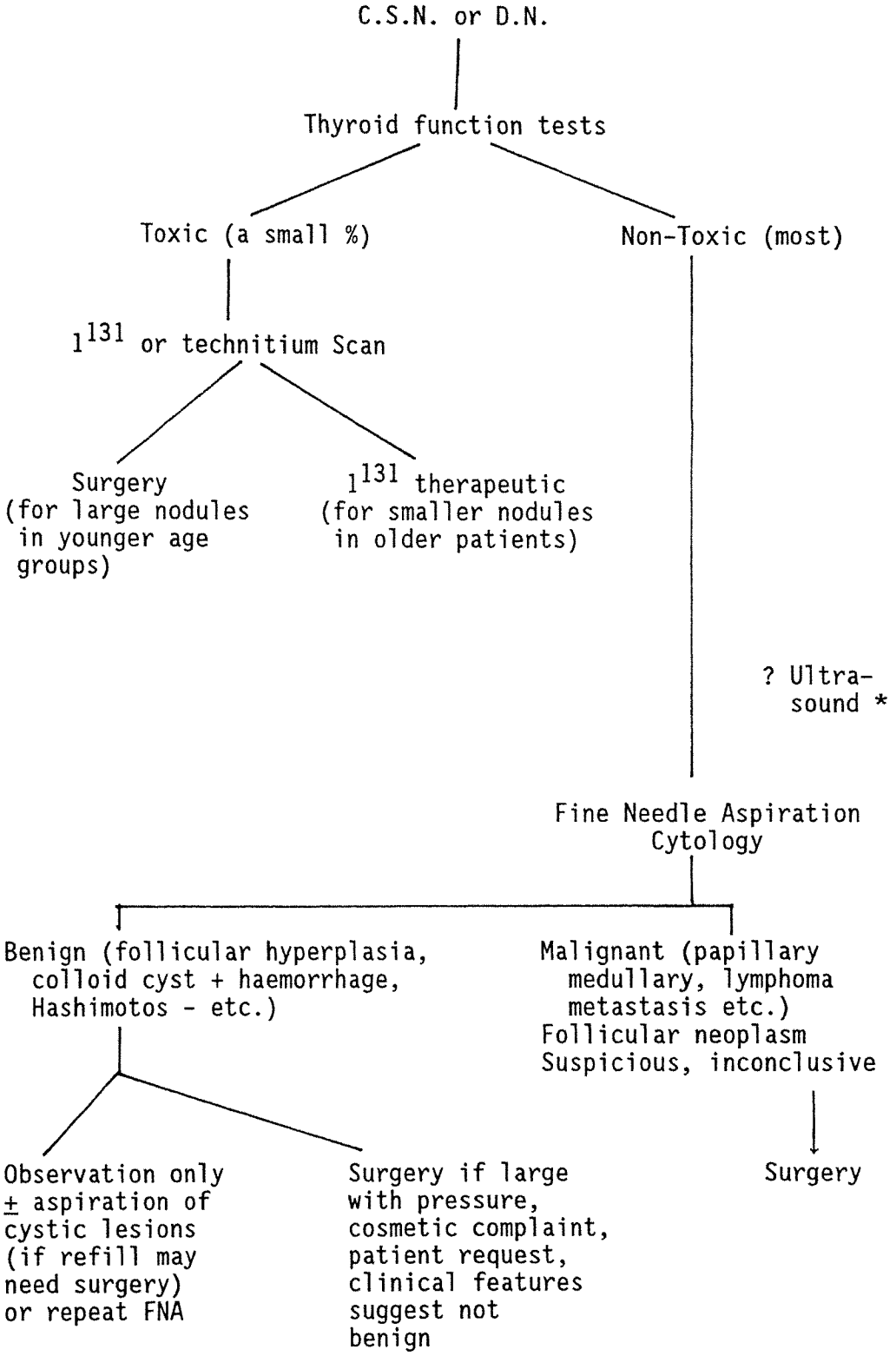
(a) An area of colloid cystic degeneration and haemorrhage as part of nodular goitre (± 70%)

(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 differentiated 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

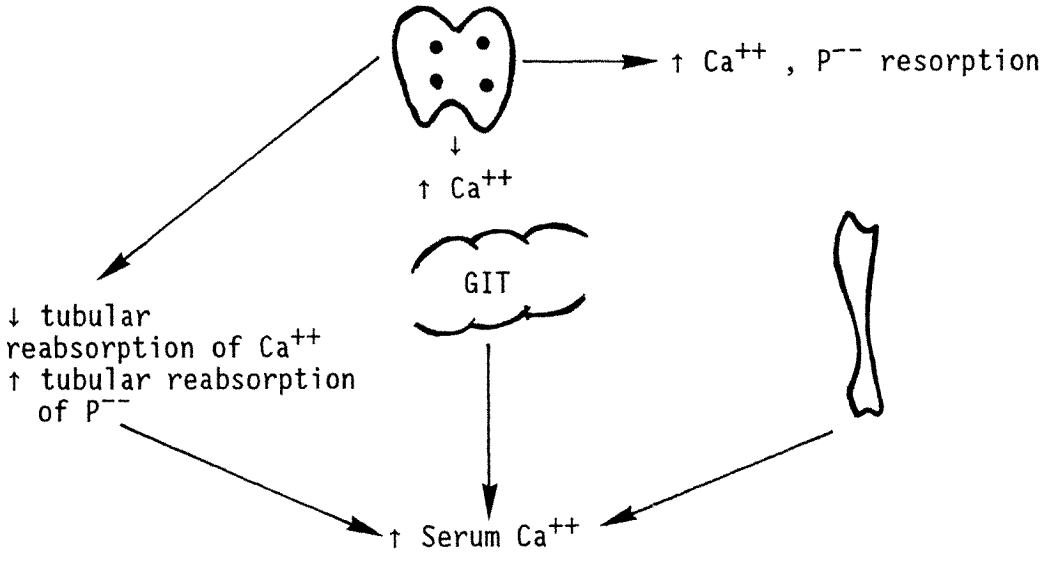
1. Ovoid, flattened, smooth, tan-coloured, 15-40 mg each
2. Superior parathyroids - 4th pharyngeal pouch
3. Inferior parathyroids - 3rd pharyngeal pouch

Regulations of Calcium Homeostasis

1. Vitamin D 25.OH cholecalciferol → 1, 25 diOH cholecalciferol
   (liver) (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^{--}, ↑ circulating PTH level
   ↑ 24 hr urine calcium
   mild hyperchloraemic metabolic acidosis
   ↑ A.P.
3. Radiological evidence
   - skull, long bones, clavicles, dental

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

Anatomical Localization

1. CT scan
2. Adrenal arteriogram
3. Adrenal venography + venous sampling

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 - cortisol 20 mg a.m.
   10 mg p.m.
   9 α -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)

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

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

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

IV. ISLET CELL TUMOURS OF THE PANCREAS

Types

All Types	Secretion	Syndrome
α	Glucagon	Hyperglycaemia
β	Insulin	Hypoglycaemia
Δ	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

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 & II
1. Partial mastectomy, axillary clearance, and RT
2. Modified radical mastectomy

Further treatment : If axillary nodes are involved by tumour, adjuvant treatment is indicated. This is usually chemotherapy for premenopausal women and tamoxifer 20mg b.i.d. for post menopausal women who are ER positive. Post menopausal ER negative women need chemotherapy.

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.

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 breast 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. There 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.

Cysts of the Breast

1. Usually in middle aged patients.
2. Usually painless. 25% associated with pain.
3. Lump change in size with periods.

Treat by aspiration. Surgical excision only if (a) a residual lump is present after aspiration; (b) aspirate is blood stained (send fluid for cytology); and (c) recurs after two aspirations.

Breast Pain (Mastalgia)

1. Fibrocystic disease
2. Trigger spot pain
3. Trauma
4. Tietze's syndrome
5. Sclerosing adenosis
6. Carcinoma of breast
7. Miscellaneous (pregnancy, oral contraceptive, fibroadenoma)

Mammary Duct Ectasia (Plasma Cell Mastitis)

Commonest in 40-45 years old age group. Affects all ducts to a varying degree. Ducts are filled with white, green or brown secretion. Periductal mastitis is present.

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. Anti-inflammatory drugs will help surgery if copious and persistent discharge persists or if a para areolar mass is present; biopsy must be done to exclude malignancy.

Traumatic Fat Necrosis

Can be confused with breast cancer. Usually seen in fat pendulous breasts with a history of trauma and bruising. Mammographic appearance can resemble carcinoma. Excisional biopsy is essential to exclude breast cancer.

Mammillary Fistula

Could present as a chronic discharging fistula either unilateral or bilateral. It could also present as a recurrent subareolar abscess. The fistula arises from a single abnormal duct, or from ducts damaged by periductal mastitis. Treatment is to lay open tract and allow it to granulate. Antibiotics of no use.

Galactocoele

Occurs during or after lactation. It is usually a solitary cyst containing milk either liquid or inspissated. Majority can be treated by aspiration.

Gynaecomastia

1. Idiopathic
2. Drugs (digitalis, spironolactone, I.N.A.H., steriod, phenothiazine, amphetamine, androgens)
3. Puberty
4. Oestrogen therapy
5. Cirrhosis of liver
6. Testicular failure (Kline Felter's syndrome, testicular agenesis, bilateral cryptorchidism, severe bilateral orchitis)
7. "Refeeding" gynaecomastia
8. Endocrine disorders (testicular tumours, adrenal carcinoma, hyper- and hypo- thyroidism, acromegaly, diabetes mellitus, Addison's disease, pituitary tumours
9. Non-endocrine tumours - Ca bronchus, renal Ca, Hodgkin's disease

Treatment is to ascertain cause and treat appropriately. For idiopathic cases, drug treatment with danocrine 100mg b.i.d. or tamoxifen 20mg b.i.d. This is usually effective in small sized gynaecomastias. For large ones, surgery is the best form of treatment.

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.

SURGICAL CONDITIONS OF STOMACH

I. HYPERTROPHIC PYLORIC STENOSIS (CONGENITAL)

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

II. ACQUIRED PYLORIC STENOSIS

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

III. ACUTE GASTRITIS

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

IV. CHRONIC GASTRITIS

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

V. GASTRIC CANCER

Epidemiology and Incidence in Hong Kong

1. Ranked 5th both for males and females for new cancers. Ranked 3rd for males and 5th for females for cancer deaths.
2. About 1000 new cases each year.
3. Sex ratio: male to female = 1.76 : 1.
4. Median age at diagnosis: male = 65; female = 69.
5. Lifetime risks: male = 1 in 38; female = 1 in 91

Aetiology

1. Dietary deficiencies and carcinogenic substances
   * nitrates and nitrites in smoked and salted food
   * cigarette smoking
   * inhalation of environmental dust

   Note : Protective substances = fresh fruits and vegetables, vegetable oils, vitamins C & E

2. Helicobacter pylori?
3. Risk factors: - chronic gastritis (both chronic atrophic and hypersecretory gastritis)
- adenomatous polyp
- chronic gastric ulcer
- genetic factors (blood group A)
- prior Billorth II gastric resection

Symptomatology

* dyspepsia
* progressive weight loss
* anorexia
* anaemia
* mass
* present with complication: bleeding, perforation or obstruction
* silent, present with metastasis

Pathology

Macroscopic types :

* superficial or early gastric cancer
* polypoidal carcinoma
* ulcerative carcinoma
* infiltrative (linitis plastica)

Histological types :

Lauren classification (1965): most commonly used
* intestinal type :- in high incidence areas, mainly in men and older patients, better prognosis
* diffuse type :- in low incidence areas, more frequent in women and younger patients, worse prognosis
* mixed type :- less common

Incidence in US: intestinal = 40%; diffuse = 50%; mixed = 10%

Spread

* direct spread
* lymphatic spread
* blood spread
* transperitoneal spread

Staging and Location

UICC staging (1987)
* size of tumour is not as important as depth of invasion

Location
* Increase incidence of upper third and cardiac cancers in US and UK
* In US: antrum or distal third = 40%
body or mid third = 25%
fundus and proximal third = 35%

Diagnosis

Screening in high incidence areas :- gastrocamera

Barium studies

Endoscopy: biopsy and brushing

Endoscopic ultrasound

Percutaneous abdominal ultrasoun

CT scan

Surgical Treatment

1. Curative treatment
   * gastrectomy - subtotal or total, depending on site of tumour
     - margin : 4 - 6cm
   * radical lymphadenectomy
   * splenectomy ?

2. Adjuvant therapy
   * intraoperative radiotherapy - no increase in morbidity
     - ? benefit for survival
   * postoperative chemotherapy
     FAM - 5-FU, adriamycin, mitomycin C - no benefit
     FAMTx - 5-FU, adriamycin, methotrexate - some benefit
   * biological response modifiers - limited success
   * combination radiation and chemotherapy - some benefit for unresectable tumours

3. Palliative treatment
   * gastrectomy
   * gastro-enterostomy
   * exclusion gastro-enterostomy

Survival

Overall 5-year survival rate
US : 15%
Japan : 50%

Survival according to stages (Japan) :
IA : 91%
IB : 88%
II : 71%
IIIA : 43%
IIIB : 23%
IV : 7%

Prognostic Factors

* distant metastases
* depth of invasion
* lymph node involvement
* histological type
* site of tumour
* size of tumour
* age of patient

PEPTIC ULCERATION

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

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

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

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

NO ACID - NO ULCER

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

I. AETIOLOGY

Duodenal Ulcer

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

Special factors associated with aetiology:

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

6. Endocrine - a. Zollinger-Ellison syndrome
   b. Hyperparathyroidism
7. Cirrhosis of liver
8. Chronic bronchitis
9. Stress and anxiety
10. H. pylori

Gastric Ulcer

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

1. Gastric condition :
   * acid pepsin concentration
   * gastric stasis
   * coexisting duodenal ulcer
   * gastritis
   * bile reflux
   * H pylori

2. Clinical condition :
   * chronic alcohol use
   * NSAID
   * smoking
   * long-term steroids
   * intra-arterial chemotherapy

II. SYMPTOMATOLOGY

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

III. DIAGNOSIS OF GASTRIC AND DUODENAL ULCERS

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

IV. TREATMENT

Duodenal Ulcer

Medical treatment consisting of antacids and H_2-Receptor Antagonist, e.g. cimetidine, ranitidine; proton-pump inhibitor e.g. omerprazole and cytoprotective agent e.g. prostaglandins, colloidal bismuth, sucralfate.

Surgical treatment is indicated for

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

Surgical treatment consists of either truncal vagotomy and drainage operation (such as pyloroplasty or gastrojejunostomy) or truncal vagotomy and antrectomy. Proximal gastric vagotomy is the procedure of choice these days. Polya type of partial gastrectomy is done less frequently.

Gastric Ulcer

Medical treatment consists of treatment with antacids and H_2-Receptor Antagonist; proton-pump inhibitor and cytoprotective agent.

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

V. ZOLLINGER-ELLISON SYNDROME

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

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

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

Treatment

Excision of the tumour if possible, otherwise highly selective vagotomy or total gastrectomy should be done. Recently H_2-Receptor Blocking drugs have been used with some success.

BENIGN DISEASES OF THE BILIARY TRACT

Bile

500-1500 ml/day

Contains: bile salts
lecithin
cholesterol
bilirubin
fatty acid
inorganic salts

Regulation of Flow

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

*cholecystokinin-pancreozymin (CCK-PZ)

Enterohepatic Circulation

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

cholate	40%
chendoeoxycholate	40%

Secondary Bile Salts

deoxycholate	20%
lithocholate	

Resorption of Bile Salts

By terminal 200 cm of ileum
95% reabsorbed

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

Gallstones

Cholesterol gallstones - pure cholesterol stones
mixed cholesterol stones

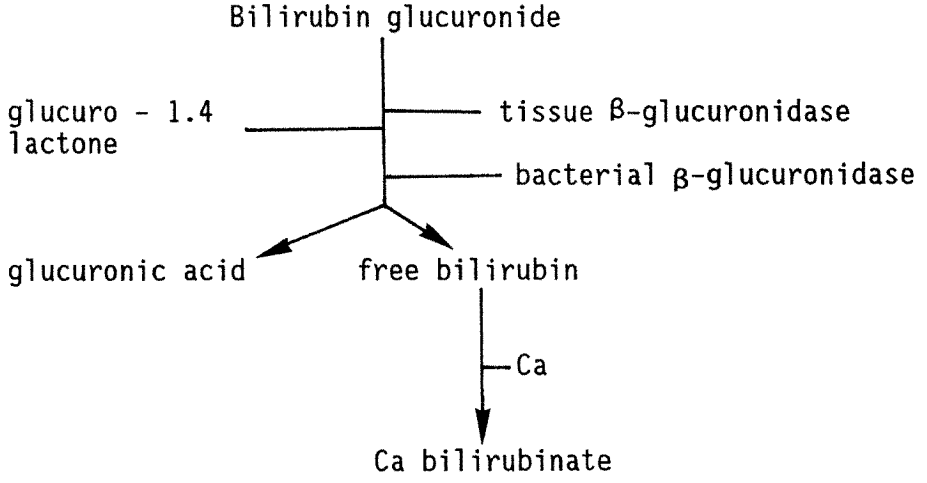
Pigment stones - bilirubin stones (black stones)
calcium bilirubinate stones - RPC

Rare stones - calcium carbonate stones
fatty acid - calcium stones

Formation of Cholesterol Gallstones

Cholesterol-holding capacity of micelles
Cholesterol secretion
Bile salt pool

Formation of Calicium Bilirubinate Stones



Diagnostic Examination of the Biliary Tree

Plain Abdominal Film

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

Oral Cholecystogram

Tyropanoate or iopanic acid taken the night before.

Cause of non-opacification

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

Highly reliable (>95% true positive)

Intravenous Cholangiogram

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

Endoscopic Retrograde Cholangiopancreatography (ERCP)

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

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

Percutaneous Transhepatic Cholangiography (PTC)

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

Complications : bleeding, bile leak - peritonitis, introduce infection

Ultrasonography

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

>90% accurate in expert hands for gallstones

Asymptomatic Gallbladder Stones

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

Indications for surgery

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

Gallstones and Chronic Cholecystitis

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

Treatment - cholecystectomy, operative cholangiogram
dissolution of stones?

Complications - acute cholecystitis, common duct stones, Ca gallbladder

Acute Cholecystitis, Empyema

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

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

Differential Diagnosis

perforated peptic ulcer
acute pancreatitis
acute appendicitis
acute viral hepatitis

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

surgery - severe symptoms, palpable tender
gallbladder

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

Gallstone Ileus

Females
Average age - 70

Symptoms and Signs

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

Investigation

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

Treatment : emergency laparotomy and removal of stones
cholecystectomy later

Choledocholithiasis

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

Recurrent Pyogenic Cholangitis

Aetiology

Bacterial infection characterized by

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

2. 30% incidence of anaerobic infection

Bacteria Involved

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

Portal of Entry

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

Clinical features

Sex : male=female
Age : 40's

Fever, jaundice, RUQ or epigastric pain

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

Investigations

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

Treatment Conservative - IV fluids, antibiotics, nil by mouth

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

Other investigations - ERCP, PTC

Principles of Treatment

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

Prognosis

About 20% will require reoperaton due to recurrence of cholangitis

Uncommon Causes of Biliary Obstruction or Cholangitis

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

LIVER ABSCESSES

Liver abscesses

Pyogenic - cholangitic
- non-cholangitic

Amoebic

Diseases predisposing non-cholangitic pyogenic abscess :

ulcerative colitis
acute appendicitis
acute diverticulitis
perforated peptic ulcer

Symptoms and Signs

fever, chills and rigor
jaundice
hepatomegaly

Investigations :

ultrasonography, CAT scan
ERCP
anti-amoebic titre

Treatment

amoebic abscess - flagyl

pyogenic abscess

solitary or large
- external drainage + antibiotics

multiple small
- antibiotics

cholangitic abscess
- common duct exploration and drainage

non-cholangitic abscess
- remove primary focus

Liver cysts

Simple cyst
Polycystic liver
Hydatid cyst
Neoplastic cyst - cystadenoma

Simple cyst, polycystic liver

congenital
arises from bile duct elements

Treatment of simple cyst

excision
fenestration

Hydatid disease

Clinical features :- exposure in endemic areas
RUQ pain
hepatomegaly
jaundice

Treatment of hydatid cyst

Excision

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

Common duct exploration -
if rupture into bile ducts has occurred

NEOPLASMS OF THE LIVER, BILIARY TRACT AND PANCREAS

NEOPLASMS OF THE LIVER

Benign : Adenoma - no ductal elements
Focal nodlar hyperplasia
Haemangioma

Malignant : Hepatocellular carcinoma
Sarcomas
Carcinoid tumour

Focal Nodular Hyperplasia, adenoma

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

Hepatocellular Carcinoma

Second most common cancer in Hong Kong.

Male : Female = 4:1

80% HBsAg positive, 80% cirrhotic.

Pathology

nodular form
massive form
diffuse form
encapsulated 5-15%

Symptoms and Signs

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

Blood Investigations

CBP, LFT, RFT
serum alphafetoprotein (80%)
serum HBsAb, HBsAg
ICG retention test
Serum transferrin

Other Investigations

hepatic arteriogram, superior mesenteric arteriogram
- look for portal vein invasion, A-V shunting.

ultrasonography, CAT scan
peritoneoscopy

Peritoneoscopy

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

Prerequisite for Surgical Resection

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

Surgery for HCC

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

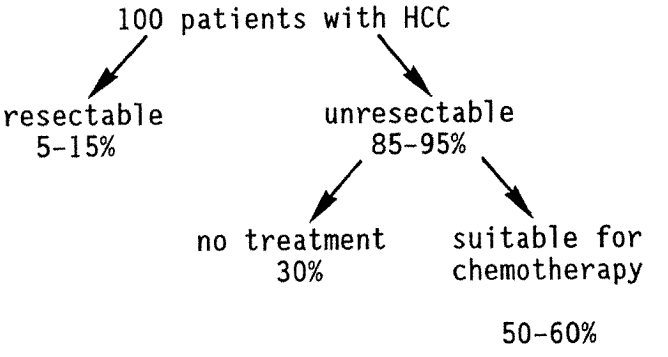
Nonoperative Treatment

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

regional chemotherapy - by cannulation of hepatic artery
25% response

embolization - for rupture
- as definite treatment

radiotherapy - ineffective



NEOPLASMS OF THE BILIARY TRACT

Benign : Cystadenoma

Malignant : Gallbladder - adenocarcinoma
- squamous cell ca

Bile ducts - cholangiocarcinoma
- cystadenocarcinoma
- mucoepidermoid carcinoma

Adenocarcinoma of the Gallbladder

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

Treatment

cholecystectomy - for those discovered incidentally and confined to mucosa

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

most are unresectable at presentation

Other Therapies

chemotherapy - ineffective
radiotherapy - ineffective

Prognosis

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

Cholangiocarcinoma

more than 80% associated with clonorchiasis

Symptoms and Signs

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

Blood investigations

CBP, RFT, LFT, CEA

Other investigations

ERCP - brushings of duct
bile for cytology

PTC

Ultrasonography and CAT scan

Treatment

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

Palliative treatment

Non-operative

insertion of endoprosthesis - endoscopically
- percutaneously

Operative

hepaticojejunostomy
cholangiojejunostomy (to segmental ducts)

Other treatment

chemotherapy - ineffective
radiotherapy - localised radiotherapy
effective for palliation

Carcinoma of the Ampulla

Symptoms and signs

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

Investigations

upper endoscopy - ampullary biopsy
ERCP
PTC
hypotonic duodenography

Treatment

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

Palliative treatment

Nonoperative - endoscopic papillotomy
- insertion of endoprosthesis endoscopically

Operative - hepaticojejunostomy

NEOPLASM OF THE PANCREAS

Benign : adeoma
cystadenoma

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

Adenocarcinoma of the Pancreas

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

Symptoms and signs

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

Blood investigation

CBP, LFT, RFT, serum CEA.

Other investigations

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

Treatment

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

Palliative treatment

Operative - bypass operations (triple bypass)

Nonoperative - radiotherapy
intraoperative, implantation

- chemotherapy
ineffective

Islet Cell Tumours

Symptoms and signs

gastrinoma - recurrent ulcers
insulinoma - hypoglycemia

Investigations

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

Treatment

gastrinoma - resection of pancreas if localized
- resection of stomach if desseminated
- cimetadine, ranitidine

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

PORTAL HYPERTENSION AND DISEASES OF THE SPLEEN

I. CLASSIFICATION OF PORTAL HYPERTENSION

1. Extrahepatic Presinusoidal Block

a. Infantile or neonatal umbilical vein sepsis
b. Pyelophlebitis from intra-abdominal sepsis
c. Malignant tumour invading portal vein

2. Intrahepatic Perisinusoidal Block

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

3. Intrahepatic Postsinusoidal Block

a. Cirrhosis : alcoholism, post necrotic, biliary
b. Wilson's disease
c. Haemochromatosis
d. Veno-occlusive disease involving the hepatic vein

4. Extrahepatic Postsinusoidal Block

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

5. Arteriovenous Shunt

II. AETIOLOGY OF PORTAL HYPERTENSION

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

Normal free portal vein pressure 15-20 cm H_2O

Portal hypertension > 25 cm H_2O

III. METHODS OF MEASURING PORTAL VEIN PRESSURE

1. Directly at laparotomy by cannulation of branch of superior mesenteric vein
2. Transumbilical portal pressure
3. Wedge hepatic venous pressure
4. Percutaneous transhepatic portal pressure

IV. CLINICAL FEATURES OF PORTAL HYPERTENSION

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

V. COMPLICATION OF PORTAL HYPERTENSION

1. Infected ascites
2. Variceal bleeding
3. Porta-systemic encephalopathy

VI. INVESTIGATIONS

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

V. CHILD'S CLASSIFICATION OF HEPATIC FUNCTIONAL RESERVE

Clinical and laboratory classification of patients with chronic liver disease

	Points scored for increasing abnormality		
Clinical and laboratory measurement	1	2	3
Encephalopathy (grade)	none	1 and 2	3 and 4
Ascites	absent	slight	moderate
Bilirubin (mg%)	16-32	33-48	>49
Prothrombin time (seconds prolonged)	1-4	4-6	>6
Albumin (g/l)	3.5	2.8-3.5	<2.8
Bilirubin in primary biliary cirrhosis	16-64	65-160	>160

Grade A	5-6 points
Grade B	7-9 points
Grade C	10-15 points

VI. MANAGEMENT OF OESOPHAGEAL VARICEAL BLEEDING

Resuscitation
Intravenous fluid and blood replacement (C.V.P. monitoring, if necessary)
Emergency upper endoscopy for verification

Anti-hepatic coma regime (in patients with poor hepatic reserve)
- Gastric lavage
- Enema
- Neomycin
- Lactulose

Parenteral Vitamin K

1. Non-operative Therapy

Intravenous pitressin
Oesophagogastric tamponade
Injection sclerotherapy of varices
Banding of varices
Transjugular intrahepatic porta-systemic shunt

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

Oesophagogastric tamponade
- Sengstaken tube (3 lumens)
- Minnesota tube (4 lumens)
- Linton tube (large gastric balloon)

Injection sclerotherapy
- Endoscope : Rigid Negus (GA) - obsolete
  Fexible ± sheath (LA)
- Sclerosant : 5% Ethanolamine oleate
  STD (Sodium tetradecyl sulphate)
- Site : Intravariceal

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

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

Disadvantages
- Multiple sessions + long-term follow-up
- Not useful for fundic varices
- High rebleeding rate (35+%) before obliteration

2. Surgical Therapy

a. Transthoracic ligation of varices for haemostasis OR
b. Transabdominal transgastric plication of varices for haemostasis
c. Devascularization (including oesophageal transection) for prevention of recurrence
d. Porta systemic shunt for prevention of recurrence
e. Liver transplantation as a definitive treatment

Porta Systemic Shunt

Shunting operation can be done as emergency after haemostasis of bleeding varices or as elective procedure after control of bleeding by non-operative means

Types

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

2. Splenorenal shunt
   - end to side
   - side to side
   - distal (Warren's)

3. Mesocaval shunt
   - end to side
   - interposition (with graft)

Complications

1. Post shunt encephalopathy
2. Thrombosis of shunt → recurrence

VII. CHOICE OF TREATMENT FOR PORTAL HYPERTENSION

Non-operative treatment is successful in controlling about 90% of variceal bleeding. If bleeding continues or recurs, further treatment depends on liver function. For child's A or B patients, shunting is the preferred treatment. For child's C patients, devascularization is preferred, but liver transplantation should be considered if organ is available. Mortality is high in child's C patients (60-70%).

VIII. SPLEEN

Indications for Splenectomy

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

Haematological Disorders

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

1° Hypersplenism

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

2° Hypersplenism

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

Other Indications for Splenectomy

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

Haematological Consequences of Splenectomy

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

Complications of Splenectomy

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

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

Rupture of Spleen

Clinical presentation

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

Diagnosis

- Trauma to left upper quadrant
- Hypotension
- Tachycardia
- Haemoperitoneum
- Peritonitis

Radiological Features

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

Clinical Features

- Kehr's sign
- Ballance sign

Treatment

- Splenectomy
- Autotransfusion

DISEASES OF THE PANCREAS

Pancreatitis

Aetiology

gallstones	55%
alcohol	15%
unknown	15%
miscellaneous	15%

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

Pathogenesis

1. Obstruction - secretion

   Obstruction alone
   - exocrine atrophy

   Obstruction + stimulation
   - pancreatitis

2. Reflux - common channel theory

   Bile reflux, especially infected bile, duodenal juice reflux

3. Vascular - ischaemia

Pancreatitis

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

Acute Pancreatitis

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

Laboratory Findings

serum amylase > 1000 iu/l

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

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

Urinary amylase - creatinine clearance ratio > 5%

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

Other non-acute conditions with raised serum amylase

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

Investigations

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

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

ERCP after recovery

Factors of Poor Prognosis :

1. Age > 50
2. Leucocytosis
3. low pO_2
4. elevated serum urea
5. elevated serum glucose
6. low serum calcium
7. elevated serum SGOT, LDH
8. low serum albumin
9. presence of methaemalbumin in serum
10. presence of DIC

Medical Treatment :

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

Surgical Treatment :

<u>During acute attack</u>

1. for pancreatic abscess

2. for haemorrhagic pancreatitis diagnosed by :
   a. serum methaemalbuminemia
   b. Cullen's or Grey Turner's signs
   c. DIC
   d. haemoglobin

   Procedure
   partial or complete excision of pancreas
   drainage

3. for other complications
   haemorrhage
   fistula

4. Miscellaneous Methods of Treatment
   a. Peritoneal lavage for severe acute pancreatitis
      some early improvement in renal function and PO_2
      die later from septic complications
   b. Nutritional support for prolonged clinical course
      via enteral tube in jejunum
      total parenteral nutrition

During delayed/convalescene stage

1. For biliary pancreatitis

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

   Procedure

   cholecystectomy,
   common duct exploration and T-tube drainage,
   ± sphincteroplasty

2. for pancreatic pseudocyst, diagnosed by
   a. epigastric mass
   b. ultrasonography, CAT Scan

   Procedure

   cystogastrostomy
   cystoduodenostomy
   cystojejunostomy
   external drainage

Chronic Pancreatitis

Aetiology

alcoholism
hypercalcaemia
hyperlipidaemia
familial pancreatitis

Symptoms and Signs :

persistent or recurrent abdominal pain
pancreatic insufficiency

Laboratory and investigational findings :

diabetes mellitus
calcifications on X-ray
ERCP - dilated and irregular pancreatic duct
secretin - CCK stimulation test

Medical Treatment :

analgesics
enzymes
insulin

Surgical Treatment :

pancreatectomy
internal drainage of pancreatic duct
sphincteroplasty

BENIGN CONDITIONS OF THE COLON, RECTUM AND ANUS

I. HAEMORRHOIDS

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

Types

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

Associated conditions

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

Clinical features

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

Degrees of haemorrhoid

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

Complications

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

Treatment

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

II. THROMBOSED EXTERNAL HAEMORRHOID

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

Clinical features

1. Sudden pain
2. Lump in the anus

Treatment

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

III. ANORECTAL ABSCESS

Organisms

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

Aetiology

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

Types

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

Clinical features

1. Pain
2. Swelling
3. Fever

Treatment

1. Incision and drainage (usually under GA)
2. 1-stage or 2-stage

Prognosis

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

IV. ANORECTAL FISTULA

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

Types

1. Low level - subcutaneous
   submucosal
   intersphincteric
   transphincteric
2. High level - very rare
   iatrogenic

Clinical features

Persistent seropurulent discharge from an opening around the anus.

Treatment

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

V. ANAL FISSURE

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

Clinical feature

Pain in anus

Differential diagnosis of anal pain

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

Treatment

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

VI. RECTAL PROLAPSE

Partial prolapse

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

Complete prolapse

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

Treatment

1. Partial prolapse : Conservative treatment
   Submucous injection of phenol
   Excision of prolapsing mucosa
2. Complete prolapse : Poor risk patient - Perianal procedure
   Good risk patient - Transabdominal fixation of rectum

VII. POLYPS OF COLON AND RECTUM

Type

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

Much evidence to support adenoma - carcinoma transformation

Clinical feature

Fresh blood per rectum

Differential diagnosis of rectal bleeding

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

Diagnosis

1. Barium enema
2. Colonoscopy
3. Arteriography

Treatment

Colonoscopic polypectomy

VIII. SIGMOID COLON VOLVULUS

Predisposing factors

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

Clinical feature

Symptom and sign of intestinal obstruction

Diagnosis

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

Treatment

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

IX. OTHER BENIGN CONDITIONS

Ulcerative colitis
Diverticular disease
Crohn's disease

MALIGNANT CONDITIONS OF THE COLON, RECTUM AND ANUS

I. INTRODUCTION

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

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

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

II. PATHOLOGY

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

1. Staging by Dukes' classification

   Stage

   A. confined to the rectal wall

   B. penetrating the rectal wall

   C. lymph node involvement
      C_1 - pericolic lymph nodes involved
      C_2 - mesenteric lymph nodes involved

2. Staging can be by TNM classification

III. SYMPTOMS

Local :

Pain —> perforation
Diarrhoea
Constipation —> obstruction
Positive occult blood in stool —> bleeding

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

Left colon lesions
Altered bowel habits
Blood in stool
Obstruction

Rectal lesions
Bleeding
Altered bowel habits
Tenesmus

Systemic :

↓ weight
↓ appetite
↓ energy
Dyspnoea
Ascites, oedema

IV. INVESTIGATIONS

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

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

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

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

V. SURGERY

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

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

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

VI. PROGNOSIS

5-year survival

Dukes' A : 90%
B : 70%
C : 30%

VII. POST-OPERATIVE MANAGEMENT

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

VIII. CHEMOTHERAPY

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

IX. RADIOTHERAPY

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

X. MALIGNANT TUMOUR OF THE ANUS

Types : Epidermoid
Basal cell (transitional) carcinoma
Malignant melanoma
Bowen's disease
Paget's disease
Malignant lymphoma

XI. TREATMENT

Radiotherapy
Chemotherapy
Local excision
AP resection

CUTANEOUS AND SUBCUTANEOUS LESIONS

1. To make diagnosis

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

2. Possible layer of tissue of origin

   Skin
   Subcutaneous tissue
   Muscle
   Bone or joint

3. Physical characteristics of lump

Site	temperature
Shape	colour
Edge	ulceration
Surface	fluctuation
Consistency	transillumination
	pulsatility

4. Epidermal cyst

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

5. Sebaceous cyst

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

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

   Treatment : excision
   incision avulsion

6. Implantation dermoid

Epithelium driven beneath skin by puncture wound
Common site - fingers

7. Sequestration dermoid

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

a. external angular dermoid
b. periauricular
c. midline

8. Branchial cyst

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

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

9. Branchial fistula

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

Treatment - excision

10. Papilloma of skin

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

11. Squamous papilloma

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

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

Infectious
Often disappears spontaneously

Treatment : cauterisation
curettage

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

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

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

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

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

13. Keratoacanthoma (molluscum sebaceum)

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

Middle age or elderly
Face or hands
Firm hemispherical nodule
Grows rapidly into a rounded, slightly umbilicated mass
1-2 cm diameter in course of 1-2/12
Summit ulcerates, forms crust beneath which is on ulcer crater
Crust is shed
Regression occurs
Heals within 6/12 or so, leaving a small pitted scar

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

Benign non-encapsulated dermal nodular lesion
Of proliferating fibroblast
Irregularly arranged collagen bundles
Capillary blood vessels
Histiocytes
Mononuclear inflammatory cells
In different proportions
Histiocytes may contain haemosiderin or become xanthomatised

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

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

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

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

15. Granuloma pyogenicum

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

Treatment - excision

16. Pilomatrixoma

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

17. Lipoma

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

18. Fibroma

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

Desmoid tumour

19. Neurofibroma

Localised
Generalised

20. Haemangioma

Capillary - salmon patch
portwine stain
strawberry angioma
Venous
Arterial

21. Lymphangioma

Capillary
Cavernous
Cystic hygroma

22. Inflammatory swelling

Boils
Carbuncles

MALIGNANT DISEASES OF SKIN

I. BASAL CELL CARCINOMA

Fair skin, sunlight
Age - middle or late

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

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

Slow growing
Locally invasive

Treatment - radiotherapy
surgery

II. SQUAMOUS CELL CARCINOMA

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

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

Sites - anywhere
common on head and neck and upper limbs

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

Treatment - local - RT
surgery
- lymph nodes

III. MALIGNANT MELANOMA

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

Benign pigmented naevus

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

Signs of malignant change in benign pigmented naevus

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

* suspicion - biopsy

Types of malignant melanoma

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

Prognostic factors

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

Treatment (malignant melanoma)

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

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 incompetence (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

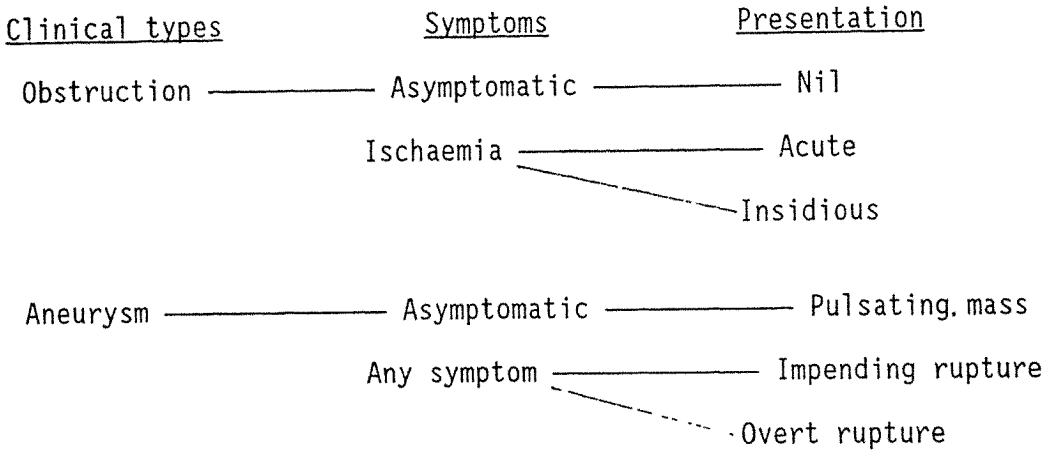
Atheromatous* - most common

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

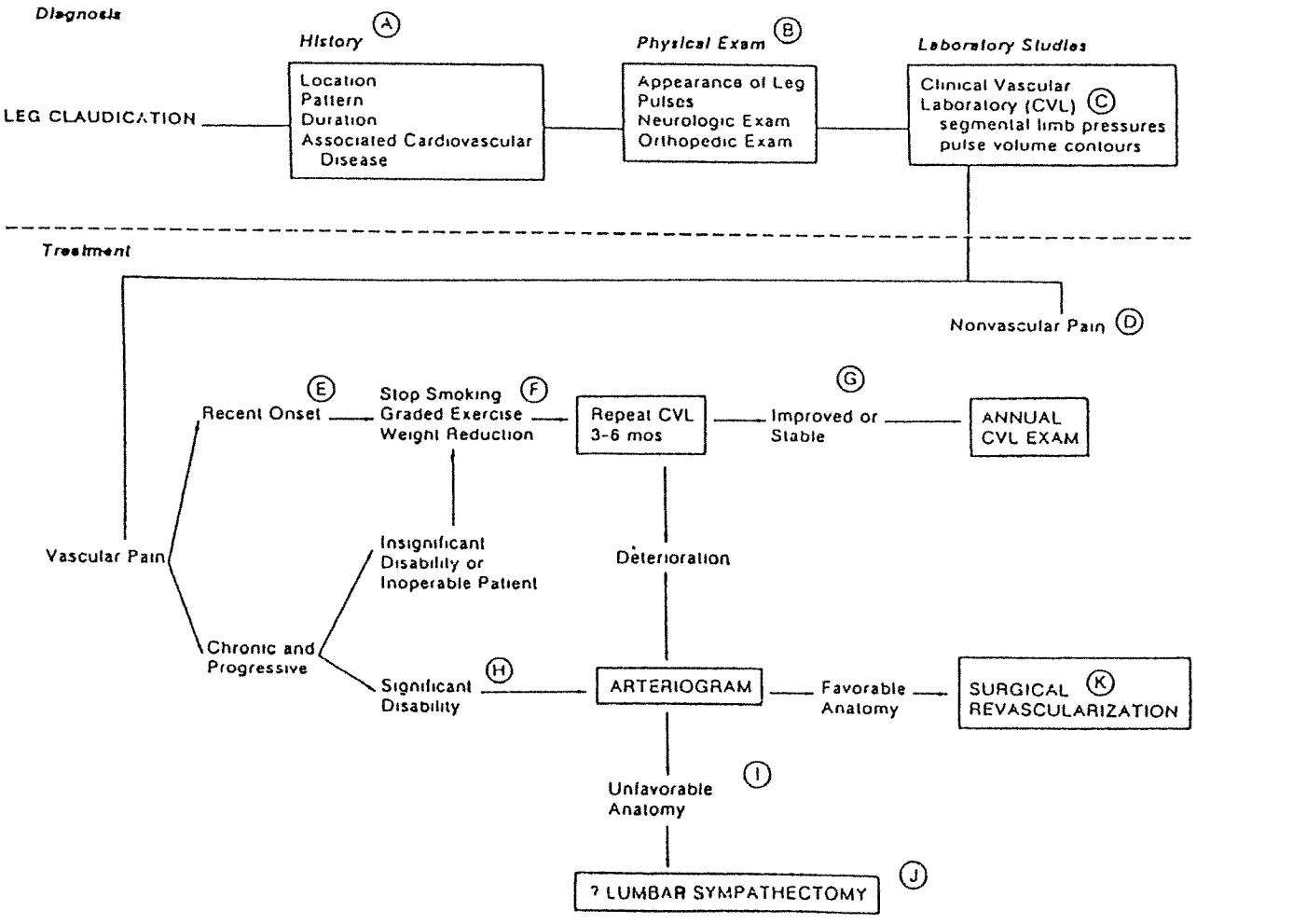
e. Reference line

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

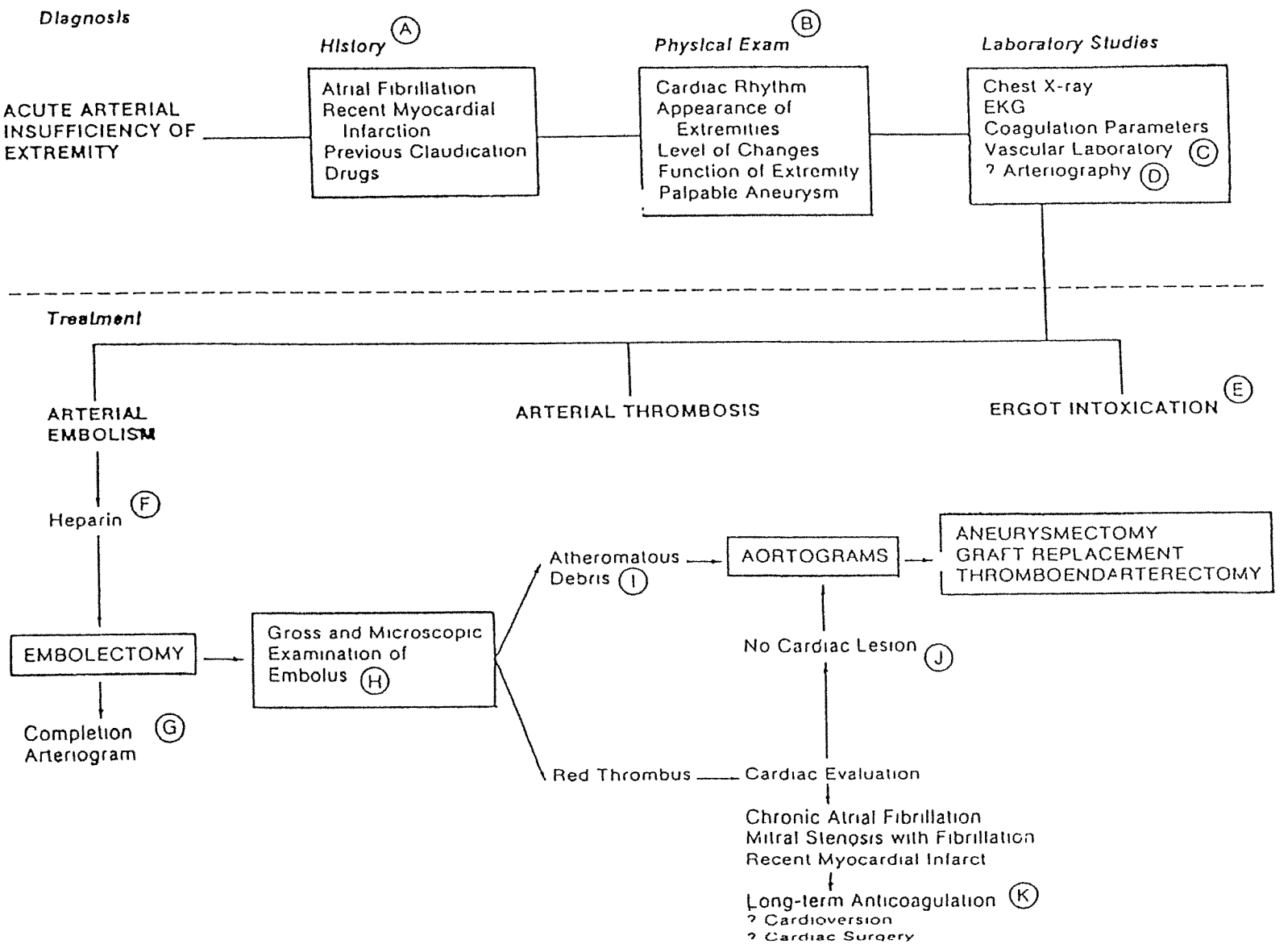
IX. HOW DOES ARTERIAL SURGERY DIFFER FROM OTHER BRANCHES OF SURGERY ?

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



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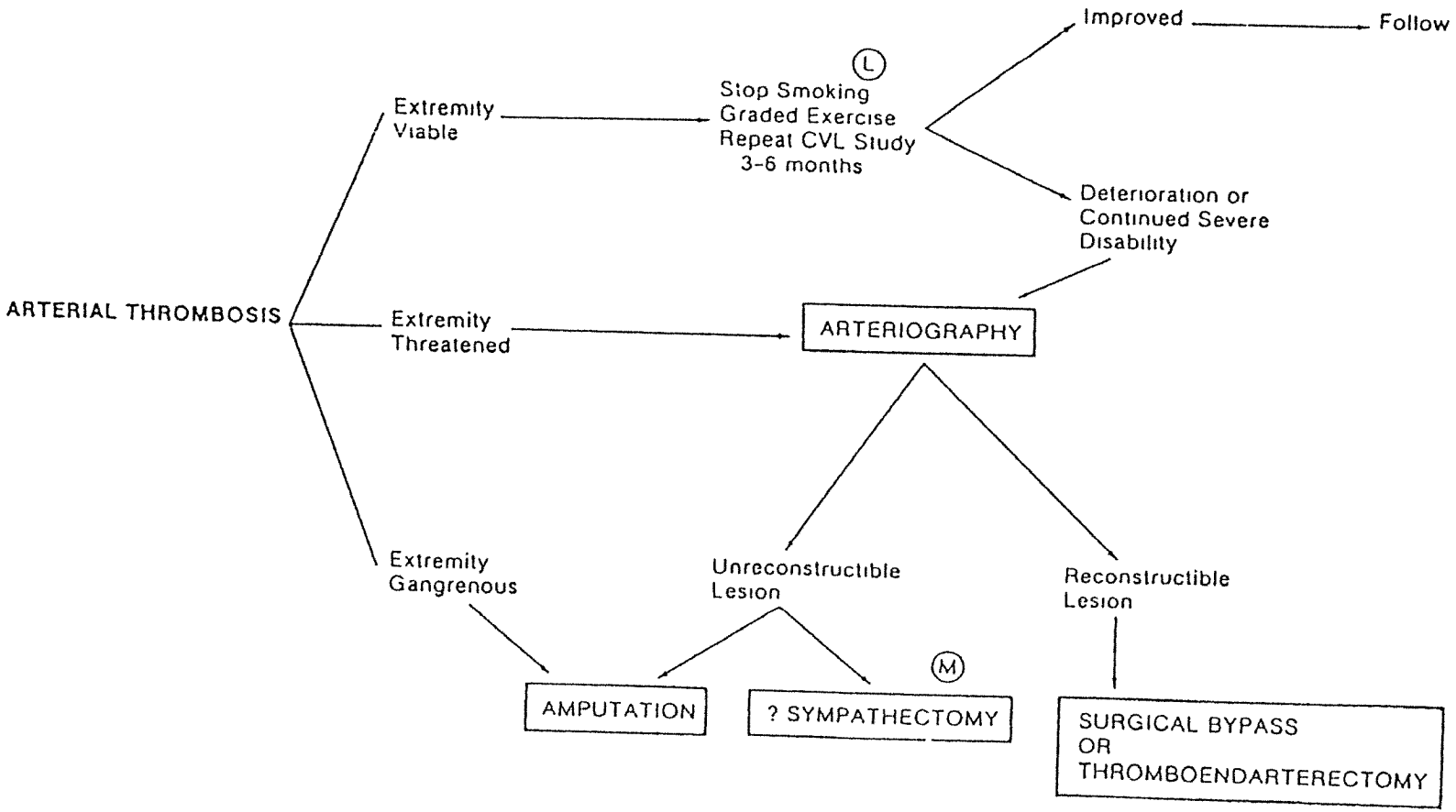
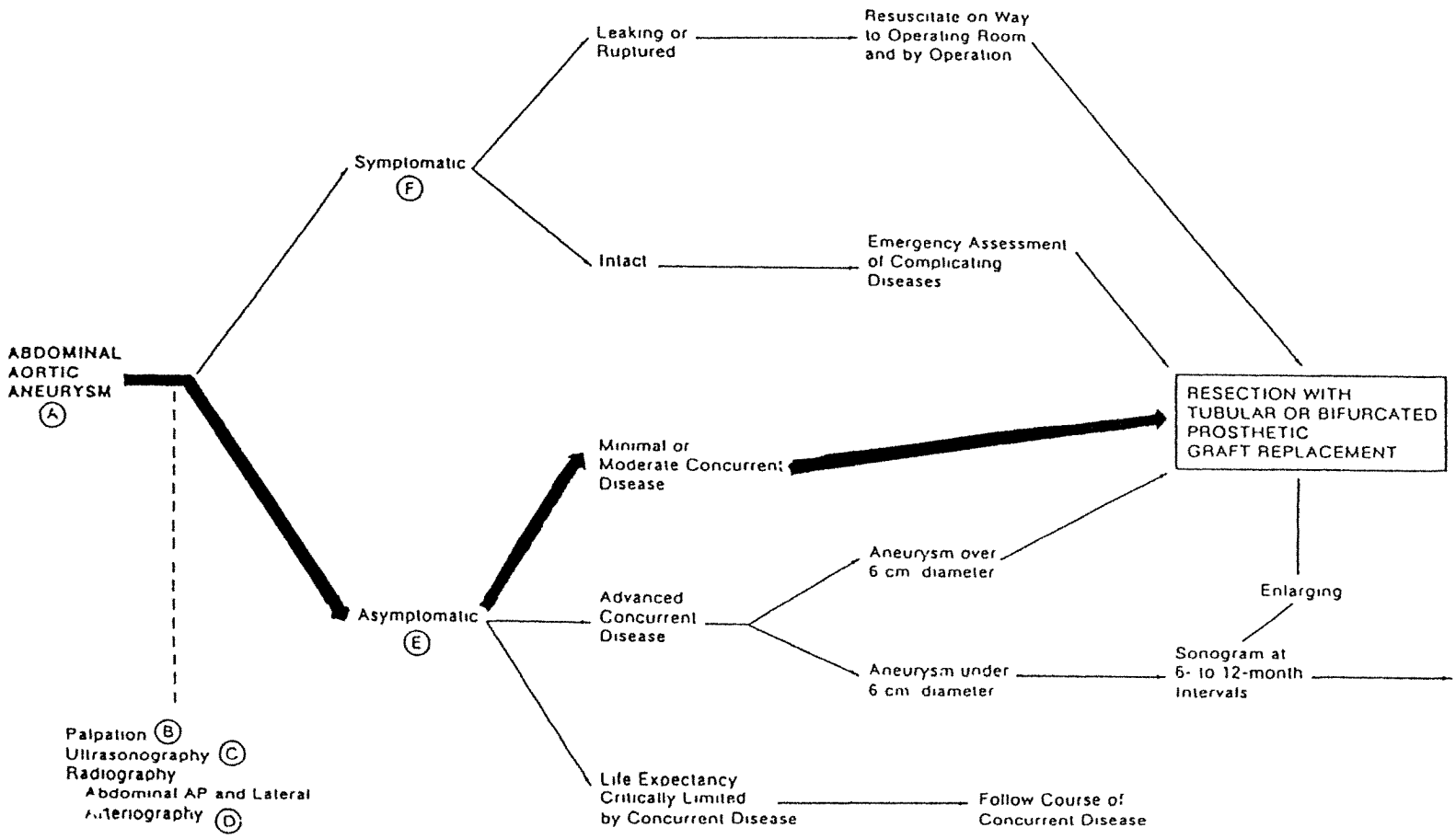
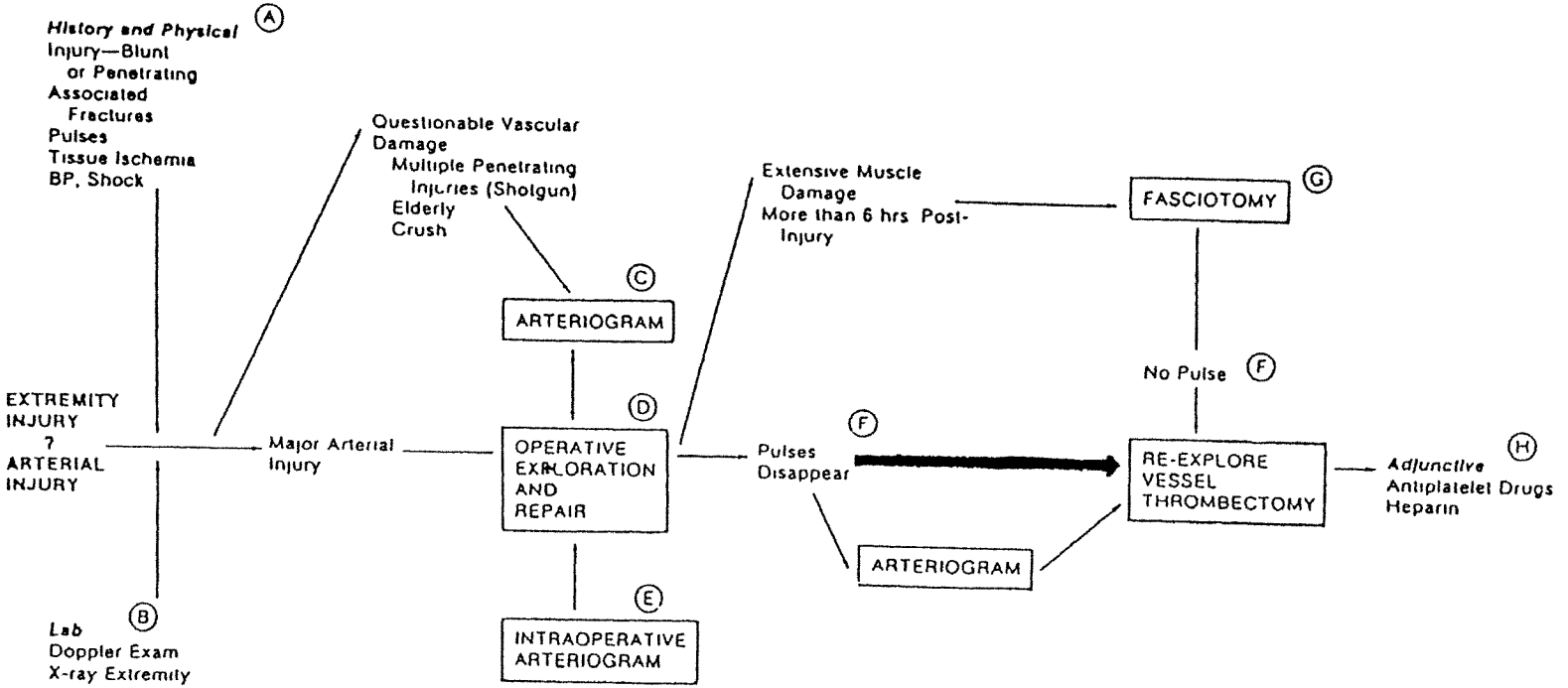


INFRARENAL ABDOMINAL AORTIC ANEURYSM



ARTERIAL INJURY OF EXTREMITY



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

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 $\pm$ 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

✓ 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

✓ 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

ORGAN TRANSPLANTATION

INTRODUCTION

Organ transplantation is required when the organ concerned is damaged by disease process and the functional deterioration will lead to eventual death of the patient.

The major problem of organ transplantation is rejection as the donor and recipient are genetically dissimilar. Thus the early results of organ transplantation were unsatisfactory except the kidney transplant between identical twins performed in 1954. Thereafter, advances in technology, organ preservation method and immunosuppressive therapy have made organ transplantation between dissimilar subjects possible. Nowadays, transplantation of kidneys, livers, cornea, heart, lung and bone marrow become established operations for patients with end-staged organ failure.

SOURCE OF ORGANS

Organs could be obtained from heart-beating brain dead patient or relative of the recipient.

Brain-dead patients are those sustaining severe head injury, spontaneous intracerebral haemorrhage or primary brain tumour and with respiratory support by ventilator. Their brain stems are basically not functional in maintaining spontaneous respiration and therefore oxygenation and heart-beating will cease immediately on disconnection from the respirator. Brain-stem death can be certified by many laboratory tests but nowadays clinical tests are reliable in making the decision. The criteria of brain stem death are as follows:

1. The apnoeic coma is not due to depressant drugs, neuromuscular blockade, hypothermia, metabolic or endocrine disturbance.
2. The pupils are both fixed to light.
3. Corneal reflex is absent.
4. There is no eye movements with cold caloric test.
5. Doll's sign is negative.
6. There is no cranial nerve motor response.
7. There is no gag reflex.
8. There is no respiratory movements on disconnection from respirator with $PaCO_2$ above 6.6 kPa.

With consent from patient's immediate relative, the donor is operated in the operating theatre and the appropriate organ removed and stored in preservation solution. Recipient operation is performed immediately to reduce cold ischaemic time.

For living-related organ transplantation, a part of the organ, e.g. liver, pancreas or one of the pair of organs, e.g. kidney was removed from a close relative e.g. sibling, parents and implanted into the recipient.

PRESERVATION SOLUTION

1. Collins' solution.

2. Euro-Collins' solution.

3. Hypertonic citrate solution.

4. University of Wisconsin (UW) solution.

UW solution is the solution recommended nowadays. The active components are the lactobionic acid and raffinose which suppress hypothermia-induced cell swelling and the adenosine which provides an effective substrate for ATP synthesis during reperfusion following cold storage. This solution allows cold storage of the organ up to 24 hours.

IMMUNOSUPPRESSIVE DRUGS

1. Steroid

2. Azathioprine

3. Cyclosporine

4. Antilymphocyte globulin

5. FK 506

OVERALL RESULTS

Satisfactory results are obtained with organ transplantation, e.g. the 5-year survival rate of liver transplantation is about 75-80%.