Thoracic Surgery Revision
Anatomy

- Right Main Bronchus has more vertical course than left main Bronchus
- Right Upper Lobe Bronchus arises more proximally than left Upper Lobe Bronchus
- The right Pulmonary artery passes anterior to Major Bronchi and is longer than the left PA
- The left pulmonary artery arches over the left main and upper lobe Bronchus to descend postero-lateral to the left lower lobe Bronchus

**Right Hilum**
- Main PA is found at junction between SVC and Azygous Vein
- Veins are just below but anterior
- Artery behind vein
- Therefore VAB configuration

**Left Hilum**
- Veins still anterior
- Bronchus before PA
- Hence VBA configuration
Anatomy - Bronchi

- From Bifurcation of Trachea each main Bronchus passes down and lateral to enter Hilum
- Main Bronchus is 5cm long with right being more vertical and slightly shorter
- Each main Bronchus gives rise to Lobar Brochi
- Each Lobar Bronchus (Upper, Lower, Middle) gives rise to Segmental Bronchi
- These are the Bronchi of the Broncho-pulmonary segments (Total of 19)
- Bronchi have smooth muscle and Hyaline Cartilage and are lined with Pseudo-stratified Cilliated Collumnar Epithelium with mucous glands
- After successive divisions become smaller and smaller
- When cartilage disappears they are now called Bronchioles
- When cilia disappear they are called respiratory Bronchioles
- Just before the respiratory Bronchiole is the terminal bronchioles
- Each terminal Bronchiole with its respiratory bronchioles is called the acinus
- 0 – 11 Bronchi – Ciliated with Cartilage
- 11-16 Terminal Bronchioles – No cilia no cartilage
- 16 – 23 Respiratory Bronchioles

Cerfolio
Airleaks

- Defined as prolonged after 7 days or that would delay discharge (some fast-track protocols discharge pts after 4/7)
- CCF paper 2005 – Majority of air-leaks following lobectomy stop by day 2-3 and < 10% persist after 7/7

Pre-operative identification of risk factors
- Emphysema patients with low FEV1 and low DLCO
- IDDM
- Patients on Steroids
- Lobectomy or LVRS
- Pleural adhesions

Operative techniques
- Fissure-less technique; not looking for PA in fissures but start at Hilum, take V,A, B then divide/complete fissures
- Buttress staple lines with bovine pericardium
- Bicycle tyre checking before closing chest
- Sealants – Foca – seal
- Some however say that there is poor correlation between intraoperative Pneumostasis and post-op leaks
- Consider Pleural tenting (parietal pleura stripped from chest wall and allowed to drape over resected staple line

Postoperative
- Under-water seal if lung up take off suction
- If Pneumothorax (lung down) use minimum suction to bring lung up
- If persistent air-leak and lung is up Heimlich valve and XRAY if lung stays up discharge
- If brisk air leak 5/7 (air leak meter) or continuous Heimlich valve will probably not work
- Elective pneumoperitoneum has been described to reduce incidence of prolonged air leak

Cerfolio
Achalasia

**Incidence**: 1 in 100,000

**Aetiology**
- Primary
  - Degenerative nerve process with loss of Ganglion cells in myenteric plexus with reduction of vagal nerve fibres in wall of oesophagus
- Secondary
  - Chagas disease
  - Amyloidosis and Sarcoidosis
  - Malignancy (Pseudoachalasia often due to gastric cardia adenocarcinoma clues with age > 55, short duration of symptoms)
  - Viral Infections
  - Diabetes

**Symptoms**
- Dysphagia (Liquids > Solids)
- Reflux of undigested foods
- Aspiration
- Squamous cell cancer of Oesophagus on average 20 years after diagnosis

**Investigations**
- CXRAY – usually normal but later on Air – fluid levels, loss of gastric bubble, perhaps signs of aspiration
- Barium – Tapering of Oesophagus “Birds Beak”, Dilated sigmoid like Oesophagus with undigested food
- Oesophageal Manometry – Incomplete relaxation of LES, Hypertensive LES, ultimately aperistalsis body of Oesophagus
- Vigorous Achalasia – High amplitude non-peristaltic contraction (Present commonly with chest pain)

**Oesophageal Motility Disorder** characterised by failure of LOS to relax
Achalasia

Oesophageal Hypo-Motility Disorder characterised by failure of LOS to relax

Barium swallow: "bird's beak" esophagus
Esophageal dilatation; megaesophagus

Peristalsis absent in esophageal body
Contractions weak at all recording levels
Failure of relaxation of a hypertensive LOS
Mirror like activity at all recording levels
Achalasia Management

**Pneumatic Dilatation**
- Successful in 70% of cases
- The dilator is placed fluoroscopically so balloon is centred on GEJ
- Dilatation to at least 3 cm, of 2-3 atmospheres, 30 – 35 ml Gruntzig Balloon image intensifier
- Should be considered first line therapy in most patients
- Patients who fail two dilatations should be considered for surgical myotomy
- Risk of perforation during 1 – 5%

**Surgical**
- Heller’s anterior and posterior Myotomy (Now modified into one single myotomy)
- Surgical transection of outer Longitudinal and inner circular muscles preserving Submucosa
- Palliation in 80 – 90% long-term
- Controversy exists as to whether anti-reflux procedure is required as well
- Oesophageal resection reserved for those failing myotomy or have end-stage mega-oesophagus
- With vigorous Achalasia myotomy may have to be extended to aortic arch!

**Medical Therapy**
- Calcium channel blockers, Nitrates, B Agonists
- Endoscopic Botulinum toxin injection (long-term as effective as Pneumatic dilatation)

**Pneumatic Dilatation Versus Surgery**
- RCT 39 treated with PD Vs 42 with surgery
- Good immediate response both but at 5yrs results sustained in 95% of surgical group vs 65% in PD

Consider Myotomy as first line treatment in young patients since efficacy of pneumatic dilatation is age dependent
ARDS

Aetiology
- Sepsis
- Direct lung trauma / contusion
- Aspiration
- Haemorrhagic Shock multiple transfusions
- MOF
- Cardiopulmonary Bypass

Pathophysiology
- In sepsis Macrophages in pulmonary endothelium activated by bacteria generate biological cascade including cytokines (TNF, IL, PAF)
- Chemotaxis of neutrophils oxygen free radicals
- Type I alveolar cells as well as damage to capillaries results in increased capillary permeability with flooding of alveoli with fluid
- Accumulation of cellular rich fluid results in development of hyaline membrane and Pulmonary fibrosis in 7 – 10 days
- In non-septic ARDS it is unknown what triggers cytokines

Diagnosis
- Rapid onset of hypoxemia within 72 hours of insult
- CXRAY – Diffuse bilateral infiltrates with interstitial infiltrates
- Oxygenation defect defined as PaO₂ / FiO₂ ratio of < 200 mmHg (27Kpa)
- Normal PCWP

Single organ ARDS Mortality 40–50 % Septic ARDS > Non-septic
ARDS Management

**Supportive**

**Monitoring**
- ICU, HR, BP, RR, Temperature, Daily fluid balance, CVP, PCWP, A-line, Acid-base, Nutritional

**Non-Ventilatory management**
- Treat underlying risk if possible
- Optimise O2 delivery, increase FiO2, Cardiac output, Hb
- Lowest possible CVP and PCWP
- Enteral feeding
- Patient positioning such as Prone can be tried

**Ventilator Management**
- Aim for Sats > 90%
- PO2 > 8
- CPAP useful
- PEEP 10 –15 cm H2O
- **Europe** – High FiO2 Low PEEP, **USA** – Lower FiO2 Higher PEEP
- Inverse I/E ratio
- Pressure control ventilation
- High frequency Jet Ventilation sometimes useful
- Nitric Oxide can be useful

Role of ECMO/Steroids have to be established
Ventilatory management of ARDS

- Traditionally ventilatory management is associated with baro-trauma from
  - High Inspiratory pressures
  - Volume-trauma from alveolar over-distension
  - Toxicity from High FiO2

- Leads to
  - Decreased compliance
  - Decreased FRC
  - Cardiac function impairment (as a result of high airway pressures)

- Evidence now suggests
  - Lower Tidal Volumes (6ml / kg) showed improved survival (ARDS network trial)
  - However lower tidal volumes results in more hypoxia and higher CO2
  - Role of ECMO comes in here for removing CO2 improving O2 and allowing lower tidal volumes

Single organ ARDS Mortality 40–50 % Septic ARDS > Non-septic
Pulmonary A-V Malformations

- Congenital malformation that results in persistence of a communication that bypasses the pulmonary capillary bed
- Association with hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu) 50% of AVM are associated with OWR
- Usually affects the lower lobe and usually peripherally, sub-pleural position
- One feeder artery more than one draining veins

Presentation
- Cyanosis due to Right to left shunt with clubbing etc.
- Right to left embolisation cerebral infarcts, abscess, endocarditis
- Bleeding, haemothorax
- Not High output failure as in other AVM because the same blood goes through the AVM as would the normal lung low pressure PA

Symptoms
- Pleurisy over AVM
- Spontaneous Haemothorax
- Polycythemia (in response to cyanosis) can then lead to haemoptysis, CVA

Investigations
- CXRAY
- Echo: Bubbles injected into right side after 5 cardiac cycles bubbles appear in the left atrium normally bubbles are filtered by capillaries
- CT
- Pulmonary Angiogram

Treatment
- Solitary AVM in cyanotic patient should be resected
- Interventional Radiological occlusion
- Endocarditis Prohylaxis

Congenital malformation of pulmonary vasculature in which there is a persistent communication that bypasses the pulmonary capillary bed
Pulmonary A-V Malformations

Congenital malformation of pulmonary vasculature in which there is a persistent communication that bypasses the pulmonary capillary bed
Bronchoalveolar Cancer

- Not generally associated with smoking
- Very often related to underlying scarring of the lung
- Age of peak incidence is around 50
- Occurs equally in both sexes
- As the incidence of smoking decreases, the incidence of BAC has increased
- Pathologically, BAC is characterized by its pattern of "lipedic growth"
- It appears to arise from type II pneumocytes
- Because these cells produce mucin, a subgroup of patients present with bronchorrhea
- Radiologically, the most common presentation is as a solitary mass
- It can also present as a unifocal or multifocal area of consolidation
- If it presents as a solitary nodule, its prognosis is better than other types of lung cancer
- Tend to be peripherally based often an air bronchogram sign reflecting the lipedic pattern of growth
Bronchiectasis

- Defined by morphology - Dilatation of Bronchi

Represents the end-stage of a variety of pathological processes

Abnormal, Irreversible Dilatation of the Bronchi
Bronchiectasis

Classification
- Cylindrical
- Varicose
- Saccular

Symptoms
- Chronic cough
- Recurrent infections
- Haemoptysis

Natural History
- Changed since antibiotics
- Now less common but they are
  - Recurrent infections
  - Abscess
  - Empyema
  - Respiratory failure
  - Cor pulmonale
  - Haemoptysis
Bronchiectasis

- Haemoptysis occurs in up to 41% of patients. Occasionally massive haemoptysis.

- Cyanosis and finger clubbing now rare.

- Bronchoscopy is important for diagnosis and for treatment. CT for diagnosis.

- Treatment is mainly medical:
  - Airway management, postural drainage, chest percussion, assisted cough techniques.
  - Antibiotics.

- Indications for Surgery:
  - If medical treatment is unsuccessful.
  - Recurrent haemoptysis.
  - Lung abscess.
  - Indeterminate mass.

- The disease should be localised enough to resect.

- Best results achieved if confined to one lobe only usually if lower lobe.

- All tips and hints for avoiding BPF.
Post-Pneumonectomy Space

- Following Pneumonectomy pleural space gradually fills with serosanguineous fluid until hemithorax is full.

- All air is absorbed.

- Average time to full radiographic filling is 4/12 with extremes of 3 weeks to 7 months.

- Progressive decrease in size of this space with mediastinal shift, elevation of hemidiaphragm.

- In 1/3rd of patients the space is totally obliterated with fibrosis helping.

- In 2/3rd of patients the space contains fluid indefinitely which can then become infected.
**Post-Resection Spaces**

- Depends on the nature & extent of resection
- The condition of the underlying lung
- Do not forget that failure to expand remaining lung may bas a result of endobronchial obstruction
- Whether air or fluid fills the space
- If air fills the space then as long as space improving (make take time) just observe
- If air fluid level is present this may signify infection or blood collection beware!
  - Has potential to cause fibrinous peel and prevent lung expansion
  - Is it infected?
  - If infected space options are
    - Drainage and accept as endpoint in elderly and debilitated
    - Sterilize space namely decortication
    - Thoracoplasty
Bronchopleural Fistula

Usually associated with infected pleural space (Empyema)

Management depends on:

- Aetiology
- Location (central airway or peripheral)
- Associated complications

Aetiology

- Postoperative (incidence following resection up to 3%)
  - Surgical technique, how the bronchus was stapled
  - Adjuvant therapies (Chemo or radiotherapy)
  - Infection particularly when lung resection was carried out for benign infective process
  - Associated illness; Chronic nutritional deficiency or steroid therapy
  - Recurrent tumour

- Spontaneous fistula
  - Pneumonia
  - Lung abscess
  - TB
  - Spontaneous pneumothorax particularly secondary

- Trauma
  - Following penetrating or blunt airway injury
Bronchopleural Fistula

**Diagnosis**
- Suspect a BPF associated with an empyema
- Persistent air leak following ICD insertion
- Failure of Ipsilateral lung to re-expand
- Recurrent contralateral pneumonias
- Drop in fluid level usually to level of bronchial stump on erect CXRAY (Post-Pneumonectomy)
- CXRAY, Bronchoscopy, CT scan

**Management**
- **General** – IV fluids, antibiotics, chest physiotherapy, posture lay patient operated side down to protect other lung
- **ICD** – All pleural collections + Suction. Beware suction may make air leak or gases worse
- **Surgery providing:**
  - No recurrent tumour
  - Control of any associated sepsis/empyema
  - Acceptable anaesthetic risk
- **Peripheral fistulae**
  - Generally seal providing full re-expansion of remaining lung parenchyma
  - Occasionally surgical intervention with stapling or direct suture of lung is required with associated pleurectomy
- **Central**
  - Fibrin glue use has been disappointing
  - Thoracotomy with closure of fistula with pedicle re-enforcement
  - Associated empyema cavity should be treated possibly thoracoplasty
**Bronchopleural Fistula – Post Pneumonectomy**

- Incidence 2-13% post Pneumonectomy
- Right more than left Blood supply of right bronchials is 1 artery versus 2 on left, left shielded by aorta
- Increasing age, Diabetics, steroids, poor nutritional state, prior radiotherapy, not re-enforcing suture
- Mortality can be as high as 30%
- Peak incidence is between 2\textsuperscript{nd} and 3\textsuperscript{rd} post-operative weeks
- Patients with late BPFs are less likely to aspirate/die due to fibrothorax formed by then
- BPF is the main cause of post-Pneumonectomy empyema (Go hand-in-hand 80% association)

**Aim of therapy is to prevent death by aspiration**

- **1–2 weeks post-operative**
  - Drain Pneumonectomy space (No suction)
  - Bronchoscopy to evaluate bronchial stump
    - If stump is grossly intact treat conservationily
    - If large defect re-operation to close fistula with pedicle re-enforcement, 2 large drains for irrigation close or perform window if excessive soil
    - If small defect Glue?
- **Over 2 weeks post-operative**
  - Drain space with ICD
  - When stable convert to open drainage
    - Thoracostomy / Eloesser flap
    - Close thoracostomy 3-6 months later (Claggett procedure)

**Timing is important**
Bronchopleural Fistula – Post Pneumonectomy

Early within 2 weeks BPF with Empyema
- Aspiration risk is high surgical emergency immediate drain, and start antibiotics, stabilise
- Objectives are then to close the fistula and sterilise the post-Pneumonectomy space
- Assess extent of Fistula by Bronchoscopy then treatment dependent on size
- If < 3mm try glue via Bronchoscopy
- If large disruption seen repeat thoracotomy with pedicle re-closure of Bronchus to close fistula
- Either
  - Leave large irrigation drains continuously irrigate until negative cultures then fill chest with antibiotic solution and remove drains
  - Or open thoracic window (Claggett window) which then needs closing either by muscle flap or simple skin closure

Late after 4 weeks up to 3 months BPF with Empyema
- Aspiration risk is reduced now due to fibrothorax more elective planning
- Still drain however closed but often closed drainage will not be adequate and will need open drainage
- Again assess extent of BPF with Bronchoscopy
- Sometimes if small defect simple closed drainage may be enough to allow closure
- Mostly however Open drainage with a window then daily inspection of cavity
  - Open drainage (Claggett) rib excision, 3, 4 ribs no more than 10 cm in length, leave BPF alone, pack wound
  - This approach minimises morbidity in sick patients by not attempting to repair the BPF
  - Sometimes again the BPF will close with adequate open drainage
  - Following sterilisation of the space elective repair of the fistula with muscle and wound closure with muscle flap
- Some advocate closure via median sternotomy initially with drainage of the space
- Some advocate going in through the thoracotomy incision, repairing the fistula, & closing the wound over drains

Timing is important
1963, Clagett and Geraci described a technique for the management of PNE that was based on sound surgical principles for the treatment of an abscess.

The 2-stage procedure consisted of open pleural drainage, closure of the BPF, removal of the necrotic tissue, and secondary closure or obliteration of the pleural cavity with antibiotic solution.

The Clagett procedure has been reported to be effective in 88% of patients, with failures resulting from persistent or recurrent BPF.

Two to four rib segments (generally not more than 10 cm long) were resected to widely expose the empyema cavity. The skin was advanced and brought down to the parietal pleura. Pus and necrotic debris were gently removed, and the cavity was filled with povidone iodine–soaked gauze. Wound inspected then patient can be discharged.

Once space is sterilised and BPF closed the cavity is then filled either with antibiotic solution and skin closed or some advocated bringing in a muscle flap.
Chest Wall

- Multiple layers based on a bony framework
- Ribs and costal cartilages are hinged upon the vertebra posteriorly and sternum anteriorly
- Neurovascular bundle inferior border of each rib, IMA, Aorta, Subclavian artery
- Muscular layers (3)
  - Innermost - intercostals; Transversus, internal, External intercostals muscles of respiration
  - Muscular attachments to superior and inferior apertures of chest wall, including SCM and Scalenes superior; y and rectus abd. Inferiorly
  - Muscles of arm function, Pectoralis Major and Minor, Lat Dorsi, Trapezius

Function

- Rigid shell for protection of thoracic viscera
- Respiration – By elevation and depression of ribs
- Framework acts as a platform for arm movements

Goals of Chest Wall Reconstruction

- Complete resection of tumour or infection
- Stabilise rib cage
- Soft tissue cover
- Return to function

Indicated when > 4 ribs or greater than 5 cm of chest wall are removed
Chest Wall Tumours

- Benign or Malignant, Primary or Secondary
- Majority are asymptomatic presenting with a painless lump
- Pain is sinister for malignancy
- History is important duration of lump, previous history of malignancy

Investigations

- **CXR**
  - Soft tissue mass may be seen
  - Lytic (metastasis, plasmacytoma) or sclerotic lesions on ribs (popcorn calcification in chondrosarcoma), mixed (Osteosarcoma)

- **CT**
  - Rapidly expanding, adherence to surrounding tissues, metastasis are all signs of malignancy
  - Note the mass seen is often smaller and only a part of the overall tumour mass therefore CT is essential

- **MRI**
  - Useful for mediastinal invasion or neurovascular involvement

- **PFTs, ECG etc.**
  - Must be able to tolerate procedure

- **Bronchoscopy**
  - If history dictates

Biopsy*

- Before treatment to identify: those that may need Chemotherapy or Radiotherapy Primarily, Secondaries, Inflammatory
- Core Needle or trucut with or without CT guidance – may seed tumour
- Incisional biopsy rarely needed now in view of CT guided biopsies
- Excisional Biopsy
  - Reserved for small < 4cm lesions

*If < 4cm take it out. If > 4cm incisional biopsy preferred then wide local resection including incisional site to reduce seedlings from biopsy
Chest Wall Tumours

Benign (All are Primaries)

- Soft tissue
  - Lipomas
    - Occur in subcutaneous tissue
    - Long history, painless lumps
  - Intra-muscular Lipoma
  - Desmoid – Intermediate tumours never metastasise but recur locally (some consider them as low grade Fibrosarcomas)

- Ribs
  - Osteochondroma
  - Chondroma
  - Fibrous Dysplasia
    - Most common benign chest lesion 30% of benign tumours

Malignant (Primary)*

- Soft tissue
  - Liposarcoma
  - Malignant Fibrous Histiocytoma
  - Fibrosarcoma

- Ribs
  - Chondrosarcoma (popcorn calcification near costo-chondral junction)
  - Ewings Sarcoma
  - Osteogenic Sarcoma
  - Plasmacytoma which is localised form of myeloma. If see this look for systemic Multiple Myeloma

*In decreasing frequency; Chondrosarcoma, fibrosarcoma, multiple myeloma, Ewings, Osteosarcoma
Chondrosarcoma
- Low-grade highly curable if adequately resected
- Requires wide local excision with 4 – 5 cm margins
- Local recurrence is most common therefore wide local excision is essential
- Not responsive to Chemotherapy or Radiotherapy therefore if they recur re-excite

Ewing’s Sarcoma
- Highly vascular tumour affecting young males
- Most frequent chest wall tumour in children
- Chest wall lesions comprise 7% of all Ewing’s Sarcomas
- Very radiosensitive but if can be resected then resect
- Chemotherapy is integral part of treatment

Osteogenic Sarcomas
- Childhood and adolescence
- 80 –90 % affects long tubular bones
- Treat with Chemotherapy then surgery
- If recurrence further chemotherapy and if possible surgery

Secondaries to chest wall through haematogenous spread to Parietal pleura then chest wall
Chest Wall Tumours - Surgery

Reconstruction

- Stability
  - Rigid reconstruction reduces need for prolonged post-operative ventilation particularly of sternum and anterior defects
  - Methylmethacrylate cement is filled in as a sandwich between two Marlex meshes
  - This composite prosthesis is then sutured in with non-absorbable sutures

- Blood Supply / Muscle / Soft tissue
  - Reduces the risk of infection bringing in a good blood supply
  - Obliterate the space
  - Covering the Marlex mesh – Methylmethacrylate sandwich
  - Either Musculo-cutaneous or Omentum
  - Some advocate just Musculo-cutaneous flaps to close defects arguing the lack of a need for rigid stability and reducing Foreign material

Prognosis

- Histology
- Adequate resection margins
- Presence of distant metastasis
Chylothorax

- Lymph flow in duct – 1.4ml/Kg/hr 70Kg 100mls/hr or 2.4 L/day
- 95% comes from liver and intestines
- 0.4 – 6 g of fat / 100mls
- 60 – 70% of diet fat is carried in duct
- Contains neutral fat, FFA, Cholesterol, phospholipids, plasma proteins
  - Neutral fats found as chylomicrons
  - Smaller fatty acids absorbed directly into portal venous system
  - Basis for use of medium chain fats in diet for conservative management of chylothorax
  - Main cellular element is lymphocytes

Aetiology

- **Congenital** – Duct atresia
- **Traumatic** – Blunt, Penetrating, Surgery
  - Cervical – Radical neck dissections, lymph nodes
  - Thoracic – Oesophagectomy, Sympathectomy, PDA, Coarctation, thoracic aneurysms, left pneumonectomy
  - Abdominal - Symphathectomy
- **Neoplasm** – Direct invasion of duct, rupture of distended tributaries due to obstruction
- **Infections** – TB lymphadenitis, filariasis
- **Venous thrombosis** – left subclavian and Jugular veins, SVC obstruction
The Thoracic Duct

- Origin: Cisterna Chyli on L2
- Enters Thorax – Aortic Hiatus T12-10
- Ascends on Right
- Crosses to left T5/4
- Drains L Jugulo/Subclavian Junction

Variations are common

- 2-3 mm in diameter
- Paper thin
- Valved
- Muscular peristaltic
- Multiple lymphaticovenous anastomoses throughout its length so can be tied off
- Damage on R – R effusion, above T4 L effusion

Origin - Cisterna Chyli on L2
Chylothorax – Presence of lymph in Pleural Space

**Presentation**
- **Acute** – SOB, fatigue, discomfort on affected side, if 2.4l loss cardiovascular instability
- **Chronic** – Loss of FFA, fat soluble vitamins, antibodies, proteins, malnutrition

**Diagnosis**
- Milky effusion that does not clot, Alkaline pH 7.4 – 7.8, High in Triglycerides, low in Cholesterol
- **Triglyceride** - Chyle > plasma typical level of more than 110mg/100ml
- **Cholesterol** - Chyle < plasma
- Chol/TG ratio < 1
- Total protein 2 – 6 g / dl
- High lymphocyte count
- Positive Sudan stain

**Treatment – Conservative at first ( 2/52)**
- Drain and expand lung with serial X-rays to ensure that drain is not blocked
- Support nutrition – Stop oral feed (including water (R Page), TPN, role of medium chain TGs
- Consider longer conservative therapy if contra-indication to surgery, vertebral fractures, etc…
- Surgery if after Oesophagectomy more likely e.g. 5-7 days post-op with 800mls/day loss

**Surgery**
- 2-3 hours pre-op 200 ml olive oil down ng, aspirate remaining stomach at induction, or IV Evans blue into leg, or Propofol!
- Go in side of leak and identify jet of chyle
- Over sew with pledgets until bone dry
- +/- Pleurectomy on same side
Carcinoid Tumours (General)

- Incidence 1: 75 000
- Neuroendocrine tumours with Amine Precursor Uptake and Decarboxylase (APUD) Arise form Kulchitsky cell
- 90% of Carcinoid tumours arise from GI tract ileum, appendix (Then Bronchus, then Gonads)
- 50% of patients with Carcinoid tumours will have Carcinoid Syndrome
- Of patients with Carcinoid Syndrome, 50% will have right sided heart involvement
- Carcinoid tumours are slow growing with a prolonged course of upto 20 yrs from development of carcinoid symptoms
- Development of Cardiac disease heralds a decline in clinical outcome
- Symptoms of Carcinoid Syndrome as a result of 5HT, Histamine, Prostaglandins secretion
  - facial flushing
  - intractable secretory diarrhoea
  - bronchoconstriction
- For heart to become involved from GI Carcinoid must have liver metastasis
- Cardiac lesions on TV or PV are Fibrous plaques sometimes on SVA as well leading to Stenosis or regurgitation
- Cardiac manifestations are caused by the paraneoplastic effects of vasoactive substances
- Diagnosis is based on Echo and 24 hour Urinary 5 HIAA levels
  - 5-hydroxytryptamine (5-HT or serotonin), histamine, tachykinins, and prostaglandins
  - Not direct metastatic involvement of the heart.
  - Ordinarily, the vasoactive tumour products are inactivated by the liver, lungs, and brain, but the presence of metastases allows large quantities of these substances to reach the right side of the heart without being inactivated by the liver.
  - The preferential right heart involvement is likely related to inactivation of the vasoactive substances by the lungs
  - In the 5–10% of cases with left sided valvar pathology, suspect extensive liver metastases, bronchial carcinoid, or a PFO
Carcinoid Tumours (Lung)

- Accounts for 5% of all lung cancers (Wrongly previously called bronchial adenoma)
- Neuroendocrine tumours with Amine Precursor Uptake and Decarboxylase (APUD)
- Arise from Kulchitsky cell of respiratory epithelium – if secrete 5HT Carcinoid syndrome
- 80% are found in major Bronchi within Bronchoscopic vision and on the right
- Classic Mulberry lesion which may bleed profusely on biopsy
- Divided into Typical and Atypical tumours (Small Cell is a distant cousin)

**Typical**

- 90% of all carcinoids
- Invade locally (only 5-15% metastasise)
- Treated by wedge or segmental resection if peripheral, if central Lobe/ Pneumo or bronchoplastic
- Must ensure enough of a margin on resection (locally recur)
- 10 year survival of 90% irrespective of size of tumour or even nodal status

**Atypical**

- More aggressive with 50 –70% local nodal metastasis
- Surgery is still the best option
Diaphragm

- Muscular component morphological derivative of the innermost (transversus) layer of the muscle of body wall
- Therefore in continuity with transversus abdominis muscle
- Develops from 4 structures however, Transverse septum, pleuroperitoneal membranes, Oesophagus, lateral Muscle
- Right is higher than left – Right up to nipple in expiration, left up to 5th ICS
- Inverted J with long limb extending up from crura (lumbar vertebrae) short limb to Xiphi
- Large right crus fixed to the upper 3 lumbar vertebrae, Smaller left crus fixed to upper 2 vertebrae
- Fibres for each overlap and pass vertically upwards before curving forward into the central tendon
- Some fibres from right on abdominal surface cross over to left and encircle Oesophagus
- Fibres from each crus unite with one another in front of the aorta to form the median arcuate ligament
- The central tendon is shaped like a club in a playing card and is fused with the fibrous pericardium
- Aortic opening T12 behind median arcuate ligament – Aorta, azygous v with thoracic duct in between
- Oesophageal opening T10 with Vagus and L gastric branch to Oesophagus
  - Lies in the fibres of the L crus but fibres from R crus encircle it as a sling
  - Phrenico-Oesophageal ligament attaches the Oesophagus to R crus sling (stretched in sliding hiatus hernia)
- IVC opening T8 right of midline in central tendon with right Phrenic nerve
- Motor supply to each hemidiaphragm solely from the corresponding Phrenic nerve
  - Reaching the abdominal surface each divides into anterior posterior and lateral branches
  - These run radially giving branches that enter the muscle from below (incisions are therefore radial)
Diaphragm

Fig. 5.29 Retroperitoneal viscera on the posterior abdominal wall.
Morgagni & Bochdalek Hernias

**Morgagni**
- Result of failure of the *transverse septum* to fuse with the sternum
- *Triangular space* between the Xiphisternum & the costal margin fibres that insert into the central tendon
- Most defects are on the right since the pericardium protects the left
- Usually have a sac and can contain large bowel
- Diagnosed incidentally rarely symptomatic
- **Repair through abdomen to remove risk of future strangulation**

**Bochdalek**
- Result of failure of *pleuro-peritoneal membranes* to develop
- Located *posterolateral* and usually on the left containing stomach, spleen, colon
- If occurs on right liver usually hides the defect and prevents complications
- Usually presents with severe symptoms in the adult with obstruction or strangulation
- Look at PA and Lateral CXRAY
- **Repair on diagnosis to prevent complications or operate on presentation of obstruction**
- Operating in an emergent basis carries a much higher mortality
Empyema

- Most commonly occurs with Pneumonia
- Also seen after thoracic surgery, trauma. Extension of infection from neck or abdomen, or haematogenous spread

How it develops
- 57% of patients with Pneumonia have a para-pneumonic effusion
- In most cases effusion is not infected (Simple effusion)
- In 10% effusion becomes infected (Complicated effusion) first step of empyema akin to Exudative classification of stage
- Effusion then has a low PH<7.2, Glucose < 2.2, LDH> 1000IU/L
- In a minority of patients infected effusion progresses to macroscopic purulent effusions
- Purulent effusions demonstrate pro-coagulant properties and reduced fibrinolytic properties resulting in Fibrin deposition (Fibrinopurulent stage)
- In this stage loculations seen
- In latter stages pleura thickens and pleural cortex (peel or rind) Organising

Clinical features
- Fever, cough, SOB, Chest pain similar to Pneumonia
- Additional signs of pleural effusion, dull base, reduced breath sounds, reduced chest expansion
- Finger clubbing if chronic
- May discharge through skin – empyema neccissitans

Referral
- Patients with pneumonia that is not responding and a pleural effusion should be referred to chest physicians

Imaging
- Ultrasound – Effusion, loculations, guide aspirations
- CT scan with contrast may distinguish empyema from lung abscess

Reported mortality 7 – 33%
Empyema

Community Acquired

- **Aerobic organisms**
  - Streptococcus Pneumoniae – commonest organism
  - Staphylococcus aureus
  - Gram Negatives – E. Coli, Haemophilus Influenza, Klebsiella Pneumiae (Latter associated with worst prognosis and alcoholics)

- **Anaerobic organisms**
  - Most common is Bacteroides Fragilis

- **Antibiotics**
  - Target above with Augmentin to cover beta-lactamase producing Staph. A, 2nd generation Cephalosporins, + anaerobic cover

Hospital Acquired

- **Organisms**
  - Staphylococcus (Including MRSA)
  - Pseudomonas
  - Enterococcus

- **Antibiotics**
  - Must include cover for potential MRSA therefore Vancomycin

Principles of treatment

- General supportive measures
  - Nutrition
  - Antibiotics
  - Drainage

- Multi-disciplinary approach
  - Chest Physicians
  - Microbiologists
  - Radiologists
  - Surgeons

BTS At least 3 weeks of Antibiotics
Empyema

No Consensus in treatment

Collection of purulent fluid in pleural cavity

Medical: Organisms in fluid, increase in polymorphs, pH, Glucose, increase in LDH, +ve culture (50%)

Surgical: Macroscopic appearance of frank pus with thickened visceral and parietal pleura healing by fibrosis
- Cortex is thickening of visceral pleura with eventual adherence to parietal pleura
- Secondary to pneumonia
- Trauma
- Medical interventions, needle aspiration, ICD

Classification – three groups
- Exudative – thin fluid with WBC on microscopy. Lung re-expands when fluid is drained
- Fibrinopurulent – Fibrin with loculations. Lung will expand when most fluid pockets are drained, loculi / adhesions cleared
- Organising / chronic – Thick cortex over visceral pleura
- Classification relies on radiology demonstrating loculations or cortex
- Not clear-cut – Differentiation between Fibrinopurulent and organising is often made at surgery

Treatment is defined by stage of disease

Complication –
- empyema necessitatis-dissection of pus through the soft tissues of the chest wall and eroding through the skin
- Fibrothorax

PH <7.2, Glucose <2.2mmol/L, LDH >1000IU/L (Exudate)
Empyema - Treatment

Exudative
- Surgery is not indicated unless progress into Fibrinopurulent
- Antibiotics
- Needle, CT/US guided aspiration or formal ICD drainage
- Fibrinolysis offers no advantage*

Fibrinopurulent
- ICD +/- Fibrinolysis*
  - If radiology is favourable
  - 40% failure rate (Colice et al review of 13 trials) and if this happens should not delay definitive surgery
- VATS (Cosmetic, less invasive, less pain, lower morbidity not substantiated by RCT results)
  - May provide superior drainage in loculated empyema. Often used as a preliminary assessment prior to thoracotomy
  - Achieves goal in 75% but 25% will require additional procedure. Shorter hospitalisation and need for chest drainage
  - 1 RCT of VATS Vs ICD + fibrinolysis – only 20 patients therefore can not be generalised **
  - Conclusion; Can not be currently recommended due to absence of data
- Thoracotomy + Decortication
  - More morbidity therefore recommended as third option for fibrinopurulent
- Rib Resection
  - Less invasive than thoracotomy or VATS more applicable to sick patient
  - 90% success rate and applicable to majority of patients

* See Next Slide **See later slide
Empyema – Fibrinolysis - Trials

Cochrane Database of Systemic Reviews (meta-analysis)

- 3 double-blind RCT comparing Streptokinase to Saline Placebo
- Compared to saline, streptokinase led to:
  - Quicker resolution of fever
  - Better resolution on CXRAY
  - Less surgical intervention
- Authors concluded however: results not consistent from trial – trial, evidence inadequate to clearly recommend use

Maskell et al, NEJM, 2005

- RCT 44 patients Ultrasound guided drainage and Streptokinase Vs Saline
- Streptokinase group achieved treatment success, better control of infection, better CXRA resolution

Cameron, Am J Resp Med, 2004

- RCT 427 patients Ultrasound guided drainage and Streptokinase Vs Saline
- Streptokinase group NO BENEFIT

Conclusion

- Unproven value and should probably not be done

Largest RCT (427 patients) showed no benefit of Fibrinolysis
Organising Empyema

- Decortication
  - Likely to be required in patients with > 4 weeks of illness/empyema
  - As effective as rib resection in success rate but more applicable to advanced stages
  - Allows resection if needed to obliterate empyema cavity
  - Most likely technique to result in full lung expansion
  - More morbidity therefore recommended as third option for fibrinopurulent
  - VATS is useless

- Open Drainage
  - Applicable to frail patients too ill to undergo thoracotomy
  - Involves rib resection in most dependent part (9th Rib posteriorly)
  - Skin is stitched to pleura to prevent premature closure
Empyema – Treatment - Results

Surgery Vs non-surgical

- Cochrane Database of Systematic Reviews, 2005, Coote N et al (this paper already mentioned in previous slides)
  - 20 adults with empyema with loculations or Ph<7.2, randomised to immediate VATs Vs ICD + Fibrinolysis
  - VATS showed superior success, fewer days with ICD in, shorter hospital LOS
  - 50% failure rate in ICD + fibrinolysis group
  - However high failure rate in control group
  - Need for surgery was decided in an un-blinded fashion
  - Conclusion: Precise role and optimum timing for VATs is unclear

- Also results of VATs, Prospective observational study 178 patients
  - Addressed fibrino-purulent stage
  - 44% of VATs group had to be converted to Thoracotomy
  - Delayed referral to Thoracic surgeons as well as Gram Negative Organisms were predictors of VATs failure
Lung Abscess
Lung Abscess

Local area of suppuration and cavitation

Primary “nonspecific”

- Most occur as a result of aspiration of infectious debris from oropharynx
- Right lung usually affected and posterior segment by gravity
- Anaerobic organisms common, then, Streptococcal, Staphylococcal
- History of URTI, febrile, toxic, offensive sputum, haemoptysis
- Suppurative Pneumonitis then occurs and can result in liquefaction necrosis

Secondary “opportunistic”

- Associated with malignancy, immunosuppression, steroids, blood dyscrasias, ...
- Usually multiple
- Organism usually Staphylococcus or Pseudomonas
**Lung Abscess**

**Indications for surgical management of lung abscesses**

- large size: diameter greater than 4 cm
- failed antibiotic therapy with persisting sepsis
- chronicity: 6-8 weeks without improvement
- inadequate resolution on X-ray: wall thickening, local bronchiectasis
- significant recurrent haemoptysis
- infective complication: bronchopleural fistula, empyema
- resectable malignancy: Stage I or II proximal obstructing bronchial or peripheral cavitating tumour
- other pre-existing lung pathology: lung cysts, pulmonary sequestration, broncho-oesophageal fistula, cavitated tuberculosis

**Surgical**

- Consider Radiographically placed drains
- If these fail consider Cavernostomy, essentially I & D with a large drain
Lung Cancer Aetiology

1. **Smoking**
   - Responsible for 85% of lung cancers

2. **Environmental Risk Factors**
   - Asbestos, Chromium, Silica, Nickel, Arsenic
   - Accounts for 3 – 17% of lung cancers

3. **Dietary**
   - Increased with increase in dietary fat/Cholesterol
   - Decreased with fruit and Vegetables, Retinoids,

4. **Pre-existing Lung Disease**
   - COPD
   - Fibrosis
   - Lung Scarring from TB / Infarction

5. **Congenital**
   - Limited case reports

6. **Molecular Genetic Alterations**
   - Numerous genetic alterations have been described in lung cancer

---

Top Cancer Killer in Men and Women. Accounts for 25% of all cancer deaths in USA
Lung Cancer Pathology

1. Squamous Cell
   - Most common 40–70%, centrally located, more common in men, Keratin pearls
   - Stage I – 5 year survival 50%

2. Adenocarcinoma
   - 5–15%, peripherally located, more common in women
   - Stage I – 5 year survival 55%

3. Undifferentiated
   - 20–30% 2 sub-types
     - Large cell – aggressive
     - Small cell – oat cell

4. Bronchoalveolar
   - Uncommon 3–7%
   - Favourable prognosis
   - Stage I – 5 year survival 60%

NSCCL 80% SCC 20%

25% Stage I&II, 30% stage III, 40% stage IV
Lung Cancer NICE

1. **CXRAY**
   - Immediate for Haemoptysis or
   - > 3 weeks cough, SOB, Hoarseness, Clubbing, weight loss, supraclavicular lymph nodes
   - If CXRAY is positive or CT is positive referral immediately to member of Thoracic Oncology MDT

2. **PET**
   - Every centre should have access to PET scanner

3. **Radiotherapy**
   - Medically inoperable patients with Stage I/II should be offered Radiotherapy CHART
   - CHART (Continuous Hyperfractionated Accelerated Radiotherapy)

4. **Chemotherapy**
   - Should be offered to patients with stages IIIA and above if performance status is 0-1

Top Cancer Killer in Men and second to breast cancer in Women
Lung Cancer NICE - PET

Every cancer network should have a system of rapid access to FDG-PET scanning for eligible patients. [D(GPP)]

Patients who are staged as candidates for surgery on CT should have an FDG-PET scan to look for involved intrathoracic lymph nodes and distant metastases. [A(DS)]

Patients who are otherwise surgical candidates and have, on CT, limited (1–2 stations) N2/3 disease of uncertain pathological significance should have an FDG-PET scan. [D(GPP)]

Patients who are candidates for radical radiotherapy on CT should have an FDG-PET scan. [B(DS)]

Patients who are staged as N0 or N1 and M0 (stages I and II) by CT and FDG-PET and are suitable for surgery should not have cytological/histological confirmation of lymph nodes before surgical resection. [A]

Histological/cytological investigation should be performed to confirm N2–3 disease where FDG-PET is positive. This should be achieved by the most appropriate method. Histological/cytological confirmation is not required: [B(DS)]

> where there is definite distant metastatic disease

> where there is a high probability that the N2/N3 disease is metastatic (for example, if there is a chain of high FDG uptake in lymph nodes).

When an FDG-PET scan for N2/N3 disease is negative, biopsy is not required even if the patient's nodes are enlarged on CT. [B(DS)]

• Summary

- If you are surgical candidate (I/II) NICE says do a PET first
- If the patient has CT N2 nodes (III and above) do a PET
  - IF PET is positive you must confirm this histologically
  - If PET is negative go ahead and resect (Contentious) False negative seen in adenos
  - If PET is not available CT N2 nodes must be sampled

Top Cancer Killer in Men and second to breast cancer in Women
Lung Cancer Fitness For Surgery (BTS) (4)

1. Age
   - Advanced age increases morbidity therefore careful workup of elderly
   - Surgery is just as effective for stages I and II for patients > 70 years-old as it is for < 70
   - Age should not be a factor for offering a Lobectomy
   - Age (>80) should be considered if offering a Pneumonectomy

2. RFT
   - IF FEV > 1.5 l no further spirometry is indicated for patients undergoing a Lobectomy
   - IF FEV > 2 l no further spirometry is indicated for patients undergoing a Pneumonectomy
   - If patient is being denied surgery based on poor spirometry follow 3 steps below;
     - Step 1 – Perform formal RFTs with TLCO, Blood gases, Quantitative VQ if considering Pneumonectomy
       Calculate epo FEV1 based on preop FEV1 and either VQ for Pneumonectomy or BPS for Lobectomy
     - Step 2 – If epo FEV1 > 40%, epo TLCO > 40 %, & Sats > 90% on air ==Average Risk
       If FEV1 < 40%, epo TLCO < 40 %, & Sats < 90% on air ==High risk. Any other combination step 3
     - Step 3 – Shuttle test – If < 250m or desaturation by > 4% == High risk. If still equivocal cardiopulmonary testing; if Vo2 peak < 15ml/Kg/min ==High Risk

3. Cardiovascular
   - All should have ECG
   - If Murmur is heard perform Echo
   - Do not operate on earlier than 6 weeks following myocardial infarction
   - If patients have had an MI within 6/12 of proposed surgery cardiology review
   - Follow ACC/AHA guidelines for estimating cardiovascular risk
   - Patients who have had a previous CABG should not be precluded from surgery

4. Performance status / Weight loss
   - Patients presenting with > 10% weight loss or WHO > 2 should be staged carefully for evidence of advanced disease
   - If nutritional status is poor, low albumin, expect greater morbidity post-operatively
<table>
<thead>
<tr>
<th>Zubrod/WHO Scale</th>
<th>Karnofsky Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>0  Asymptomatic</td>
<td>100  Asymptomatic</td>
</tr>
<tr>
<td>1  Symptomatic, but ambulatory</td>
<td>90   Normal activity, minor symptoms</td>
</tr>
<tr>
<td></td>
<td>80   Normal activity, some symptoms</td>
</tr>
<tr>
<td>2  In bed &lt; 50% of day (unable to work</td>
<td>70   Unable to work, care for self</td>
</tr>
<tr>
<td>but able to live at home with some</td>
<td>60   Occasional assistance with needs</td>
</tr>
<tr>
<td>assistance)</td>
<td></td>
</tr>
<tr>
<td>3  In bed &gt;50% of day (unable to care</td>
<td>50   Considerable assistance</td>
</tr>
<tr>
<td>for self)</td>
<td>40   Disabled, full assistance needed</td>
</tr>
<tr>
<td>4  Bedridden</td>
<td>30   Needs some active supportive care</td>
</tr>
<tr>
<td></td>
<td>20   Very sick, hospitalisation needed</td>
</tr>
<tr>
<td></td>
<td>10   Moribund</td>
</tr>
<tr>
<td></td>
<td>0    Dead</td>
</tr>
</tbody>
</table>
Lung Cancer Cardiovascular Risk Stratification (AHA/ACC)

**Major (4)**
- Unstable Coronary Syndromes, CCS III/IV or recent MI with evidence of ongoing ischaemia
- Decompensated Heart Failure
- Arrhythmias
  - High grade AV block
  - Ventricular arrhythmias with structural heart disease
  - SVT with uncontrolled ventricular response rate
- Severe Valvular heart disease

**Intermediate (4)**
- CCS I/II Angina
- Previous History of MI
- Compensated Heart Failure
- Diabetes

**Minor (6)**
- Advanced age
- Rhythm other than SR (eg) AF
- Abnormal ECG (LBBB, LVH, ST segment abnormalities)
- Previous CVA
- Uncontrolled hypertension
- Poor Functional status (inability to climb one flight of stairs)

*Major Risk Group* – Cardiology opinion, MDT, CABG if CAD is found - *Intermediate/Minor* – No further investigations if good functionally, ETT if not...
6 minute Shuttle Test

- Patient accompanied by nurse & doctor to designated area where distances are marked
- Need Pulse Oximeter, Oxygen Cylinder, stop-watch
- Explain test to patient put pulse Oximeter on
- Take Sats resting and start stop-watch
- Note number of stops patient takes for SOB
- Note maximum distance achieved in 6 minutes
- Reasonable correlation found between distance and VO2 max measured

**Cambridge Paper** 125 patients with lung cancer underwent shuttle and VO2 max assess

- 55 patients reached 400 m and had a VO2 > 15 ml / Kg / min
- The remaining 70 patients reached less than 400 m
- In this group 22 / 70 (30%) had VO2 < 15 mls / Kg/ min
- 17 reached < 250 m however 9 / 17 over half had VO2 > 15 mls / Kg / min

This paper concluded that 400 m was discriminatory distance if above no VO2 needed
- < 400 m should test VO2 particularly < 250 m because shuttle is not discriminatory
- Some patients with shuttle < 250 will have VO2 max > 15ml / Kg / min
Cardiopulmonary Testing

During exercise heart increases its Cardiac Output up to 20l/min
- HR goes up linearly with exercise due to increase Sympathetic tone as well as vagal withdrawal
- SV goes up by 50 – 100%

Rate of Oxygen absorption in lungs is increased by
- Partly due to increased Cardiac Output but
- also 3 fold increase in amount of O2 added to pulmonary blood by
- decreasing Venous Oxygen Saturations during exercise allowing 13-fold increase in Pulmonary Oxygen uptake

CPET non-invasive mean of assessing exercise response patients with Cardiac/Respiratory condition

Breath by Breath assessment of respiratory gas exchange during graded symptom limited exercise

Principal
- As exercise proceeds ATP is used up and therefore ATP generation is stepped up requiring more O2
- More O2 is taken up by mixture of increase in CO and greater O2 extraction
- Also CO2 production is increased
- According to Fick’s Equation O2 consumption = CO X A-V O2 difference
- However fit a person is there will come a time when the Cardiopulmonary unit can not deliver anymore O2
- This is the VO2 max when maximum CO and A-V O2 difference are maximal

Ultimate Maximal Oxygen Uptake and Anaerobic thresholds are obtained

Nose is clamped subject breathes in & out via mouthpiece so concentrations of gas flow measured

Treadmill / Bicycle ergometer exercise is started and sped up

HR, BP, Saturations, TV, as well as obviously the O2 and CO2 are all measured

In Heart failure the VO2 max reflects the Maximal Cardiac Output response

In Lung disease VO2 max reflects O2 delivery and extraction
Cardiopulmonary Testing

- There will come a time when ATP production is produced Anaerobically.
- Anaerobic Respiration results in build up of LACTIC ACID.
- LACTIC ACID is buffered by HCO3 to LACTATE and CO2.
- Excess Non-aerobic CO2 production is detected and marks the Anaerobic threshold.
- Anaerobic threshold is measured by looking at the VO2 / VCO2 ratios as exercise continues or.
- As the level of exercise at which this anaerobic threshold lowers this indicates impaired aerobic capacity or cardiac function.
- In patients with heart failure this point is around 70% of VO2 max.
- Patients with a respiratory problem:
  - rarely reach the anaerobic threshold
  - Desaturate quickly
  - Minute volume increases by increasing RR not TV.
Figure 1  Algorithm for selection of patients suitable for resection for lung cancer.
Staging of Lung Cancer

**CT**
- Determine tumour size (T stage) and potential resectability
- Determine Nodal involvement (N Stage) – Nodes in short axis > 1cm are considered abnormal
- Determine Possible Metastasis in Chest (Upper abdomen, adrenal glands) (M Stage)
- Sensitivity only 60 – 65 % for N staging (Dales meta-analysis false negatives 20%)
- Specificity only 60 – 70% for N staging (Dales meta-analysis false positives 21%)
- *Do not deny patients surgery on basis of equivocal CT findings*

**MRI**
- Not routine but reserved for when CT is equivocal
- Helps clarify mediastinal invasion
- Superior Sulcus tumours
- Chest wall and Diaphragm invasion

**PET**
- Based on the fact that *tumour cells have a higher rate of glycolysis* than surrounding normal cells
- FDG competes with glucose, is taken up, accumulates, and lights up (measured as standardised Uptake Value (SUV))
- Most sensitive tool for detecting nodal involvement both intra- and extra-thoracic
- Best used in conjunction with CT to localise nodal involvement (i.e., CT/PET, increased Sens & Spec)
- Sensitivity 88%
- Specificity 90% (*granulomas, infection, adenocarcinomas, atelectasis, aspergillomas, central tumours mistaken for N2*)
- Often shows involvement of nodes that are less than 1cm in diameter

**Bone Scan**
- Low yield with NSCCL therefore not routinely recommended unless symptoms or raised ALP

* 19 Causes of false positive PET scans (Bakheet and Powe)
Staging of Lung Cancer (T1)

< 3cm and totally surrounded by lung parenchyma or visceral Pleura. No Spread to Proximal Lobar Bronchus
Staging of Lung Cancer (T2)

Proximal Extent within one lobar Bronchus but > 2cm from Carina

Any size breaching visceral pleura

> 3cm
**Staging of Lung Cancer (T3)**

- Any size to chest wall
- Superior Sulcus Tumours Not with Pancoasts Syndrome (T4)
- < 2cm from Carina or Atelectasis of whole lung
- Parietal Pericardium
- Phrenic Nerve

**Diagram:**
- Diaphragm
- Atelectasis
- Superior Sulcus Tumours
- Pancoasts Syndrome
- Carina
- Parietal Pericardium
- Phrenic Nerve
Staging of Lung Cancer (T4)

Also: multiple nodes in same lobe

Pancoast’s Syndrome
- Brachial plexus (arm and shoulder pain)
- Sympathetic trunk (Horner’s syndrome)
- Subclavian artery and vein

Vertebral body
Vagus nerve
Recurrent nerve (vocal cord paralysis)

Trachea or Carina

Also

Pleural Effusion
Nodal Status

Superior mediastinal nodes
- 1 Highest mediastinal
- 2 Upper paratracheal
- 3 Prevascular and retrotracheal
- 4 Lower paratracheal (including azygos nodes)

N1 nodes
- 10 Hilar
- 11 Interlobar
- 12 Lobar
- 13 Segmental
- 14 Subsegmental

Aortic nodes
- 5 Subaortic (A-P window)
- 6 Para-aortic (ascending aorta or phrenic)

N2 = Single Digit, Ipsilateral, N3 = Single Digit, Contralateral or Supraclavicular
Cervical Mediasinoscopy

Very rarely access to Station 7

Mainly access to Stations 1 - 4
Stations 5 & 6 Can be approached through Left anterior Mediastinotomy
Relevance of N2 aorto-pulmonary window nodes

Solitary aortopulmonary window nodal involvement, (5, 6)
- Particularly with squamous cell carcinomas involving the left upper lobe
- Identified as a sub-population who can do well
- Five-year survival rate of 35-45% following complete surgical resection

Involvement of
- Subcarinal (7)
- Paraesophageal (8)
- Inferior pulmonary ligament nodal stations (9)
- Predicts limited survival even after complete resection
### TNM Staging

**Primary tumour (T)**

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tx</td>
<td>Primary tumour cannot be assessed or tumour proven by the presence of malignant cells in sputum or bronchial washings but not visualised by imaging or bronchoscopy</td>
</tr>
<tr>
<td>T0</td>
<td>No evidence of primary tumour</td>
</tr>
<tr>
<td>Tis</td>
<td>Carcinoma in situ</td>
</tr>
<tr>
<td>T1</td>
<td>Tumour &lt;3 cm in greatest dimension, surrounded by lung or visceral pleura, without bronchoscopic evidence of invasion more proximal than the lobar bronchus</td>
</tr>
</tbody>
</table>
| T2   | Tumour with any of the following features of size or extent:  
|      | • >3 cm in greatest dimension |
|      | • involves main bronchus >2 cm distal to the carina |
|      | • invades the visceral pleura |
|      | • associated with atelectasis or obstructive pneumonitis that extends to the hilar region but does not involve the entire lung |
| T3   | Tumour of any size that directly invades the following: chest wall (including superior sulcus tumours), diaphragm, mediastinal pleura, parietal pericardium; or tumour in the main bronchus <2 cm distal to the carina but without involvement of the carina; or associated atelectasis or obstructive pneumonitis of the entire lung |
| T4   | Tumour of any size that involves any of the following: mediastinum, heart, great vessels, trachea, oesophagus, vertebral body, carina; or tumour with a malignant pleural or pericardial effusion, or with satellite tumour nodule(s) within the ipsilateral primary tumour lobe of the lung |

**Regional lymph nodes (N)**

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Nx</td>
<td>Regional lymph nodes cannot be assessed</td>
</tr>
<tr>
<td>N0</td>
<td>No regional lymph node metastases</td>
</tr>
<tr>
<td>N1</td>
<td>Metastases to ipsilateral peribronchial and/or ipsilateral hilar lymph nodes and intrapulmonary nodes involved by direct extension of primary tumour</td>
</tr>
<tr>
<td>N2</td>
<td>Metastases to ipsilateral mediastinal and/or subcarinal lymph nodes</td>
</tr>
<tr>
<td>N3</td>
<td>Metastases to contralateral mediastinal, contralateral hilar, ipsilateral or contralateral saclene or supraclavicular lymph nodes</td>
</tr>
</tbody>
</table>

**Distant metastases (M)**

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mx</td>
<td>Presence of distant metastases cannot be assessed</td>
</tr>
<tr>
<td>M0</td>
<td>No distant metastases</td>
</tr>
<tr>
<td>M1</td>
<td>Distant metastases present</td>
</tr>
</tbody>
</table>

**Stage grouping TNM subsets**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Carcinoma in situ</td>
</tr>
<tr>
<td>IA</td>
<td>T1N0M0</td>
</tr>
<tr>
<td>IB</td>
<td>T2N0M0</td>
</tr>
<tr>
<td>IIA</td>
<td>T1N1M0</td>
</tr>
<tr>
<td>IIB</td>
<td>T2N1M0</td>
</tr>
<tr>
<td>IIIA</td>
<td>T3N1M0</td>
</tr>
<tr>
<td>IIIB</td>
<td>T4N0M0</td>
</tr>
<tr>
<td>IV</td>
<td>Any T Any N M1</td>
</tr>
</tbody>
</table>
Clinical evaluation for distant metastases

- Positive
  - Imaging and/or biopsy of potential metastatic sites:
    - Brain: MRI/CT
    - Bone: X-ray and bone scan or MRI if required
    - Liver and adrenals: CT if not performed already

- Negative
  - Examine chest CT scan
    - But:
      - Consider other techniques such as ultrasound or surgical assessment for mediastinal and chest wall invasion as CT alone may not be reliable.
      - Use MRI if necessary to assess extent of disease for superior sulcus tumours.
      - NB: MRI scanning should not be routine in T-staging

- Is the patient in one of these groups:
  - a candidate for surgery
  - a candidate for radical radiotherapy
  - a candidate for radical combination treatment
  - has limited N2/3 disease (1–2 stations) of unknown pathological significance and is otherwise a surgical candidate?

  - Yes
    - PET scan
    - Positive
      - Histological sampling of suspected N2/3 disease (nodes with a short axis greater than 1 cm on CT) for patients being considered for surgery or radical radiotherapy
    - Suspicious
    - Negative
      - Histological/cytological confirmation not required

  - No
    - Histological/cytological confirmation of appropriate site

Positive for mediastinal disease or distant metastases (definite distant metastases or high probability that N2/3 disease is metastatic, for example there are chains of high FDG uptake)

Histological/cytological confirmation not required

Suspicious for mediastinal disease or distant metastases

Histological/cytological confirmation of appropriate site

Negative for mediastinal disease and distant metastases

Histological/cytological confirmation not required
# Impact of Staging on Post-surgical Survival

<table>
<thead>
<tr>
<th>Stage</th>
<th>5-Year Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia(T1N0)</td>
<td>80%</td>
</tr>
<tr>
<td>Ib(T2N0)</td>
<td>50%</td>
</tr>
<tr>
<td>II (T1N1 IIA, T2N1, T3N0 IIB)</td>
<td>25-35%</td>
</tr>
<tr>
<td>IIIa (N2)</td>
<td>10</td>
</tr>
</tbody>
</table>

If you combine Ia and Ib, 5 yr survival is 60%, 30% will relapse with Metastasis.
Role Of Adjuvant Therapy

Chemotherapy
- Most studies have addressed its role in stage IIIA (N2) disease
- No studies have looked at its role stages I and II
- Induction Chemotherapy in Stage IIIA or Stage IIIB is still considered experimental
- Use of multiple agents not just one
- Patients with known stage IIIA disease preoperatively have a low chance of being cured by surgery alone; Add Chemotherapy?

Radiotherapy
- Reserved for patients who are either unfit or refuse surgery in Stages I or II
- High local failure rates for T1 and T2 tumours
- Trials addressing its role post resection in N1 and N2 disease have shown no benefit in survival
- Has been used for Pancoast (T3) Superior Sulcus tumours in a preoperative and post-operative regimes
- Palliative radiotherapy offers good symptomatic relief, bone, cerebral metastasis
- Brachytherapy permits delivery of localised high dose of radiotherapy
- No Role for radiotherapy in patients with completely resected stages I and II lung cancer
Role Of Adjuvant Therapy Trials - Radiotherapy

**Radiotherapy**

- Medically inoperable patients survival 25% at 5 years, Locally advanced disease 15-20% at 2 years
- Better outcome if WHO performance status 0-1, Tumour < 3cm, at least 60 Gy or Chart given, No effect of age or Histology
- Improving outcome in stage III with induction chemotherapy NSCLCCG metanalysis Chemo in addition to radical RT (BMJ '95)
  - 4% absolute benefit in survival at 2 years
  - 2% benefit in survival at 5 years
- Post-operative RT (PORT meta-analysis Lancet 1998 9 RCT of post-operative RT after complete resection)
  - 7% Reduction in overall survival and 5% reduction in relapse free survival
  - Reduction in survival only seen in patients with stage I and II disease
  - 2 of studies showed improvement in loco-regional disease control in N2 disease
  - 1 of these showed delay in local or systemic relapse but survival did not reach significance
  - Concluded – No role for RT in completely resected stage I and II disease
  - In patients with N2 disease role of RT uncertain
  - No RCT looked at role of RT in incomplete resection but should consider RT in patients with positive margins

**Palliative Radiotherapy in NSCLC**

- MRC lung cancer working party
- Concluded good symptomatic palliation cough, Chest pain, Haemoptysis, anorexia
- Slight survival advantage 1yr 36% Vs 31%
- Shorter courses give good palliation as longer courses

**NICE – Post-op radiotherapy only for positive resection margins**
Role Of Adjuvant Therapy Trials - Chemotherapy

- Adjuvant post surgical use of chemotherapy in all stages
- NSCLCCG meta-analysis (12 studies enrolling early stage I, II, as well as IIIa)
  - Overall the use of post-operative chemotherapy was not recommended by this meta-analysis
  - There was a small absolute survival benefit of 5% at 5 years however but this did not justify its recommendation
  - Major shortcoming of the above metanalysis was outdated chemotherapy agents with toxicity and lack of compliance
  - 2 further studies Mineo & Le Chevalier (International Adjuvant Lung Cancer Trial IALT) show a striking survival benefit

NICE – Adjuvant Chemotherapy should be offered to all surgically resected patients irrespective of staging and completeness of resection
Role Of Neo-Adjuvant Therapy Trials - Chemotherapy

- May facilitate surgery
- May treat micro-metastasis early
- Reduce stimulus to cancer cells by surgery
- Most studies have addressed its application in stages IIIA

**Sloan-Kettering experience (1993) (Stage IIIA – N2 disease)**
- Neo-adjuvant Chemotherapy for N2 disease who had complete resections after major response to Chemotherapy had 41% 3 year survival

**CALGB 8935 study 1997 (Stage IIIA – N2 disease)**
- Patients down-staged by neo adjuvant Chemotherapy to N2 negative were more likely to benefit from surgical resection
- Conversely if residual N2 disease is seen following neoadjuvant chemotherapy this was a negative prognostic indicator

**Rosell NEJM (1994) (Stage IIIA – N2 disease)**
- 60 pts randomised to Chemotherapy pre-surgery, all given post-operative RT
- **Median survival benefit 26 V 8 months**

**Roth J Natl Cancer Inst (1994) (Stage IIIA – N2 disease)**
- 60 pts randomised to Chemotherapy pre-surgery, responders had post-operative chemotherapy
- **Survival benefit 64 V 11 months survival**

**NICE** – Surgery alone for stage IIIA patients carries a poor result therefore these patients should be discussed in the contexts of a MDT ? Neoadjuvant Chemotherapyin
Advanced Local Disease

Chest Wall (T3)
- About 5% of lung cancers extend across Parietal Pleura to involve Chest Wall
- Some T2 across Visceral Pleura may mimic Chest Wall Involvement
- IF performing extra-pleural strip and you encounter severe resistance consider Chest wall resection
- MRI may give more Clarity in resolution
- Must establish that there are no N2 disease
- Successful Surgery for T3 NO (Stage IIb) can give 5 year survival of up to 67% (if N1 survival is between 9 – 27%)
- Defects up to 3 ribs in the paraspinal or Scapular regions do not need to be reconstructed otherwise elsewhere use Marlex
- Role of postoperative Radiotherapy is controversial. If any doubt about resection margins use it!

Mediastinal Invasion (T3)
- Mediastinal pleura, fat, nerves, pericardium, have poor outcome since have lower incidence of complete resection
- 5 Year survival only 30%

Carinal (T3)
- Affected negatively by peri-bronchial invasion 5 year survival if present is only 30% if absent 80%

Tumour adherent to Vertebral Column (T4)
- Some reports of curative resections following removal of part or even whole thoracic vertebra

Superior Sulcus (Pancoast) tumours (T3/T4)
- Involvement of Brachial plexus, upper ribs, stellate ganglion, Subclavian vessels
- Without treatment life expectancy 10/12
- Preoperative Radiotherapy (Paulson Regime) essential, postoperative DXT very variable
- Impressive results from Darteville with resection of all through supra-clavicular incision giving 31% 5-year survivals

With Any Locally Advanced NSCCL You must not have N2 Disease
Superior Sulcus Tumours

- Constellation of Benign and Malignant tumours in Sulcus
- Sulcus is groove formed by subclavian artery passing under clavicle over 1st rib
- Arise from upper lobes & invade parietal pleura, endothroughic fascia, subclavian artery, brachial plexus, vertebra, ribs
- Invading the superior thoracic inlet and causing steady, severe, unrelenting shoulder pain
- Can be anterior to anterior Scalenus muscle
  - Invades Platysma, SCM, External& Anterior Jugular veins, Subclavian & internal Jugular veins, 1st intercostal nerve and rib not Phrenic
- Can be between anterior and middle Scalenus muscle
  - Invades Phrenic sitting on Scalenus, subclavian artery and Brachial Plexus (pain radiation to shoulder and upper limb)
- Can be posterior to middle Scalenus muscle
  - Sitting in costovertebral groove, invades nerve roots of T1, posterior aspect of Subclavian artery, Stellate Ganglion with Horner’s
- Pancoast-Tobias syndrome is tumour associated with nerve involvement C8-T1 atrophy of hand & possible Horner’s
- By virtue of rib / chest wall involvement they are classified as T3 tumours. If involve Vertebra then T4. Can be both
- In Pancoast syndrome due to nerve involvement these are T4 tumours

Irradiation followed by radical resection 5 yr survival 44%
Superior Sulcus Tumours

**Diagnosis**
- CXRAY – Mass with rib destruction
- Tissue diagnosis (<10% these are not lung tumours but carcinomas of thyroid, pharynx, mesothelioma, aspergilloma)
- CT – Good for N2 detection and distant metastasis
- Must ensure no N2 nodes therefore Mediasinoscopy in everybody
- MRI – Superior for T stage as well as nerve and vertebral formina extension
- Nerve studies to delineate tumour extension into Brachial Plexus

**Absolute contraindications to surgical resection**
- N2 disease hence some advocate routine Mediasinoscopy or PET
- Distant metastasis
- Brachial Plexus involvement above T1
- Invasion of the spinal canal through the intervertebral foramen

**Relative contraindications to surgical resection**
- Vertebral involvement
- Encasement of great vessels

**Prognostic Factors**
- Presence of Horner’s syndrome is a negative prognostic marker 5 yr survival of 8% in those with versus 35% with-out
- T4 worse than T3
- N2 worse than no N2 (If N2 nodes are positive 5 year survival post resection is 0)
- Complete resection is best

**Neoadjuvant Therapy**
- Phase II Intergroup study-Induction with combined Chemoradiotherapy has survival benefit prior to surgery
- Previously Only Paulson Regime Radiotherapy was and is still being given

Irradiation followed by radical resection 5 yr survival 18 - 56%
Limited Resection for Lung Cancer (Wedge & Segment)

- Only Stage I NSCLC patients are candidates for curative limited resection (How do you define them preoperatively)
- Namely it must be a peripherally located tumour with no regional lymph nodes being free from involvement
- PET scan may have a role to play for excluding the involvement of N1 nodes

Lung Cancer Study Group (1995 Gingsberg)
- RCT in stage Ia lung cancer of Lobectomy versus limited resection
- 247 patients
- 122 randomised to limited resection (82 segmental, 40 wedge)
- 125 Lobectomies
- "Survival" was superior in Lobe group over limited resection but not statistically significant
- Local Regional recurrence was 3 fold in wedge group and 2 fold in segment group
- Gingsberg concluded that Lobectomy is treatment of choice

Warren & Faber
- 66 patients with NSCLC undergoing limited resection Vs 105 Lobectomies
- Local regional recurrence 22.7% after segmental resection Vs 4.9% for Lobectomy
- Survival superior in tumours > 3cm in the lobectomy group(< 3cm Lobe=Segmental resection)

Martini et al
- Stage I Lobectomy vs segmental resection
- 5 year survival for segmental resection was 59% Vs 77% for Lobectomy
- Also the limited resection group had more local recurrence

Limited resection plays a part in both second Primaries as well as Synchronous tumours

Segmentectomy Vs Wedge resection
- Segmentectomy take out originating bronchus, artery, and lymphatics allowing resection of draining lymphatics
- Wedge resection crosses lymphatics, does not remove originating Bronchus with Parenchymal margins close to tumour

Limited resection can afford lower early mortality and almost equal long term survival to marginal patients but with increased incidence of local recurrence
Lung Volume Reduction (LVRS)

- Resecting areas with little function and reducing hyper-expansion other areas of healthy lung function
- Improvement in lung elastic recoil results in more rapid and complete expiratory flow
- 5 RCTS
- No uniformity of treatment in the 5 RCTs
- Strict entry criteria in which between 17 – 32 % of patients screened are enrolled
- NETT highest inclusion % since their objective was to identify criteria for entry
- Control group – medical Rx is not always detailed (mixture of Physiotherapy & Intensive Medical Rx)
- Most studies did not allow cross-over between the two groups
- All had strict defined quantitative definition of lung hyperinflation
- Most exclude Asthmatics, High PaCO2, high PA pressures, over 75s, not stopped smoking

Entry Criteria
- NYHA 3 - 4
- FEV1, 45%, TLC > 100%, RV > 120%
- Emphysema – Heterogeneity on CT, No Isolated Bullae
- Ventilation – Self ventilation
- Exclude high anaesthetic risk, Angina, > 10mg steroid use
### Lung Volume Reduction (LVRS)

#### Table 1. Randomised controlled trials entry criteria.

<table>
<thead>
<tr>
<th>Name</th>
<th>Criner</th>
<th>Pompeo</th>
<th>Geddes</th>
<th>Goldstein</th>
<th>Fishman (NETT)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screened</td>
<td>200</td>
<td>237</td>
<td>174</td>
<td>328</td>
<td>3777</td>
</tr>
<tr>
<td>n</td>
<td>37</td>
<td>60</td>
<td>48</td>
<td>55</td>
<td>1218</td>
</tr>
<tr>
<td>n/screened (%)</td>
<td>19</td>
<td>25</td>
<td>28</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>NYHA 3-4</td>
<td>Dyspnoea score&gt;3</td>
<td>NA</td>
<td>FEV&lt;40%</td>
<td>FEV&lt;45%</td>
</tr>
<tr>
<td>Hyperinflation</td>
<td>FEV&lt;30%</td>
<td>FEV1&lt;40%</td>
<td>FEV1&gt;500mls</td>
<td>FEV1/FVC&lt;0.7</td>
<td>RV&gt;150%</td>
</tr>
<tr>
<td></td>
<td>FRC or TLC&gt;120%</td>
<td>RV&gt;180%</td>
<td>TLC&gt;120%</td>
<td>TLC&gt;120%</td>
<td>TLC&gt;100%</td>
</tr>
<tr>
<td>Asthma/Bronchitis</td>
<td>NA</td>
<td>No sputum, bronchiectasis or asthma</td>
<td>No asthma</td>
<td>No asthma</td>
<td>NA</td>
</tr>
<tr>
<td>Emphysema</td>
<td>Hyperinflation on CXR and diffuse bullous emphysema on HRCT</td>
<td>Diffuse bullous and non-bullous severe heterogeneous emphysema on HRCT</td>
<td>No isolated bullae, no restriction on pattern or distribution of emphysema on CT or VQ scan</td>
<td>Heterogeneity of emphysema</td>
<td>HRCT evidence of bilateral emphysema</td>
</tr>
<tr>
<td>Perfusion scan</td>
<td>Areas of decreased perfusion</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Oxygenation</td>
<td>PaO2/FiO2&lt;150</td>
<td>NA</td>
<td>O2 use &lt;18hrs/day</td>
<td>NA</td>
<td>Requiring &lt;6L O2 to maintain O2 sats &gt;90% with exercise</td>
</tr>
<tr>
<td>Ventilation</td>
<td>Self-ventilating</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PaCO2 (kPa)</td>
<td>NA</td>
<td>7.3</td>
<td>6</td>
<td>6.6</td>
<td>8</td>
</tr>
<tr>
<td>DLCO</td>
<td>NA</td>
<td>&gt;20% pred</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Mean &lt;35mmHg</td>
<td>Peak systolic &lt;50mmHg</td>
<td>Mean &lt;35mmHg</td>
<td>Mean &lt;35mmHg</td>
<td>Mean &lt;35mmHg</td>
</tr>
<tr>
<td>pressure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (BMI)</td>
<td>&lt;70% predicted</td>
<td>18-29</td>
<td>NA</td>
<td>NA</td>
<td>&lt;31.1</td>
</tr>
<tr>
<td>Age (years)</td>
<td>NA</td>
<td>&lt;75</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<tr>
<td>Psychology</td>
<td>Assessed</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Smoking (x months cessation)</td>
<td>6</td>
<td>4</td>
<td>6</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Anaesthetic risk</td>
<td>NA</td>
<td>ASA &lt;/3</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Other exclusions</td>
<td>Cardiovascular disease</td>
<td>Unstable angina</td>
<td>Previous thoracic surgery</td>
<td>Previous thoracic surgery or pleural disease</td>
<td>Approval of anaesthetist sought</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ventricular arrhythmia</td>
<td>&gt;10mg steroids per day</td>
<td></td>
<td>Previous transplant, lobectomy, LVRS or median sternotomy &gt;20mg steroids per day</td>
</tr>
</tbody>
</table>

NA = criteria not applied; NYHA = New York Heart Association dyspnoea score; FEV1 = forced expiratory volume in 1 second; FRC = functional residual capacity; TLC = total lung capacity; RV = residual volume; CXR = chest x-ray; CT = computed tomography; HRCT = high resolution computed tomography; VQ = ventilation perfusion.
Table 2. Surgical and medical treatments in the randomised controlled trials.

<table>
<thead>
<tr>
<th>Name</th>
<th>Pre-randomisation treatment</th>
<th>Surgical treatment</th>
<th>Control medical treatment</th>
<th>Cross-over allowed?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criner</td>
<td>8 weeks pulmonary rehabilitation for all patients</td>
<td>Bilateral LVRS via median sternotomy. Stapled resection of 20-40% of the volume of each lung</td>
<td>3 months pulm rehab</td>
<td>Yes, 13 of 18 patients in the medical arm crossed over after pulmonary rehab</td>
</tr>
<tr>
<td>Pompeo</td>
<td>No patients in the surgical arm had pre-operative pulmonary rehab</td>
<td>Unilateral or bilateral VATS Stapled resection of 20-30% lung vol</td>
<td>6 weeks pulm rehab</td>
<td>No</td>
</tr>
<tr>
<td>Geddes</td>
<td>All patients had intensive pulmonary rehabilitation before randomisation</td>
<td>Bilateral lung resection via VATS or median sternotomy Stapled resections were buttressed with bovine pericardium or fibrin glue VATS mainly, some median sternotomies, 20-30% reduction in lung volume, bilateral where possible</td>
<td>Continued medical treatment for 12 months</td>
<td>No, but 6 of 21 medical patients had surgery after the trial analysis</td>
</tr>
<tr>
<td>Goldstein</td>
<td>All patients had 6 weeks of rehabilitation</td>
<td>Continuation of medical treatment or median sternotomy (20-30% reduction in lung volume)</td>
<td>Continued medical treatment for 12 months</td>
<td>No</td>
</tr>
<tr>
<td>Fishman (NETT)</td>
<td>All patients had 6 to 10 weeks of pulmonary rehabilitation</td>
<td>VATS (30%) or median sternotomy (70%) bilateral stapled wedge excision of 20-35% of each lung</td>
<td>Continued medical treatment for 24 months</td>
<td>No, but 33 of 610 patients in the medical group underwent LVRS outside the study</td>
</tr>
</tbody>
</table>
LVRS – Results of RCTs

**Criner et al 1999**
- Most of analysis compared pre- and post-operative changes in LVRS group not between LVRS and Controls
- Increased FEV1 and FVC, decreased TLCO and RV
- Decreased PaCO2
- Increased maximal exercise performance and 6 minute walk

**Pompeo et al 2000**
- Significant benefit in LVRS group for dyspnea index, FEV1, FVC
- Improved PaO2 in LVRS group
- Improved 6 minute walk

**Geddes et al 2000**
- Primary outcomes were mortality, FEV1 change, shuttle walking distance, QOL at 3, 6, 12 months
- No difference in mortality between LVRS and medical groups
- LVRS improved shuttle walking distance and QOL. FVC increased
- FEV1 improved at 3/12 but declined again suggesting short-term improvement but long-term disease progression
- Patients that did not improve had more homogenous COPD suggesting LVRS more effective in heterogeneous disease

**Goldstein et al 2003**
- Concentrated on QOL
- LVRS improved all aspects of QOL over intensive medical therapy
- No difference in mortality
LVRS – Results of RCTs

Fishman et al 2003 (NETT)

- Largest of all RCTs, 17 centres, longest follow-up
- Main outcome was all cause mortality and maximal exercise tolerance at 2 years
- Classification of distribution of emphysema;
  - Heterogeneous upper lobe predominance
  - Homogeneous (or diffuse) emphysema
  - Lower lobe emphysema (including alpha antitrypsin deficiency)
  - Emphysema of the apical segment of the lower lobe
- 90 day mortality in LVRS group was significantly higher, VATs=MS, if you exclude 140 very high risk patients mortality==
- 2 year survival was the same for both groups
- Exercise capacity, 6 minute walk distance, degree of dyspnoea, FEV1, and QOL all better in LVRS group
- Patients with Heterogeneous upper lobe predominance had best outcome even lower two year mortality in LVRS group

Based on these trials and others some suggestions have been made regarding patient selection

- Pulmonary Function Tests
  - Hyperinflation (TLC > 130%) Plethysmography is essential. Also FEV1 < 35%
- Pattern of Emphysema
  - Heterogeneous more than Homogeneous (Diffuse)
- Age
  - Glasspole et al found that Age > 70 carried a 9 fold increase in mortality in patients undergoing LVRS
- Hypercapnia
  - Conflicting evidence some advocating that a PaCO2 > 6 is a risk factor for mortality
- TLCO
  - A low TLCO < 20% carries a very high operative risk
- Nutritional Status
  - Outside 70 –130% of predicted BMI are at particularly high risk for LVRS
LVRS – Surgical Aspects

Laser Vs Stapling
- McKenna 1996 Laser Vs Stapling
- Higher rate of delayed Pneumothorax, higher post-operative 02 dependency and lower FEV1 improvement in Laser group
- Laser now abandoned

Bilateral Vs Unilateral
- No RCT
- Theoretical advantage to bilateral surgery for maintenance of chest symmetry are unsupported by any evidence
- Needs RCT

Bilateral Vs Staged Unilateral
- No RCT
- No evidence to support one over the other

VATS Vs Median Sternotomy
- No RCT
- One comparative study found
  - comparable for the two techniques in respect to Pulmonary function and symptoms
  - VATS group reduction in respiratory failure

Buttressed Vs no Butressing
- Numerous RCT
- Significant improvement using buttressing found in all RCTs
  - Earlier drain removal, reduction hospital LOS
  - Reduced median air-leak time
## LVRS – Recommendations

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Evidence level</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVRS benefits select patients with emphysema (see selection recommendations).</td>
<td>Ib/A</td>
</tr>
<tr>
<td>Patients with heterogeneous (particularly upper lobe) emphysema are more likely to benefit.</td>
<td>Ib/A</td>
</tr>
<tr>
<td>Patients with FEV1 less than 20% predicted and either a CO transfer factor less than 20% predicted or a homogeneous distribution of emphysema have a high mortality.</td>
<td>Ib/A</td>
</tr>
<tr>
<td>Stapled resection is superior to laser.</td>
<td>Ib/A</td>
</tr>
<tr>
<td>Buttressing of staple lines with bovine pericardium or collagen reduces air leak.</td>
<td>Ib/A</td>
</tr>
</tbody>
</table>
Giant Bullous Disease

- Bullae that encompass more than 1/3\textsuperscript{rd} of Hemithorax
- Bullae distinction from bleb by size, Bullae > 1cm Bleb < 1cm
- Bullae located apically except in alpha anti-trypsin deficiency in whom basilar bullae seen
- Look for incidental lung cancers in this group

Pathophysiology

- Air filled sacs that do not participate in ventilation causing compression of surrounding lung
- Hyperinflation also causes downward pressure on diaphragm, flattening it and impairing its function
- Air enters Bulla on inspiration but because airways collapse, ball valve Phenomena whereby air trapping occurs
- Due to compression of blood vessels right to left shunt occurs and hypoxia ensues

Complications

- Causes SOB by their presence and size – Size / symptoms / fitness of patient dictates management
- If rupture Secondary Pneumothorax treat with ICD (16 % dies !)
  - After ICD placement for Pneumothorax If prolonged (> 5 days) air leak intervene with
  - Pleurodesis vs. Surgery depending on fitness
- Air fluid levels represent inflammation or true infection
  - Infection is treated as lung abscess with antibiotics
  - Failure of resolution with antibiotics indication for surgery dangerous however in presence of infection
- Haemoptysis
  - Source usually Bronchial: as for any Haemoptysis exclude Malignancy & deal with Bronchial (Embolisation)
Giant Bullous Disease

Preoperative
- Fitness, Age, Cachexia, Cardiac co-morbidities
- CT – Size and other associated pathology
- Lung Function tests (Evidence of hyperinflation) including DLCO < 25% of predicted is contra-indication
- 6 minute shuttle test if less than 600 feet operative risk is high
- ABG – Severe CO2 retention relative contra-indication

Surgery (3 options)
- Intracavity drainage (Monaldi procedure)
  - Initially described for relief of tension in Tuberculous cavities now applied to Emphysema
  - Rib resection and packing of space for < 2 weeks to allow adherence of Bulla to Pleura then Bulla is incised and suction drain inserted
  - Now modified to single procedure with purse string to bulla and Pleura with Foley Catheter on suction to drain
- VATS
  - Single lung ventilation* 3 Port VATS with Pericardial re-inforced staple line and Pleurectomy – Less pain
  - No evidence to support the use of staged procedures for bilateral disease – I.e. do them at the same sitting
- Thoracotomy
  - More pain, Various rolling methods after opening the Bulla and using the Bulla wall itself to buttress the closure
- Median Sternotomy
  - Less pain than thoracotomy for bilateral disease. No difference between Bilateral VATS and Median Sternotomy

Post-operative
- Pain relief and Pulmonary toilet
- Judicious O2 in view of some patients need for hypoxic drive to ventilation
- Chest tubes are left to underwater seal s even if apical space exists
  - Avoidance of suction decreases the duration of the leak
  - Hemilch valves can be used to discharge patients with air leak

* Bullous areas tend to remain Inflated compared to well-perfused lung which collapse (resorptive atelectasis)
Malignant Pleural Mesothelioma (MPM)

Cell Origin – Mesothelial - 3 Subtypes
- Epithelial, Sarcomatoid, Mixed (Biphasic), presenting with SOB & Chest pain

Incidence
- 1600 lives in UK / year
- Set to rise to 3000 lives In next 20 years
- Predicted that 1% of men born in the 1940s will die from Mesothelioma

Aetiology – Asbestos Exposure (90% of Patients with MPM have been exposed to asbestos)
- Damage occurs by oxygen free radicals induced by ferric Iron impurities in fibres
- 40 year lag between exposure and cancer

Survival
- Median Survival 5 – 13 months

Prognostic Factors indicating poor survival
- Cell type other than epithelial
- > 5% loss weight loss
- FBC: Low Hb, abnormal WCC, Increased platelet count
- Poor performance status
- Males
- High tumour volume indicates also nodal status (i.e. The higher the T the more likely the higher the N)
Role of Surgery

Diagnosis

- CXRAY – Pleural plaques, unilateral pleural effusions
- Pleural fluid cytology – Sensitivity 35 –45%
- Blind biopsy – (Abram’s needle) - Sensitivity 35 –45%
- Δ Δ – Metastatic Adenocarcinoma (Rediagnosis after surgery changes up to 20% of MPM to Metastatic Adenocarcinoma)
- Immunohistochemistry routinely used
- VATs Vs Open Biopsy

Fitness for Surgery

- BTS guidelines followed for Pneumonectomy, ppo FEV1 = 40% & ppo TLCO of 40%
- BTS guidelines for Cardiac fitness also followed
- WHO performance status of 0 –1 for EPP
- Mediastinoscopy (Nodal size correlates poorly with metastasis any visible nodes should be sampled)
- PET scanning is indicated for potentially resectable disease
Figure 1. The role of surgery in mesothelioma.
Unresectable Disease

Not fit for surgery
- Mobile Lung
  - Pleurodesis with talc achieves life-long effusion control in 90%
- Trapped lung
  - Pleuro-peritoneal shunt (Denver shunt)

Fit for Surgery
- Debulking
- Parietal Pleurectomy
- Parietal and Visceral Decortication
- Waller paper: no data on effect of debulking on survival given
- Other series indicate improved survival when surgery is combined with adjuvant therapy either chemotherapy or DXT

Port Site Radiotherapy
- All patients undergoing invasive procedures with MPM potential for port site metastasis
- Type I evidence (RCT) shows benefit of DXT post procedure on port site metastasis
Resectable Disease

Not fit for Pneumonectomy

- Pleurectomy and Decortication (P/D)
  - 8 published series P/D alone median survival 5 – 17 months
  - Other series report P/D as part of multi-modality Regime
  - Overall appears to be net benefit if chemotherapy (Intraoperative or post-operative) is added to P/D
  - DXT – Post-operative with DXT no survival benefit – If Intra and post-operative with P / D gave survival benefit
  - P / D as part of trimodality therapy results Disappointing

Fit for Surgery

- Extra Pleural Pneumonectomy (EPP)
  - Can now be offered at acceptable peri-operative morbidity and mortality
  - No randomised trials reported presently
- Mesothelioma And Radical Surgery (MARS)
  - PRCT
  - Includes neoadjuvant Chemotherapy and post-operative hemithorax irradiation
  - Will report results 2010
Glasgow Paper*

- 10 year review of 302 patients
- 111 Surgery Vs 191 no surgery
- 47 P / D  64 Radical EPP
- Of 64 EPP, 51 also had intra-operative as well as postoperative chemotherapy
- In EPP group operative mortality 9%, morbidity in 21%
- In EPP group overall median survival disappointing 13 months
- But for 51 EPP with Chemotherapy, median survival 35 months, 48% 3 years, 18% 5 years
- Survival dependent on T stage (T1 better than T3)
- Nodal status not statistically relevant to survival
- No radiotherapy used!

EPP (Evidence For!) II

Sugarbaker (JTCVS 1999;117;54-65)

- 183 Patients EPP followed by adjuvant chemoradiotherapy
- In-hospital Mortality = 3.8%
- Morbidity 50 – 60%
  - 44% AF
  - 8% Prolonged intubation
  - Vocal cord paralysis 7%
  - DVT 6%
- 38% Survival at 2 years, 15 % at 5 years with median survival of 19 months
- In subgroup of Epithelial type, negative resection margins, node negative
  - 68% Survival at 2 years, 46 % 5 years, median survival 51 months
Rusch et al summarises role of surgery in MPM

- 306 patients
- Either P / D or EPP along with range of Chemotherapy and Radiotherapy regimes
- Four factors Significant multivariable for good outcome
  - Early tumour stage (IMIG I/II)
  - Epithelial type
  - Radiotherapy
  - Chemotherapy
- Operative Procedure NOT Significant in multivariable analysis
- Therefore evidence for which type of Radical Surgery “Jury is still out”
- Surgery alone is not indicated but multimodality therapy has a role
BTS recommends Active Symptom control (ASC)
- Close Patient monitoring, Nurse Support
- Regular assessment & Evaluation of disease CXRAY, CT

MESO – 1 trial 3-arm study to assess feasibility of using ASC alone

MESO-1 a Pilot study MS01 is now the current study, 60 Centres, needs 840 Pts, 2-300 recruited/year

Aims: Survival, Quality of Life, Symptom relief, Toxicity, Tumour response, recurrence / progression

Patients are randomised to one of 3 arms
- ASC alone
- ASC with Mitomycin, Vinblastin, Cisplatin
- ASC with Navelbine

Entry Criteria
- Microscopically and immunohistochemically confirmed malignant pleural mesothelioma, any cell type
- Any symptomatic pleural effusion under control by drainage, pleurodesis, or pleurectomy
- CT scan within 1 month before randomisation (preferably after pleurodesis)
- if mesothelioma resected, two CT scans 6 weeks apart showing assessable stable or progressive disease;
- no previous chemotherapy for mesothelioma
- no other disease likely to interfere with protocol treatments or comparisons
- WHO performance status 0–2
- WCC >3 x 10^9/l, neutrophil count >1.5 x 10^9/l, platelet count >100 x 10^9/l, and no clinical evidence of infection
- medically fit to receive chemotherapy
- informed consent form signed following full discussion of a patient information sheet describing the study design and stating that the doctor would discuss with the patient which comparison was most suitable
- quality of life forms completed before patient told the treatment allocated

By 2003 recruited 179 patients, Latest Publication was Thorax 2004
Massive Haemoptysis Case report
Presenting Complaint

- J.M. 43 Year-Old man
- 3/52 Hx Productive Cough
- Significant Haemoptysis (Bright Red Blood)
- Tachycardia, Normotensive
- Hypoxia, Sats 80% on Air

Definitions range from 100 – 1000ml /24 hours (Consensus > 600 mls/24 hours)
Past Medical History

- 1989 Open Mitral Valvotomy (Rheumatic MS)
- 1997 MI, AF, Saddle embolus C.I-Embolectomy
- Warfarin, Digoxin, ACE I, Diuretics
- Smoker
- Heavy ETOH Consumption
Baseline Investigations

- Hb 13
- ECG AF Rate 140
- INR 2.1, APTT 40/30
- CXRAY
Initial Management

- Blood Tx
- FFP
- Vitamin K

Flexible Bronchoscopy - Right Normal

- Left Full of blood, Not Clearly Seen

Rigid Bronchoscopy
Further Investigations & Rx

- Echocardiograph
  - Rheumatic Mitral Valve
  - Moderate Mitral Stenosis, MVA 1.8 cm²
  - No Atrial thrombus

- CT Thorax
  - No Parenchymal Lung lesions
  - No evidence of Bronchiectasis

- Gentle Heparin
History Continued

- 5/7 Further Massive Haemoptysis
- Sats 50 % on 98% Oxygen
- Heparin Reversal, Blood Tx
- Emergency Rigid Bronchoscopy + Toilet
- Intubated, ITU
Angiography

- **Pulmonary Angiogram**
  - No evidence of PE
  - No AVM

- **Bronchial Angiogram**
  - Left Bronchial Blush
  - Particle Embolisation
Massive Haemoptysis Causes

- Inflammatory lung disease (85%)
  - TB accounts for majority of cases (Rupture of Ramussen aneurysm of bronchial artery crossing cavity)
  - Aspergillosis
  - Lung abscess
  - Cystic Fibrosis

- Bronchiecatis

- Carcinoma – Rare

- Trauma

- PE

- AV fistula – Osler – Webb – Rendu syndrome

- Cardiac Disease – Mitral Stenosis (Hypertensive rupture of small pulmonary vessels, venous connections, bronchial vein)

- Iatrogenic – PA catheters

Definitions range from 100 – 1000ml /24 hours (Consensus > 600 mls/24 hours)
Massive Haemoptysis – Blood Supply

- Bronchial Circulation 95%
  - Most commonly inflammatory lung disease particularly TB

- Pulmonary Circulation 4%
  - Most common is PA catheters

- Systemic Collaterals 1%
  - Fistulisation of bronchial tree with systemic artery (Bronchial artery is systemic!)

Incidence rare 1-4% of patients with haemoptysis. Mortality up to 80%
Massive Haemoptysis

**Diagnosis**
- Complete history including anti-coagulations history etc… exclude haematemesis and epsitaxis
- Radiography – Plain CXRAY, CT, Radionuclide perfusion scan, Selective bronchial angiogram
- Bronchoscopy – During acute bleed, secure airway, localise bleeding, control bleed if possible

**Management**
- Prevent asphyxiation (AIRWAY MAINTENANCE)
- Stop ongoing bleeding
- Provide definitive treatment plan of underlying pathology

**Immediate**
- Position patient head down and bleeding side down
- Oxygen sedation
- IV access blood crossmatch

**Rigid Bronchoscopy**
- Control airway, toilet, locate bleed, lavage with ice-cold saline, or tamponade with bronchial blocker, leave intubated
- Double lumen tube can be used if put in quickly

*Incidence rare 1-4% of patients with haemoptysis. Mortality up to 80%*
Massive Haemoptysis Management

- Medical
- Surgical
- Interventional Radiology

Definitions range from 100 – 1000ml /24 hours (Consensus > 600 mls/24 hours)
Medical

- Airway Management including selective Intubation
- Bronchoscopy + endobronchial therapy
- Correction of coagulation abnormalities
- IV Vasopressin
- Treatment of underlying Pathology
- Recurrence Rate High
Surgical

- Emergency Surgical Resection (Avoid – stabilise first)
- Laser Photocoagulation
- More Definitive Management
- Sometimes difficult to localise specific point
- High Mortality Rate
Bronchial Artery Embolisation

- Good Immediate and Long term results
- Allows Surgery TBA electively
- Not without its complications
Summary

- Most Cases arise from Bronchial Arteries
- High Risk of recurrence
- Often fatal haemoptysis
- Definitive management of bronchial arteries before hospital discharge
Other Cause of Massive Haemoptysis

This example is referred to as complex Aspergilloma since lung is abnormal. Massive Haemoptysis again occurs from Bronchial Artery Bleeding.

Aspergilloma: Abnormal Right Lung with Bullae hence term “Secondary”
Aspergillosis

3 Classifications

- Aspergilloma, Invasive infection, non-invasive bronchial allergic

- Surgery is reserved only for Aspergiloma

Aspergilloma

- Sub-divided into Simple when surrounding lung is normal & Complex in setting of abnormal lung

- Presentation is Haemoptysis often severe

- Diagnosis based on CT

- Management medial management for Aspergilloma is usually not effective

- Surgery is indicated particularly for recurrent haemoptysis

- In setting of Complex Aspergilloma Cavernostomy may be better option to surgical resection
Old classification Superior and Inferior (Angle of Louis) with Inferior being divided into Ant. Middle, Posterior
now just Anterior, Middle=Visceral, Posterior = Para vertebral
Mediastinum

- Space between the pleural cavities occupying the centre of the thoracic cavity
- Superior border is the thoracic inlet – T1 vertebra posteriorly, 1st rib laterally, manubrium anteriorly
- Inferior border is the diaphragm
- Lateral border is the mediastinal pleura
- Divisions of anterior, middle (Visceral), posterior (para-vertebral)

Presentation
- Symptoms chest pain, fever, cough, dyspnea. Severe pain, Horner’s, Hoarseness (RLN), SVC obstruction point to malignancy more
- Symptoms from ectopic hormones, antibody production, more for anterior and posterior site lesions than middle
- Examine testicles

Diagnosis (Goals)
- Differentiation between 1 masses and 2 masses (Particularly high index of suspicion for Germ/ cell & lymphoma)
- Detection of compression of either airways or vessels that determine resectability
- Operative risk assessment

Investigations
- CXRAY PA and Lateral
- CT with contrast (CT guided core biopsy)
- MRI useful for Vascular or neurogenic or cysts structures. Cysts have low signal intensity on T1 weight higher on T2
- Mediasinoscopy (access mainly to middle mediastinum not anterior!)
Indications for surgery

- Obtaining tissue diagnosis for anterior mediastinal mass is not always an indication for surgery since you have CT guided core biopsy.

Anterior Germ cell tumours (Seminomas)

- Seminomas are very Chemosensitive, usually no tumour marker elevation, no role for surgery.
- 5 year survival with Cisplatin-based chemotherapy is 70 – 80%.

Anterior Germ cell tumours (Non-Seminomas)

- Again little to no role for surgery but more of a role than Seminomas.
- Frequently large and infiltrative with elevated tumour markers.
- Treated primarily with Cisplatin-based chemotherapy.
- 5 year survival with Chemotherapy 40 – 50%.

Role for surgery for

- Patients who have a response to Chemotherapy with disappearance of tumour markers but have a residual mass.
- Patients who have a residual mass on CT but still have elevated tumour markers after chemotherapy (controversial).
Mediastinum

Anaesthetic
- Careful induction of anaesthesia with surgeon in anaesthetic room
- Consider awake intubation for large compressive tumours
- Rapid induction of anaesthesia with loss of muscle tone may make securing airway difficult

Surgical Considerations
- Median Sternotomy vs. VATS
- If VATs right side is larger therefore more preferable
- Open both pleura and identify Phrenic nerves, assess relation to veins (I&SVC), great arteries, pericardium, lung
- Substernal Goitres usually through collar incision
  - Beware of larger blood vessels coming off aortic arch
- Innominate vein involvement
  - May be divided with impunity
- SVC
  - Tangential resection
  - Total resection and PTFE graft re-construction with or without CPB
  - Patients who have a response to Chemotherapy with disappearance of tumour markers but have a residual mass
  - Patients who have a residual mass on CT but still have elevated tumour markers after chemotherapy (controversial)
## Mediastinum

### Tumors and Cysts by Location

<table>
<thead>
<tr>
<th>Anterior (6)</th>
<th>Middle (7)</th>
<th>Posterior (8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thymoma/Thymic cyst</td>
<td><strong>Bronchogenic cyst</strong></td>
<td>Neurogenic origin</td>
</tr>
<tr>
<td>Thyroid (Aberrant)</td>
<td><strong>Pericardial cyst</strong></td>
<td>Neurenteric cyst</td>
</tr>
<tr>
<td>Teratoma (Germ cell tumors)</td>
<td>Tracheal Tumours</td>
<td>Lymphoma</td>
</tr>
<tr>
<td>T cell Lymphoma</td>
<td>Lymphoma</td>
<td>Thoracic duct cyst</td>
</tr>
<tr>
<td>Lymphadenopathy</td>
<td>Aneurysms</td>
<td>Oesophageal Tumours</td>
</tr>
<tr>
<td>Parathyroid Tumour/cyst</td>
<td>Neurogenic (Phrenics)</td>
<td>TB (Infection)/abscess</td>
</tr>
<tr>
<td></td>
<td>Parathyroid Tumour/cyst</td>
<td>Chondroma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hiatus Hernia</td>
</tr>
</tbody>
</table>
Thymoma

- Arise from Epithelial aspect of Thymus Gland. 3rd and 5th Decades. Rare in children
- 2nd Commonest mediastinal tumour in adults
- Anterior and sometimes extending into Visceral Mediastinal masses
- Variable clinical presentation
  - 30% Asymptomatic
  - 30% Local symptoms, cough, dyspnoea, chest pain
  - 30% Systemic Symptoms
  - Up to 50% have Myasthenia Gravis (MG) (NOTE: only 15% of patients with MG have a Thymoma)

Investigations

- CT with contrast
- B HCG, a fetoprotein to aid in differential diagnosis with germ cell tumours
- Tensilon test (symptomatic improvement with anti-cholinesterase), EMG
- Acetylcholine receptor antibody assay

Second Commonest mediastinal tumour in adults Commonest tumour of anterior mediastinum
Thymoma

**Diagnosis**

- Biopsy - Needle (core) Vs Open (Big differential diagnosis is Lymphoma)
  - Needle – sometimes insufficient material to yield diagnosis, may seed tumour “drop metastases”
  - Open via anterior mediastinotomy (Chamberlain procedure), seeding still a concern
- Diagnostic accuracy is 59% for needle Vs 81% for open biopsy
- Preoperative tissue diagnosis is recommended for cases with invading mediastinal structures or Lymphoma suspected

**Role of Radiotherapy**

- Very radiosensitive
- Recurrence for fully resected stages II and III = 5% with DXT Vs 28% with no DXT

**Role of Chemotherapy**

- Very Chemosensitive
- Regimes and role still to be determined
- Complete remission seen in 10 – 68%

*MULTIMODALITY therapy with preoperative Chemo + Post Operative Radiotherapy may have a role in stages III and IV*
Masaoka Staging

Stage I (50% present at this stage)

Completely encapsulated

Stage II (27% present at this stage)

IIA Microscopic invasion into capsule

IIB Macroscopic invasion into capsule, surrounding fatty tissue or mediastinal pleura

Stage III Macroscopic invasion into neighboring organs lung, great vessels, pericardium

Stage IV (4% present at this stage)

IVA Pleural or pericardial dissemination

IVB Distant metastasis
### Histology

<table>
<thead>
<tr>
<th>WHO type</th>
<th>Marino and Muller-Hermelink</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Medullary thymoma</td>
</tr>
<tr>
<td>AB</td>
<td>Mixed thymoma</td>
</tr>
<tr>
<td>B1</td>
<td>Predominately cortical thymoma</td>
</tr>
<tr>
<td>B2</td>
<td>Cortical thymoma</td>
</tr>
<tr>
<td>B3</td>
<td>Well differentiated thymic ca.</td>
</tr>
<tr>
<td>C</td>
<td>Thymic carcinoma</td>
</tr>
</tbody>
</table>

Squamous cell ca.
Epidermoid non keratinizing ca.
Lymphoepithelioma-like ca.
Carcinosarcoma
Clear cell carcinoma
Basaloid carcinoma

**WHO A, B, C, progressively show an increased local aggressiveness** Medullary better than Cortical

Staging and Histology Considered together to give Prognosis
Thymoma

Management

- Stage I: Total Thymectomy
- Stage II: Total Thymectomy + Post-operative radiotherapy
- Stage III: Total Thymectomy + pericardium, lung, innominate vein + Post-operative radiotherapy (chemotherapy optional)
- Stage IVa: Chemotherapy followed by debulking (5yr survival up to 75% with Vs only 40% without) (controversial)
- Stage IVb: Chemotherapy, Octreotide

If associated with MG then “Maximal” Thymectomy must be performed

Recurrence

- Stage I - < 5%
- Stage II - 5-20%
- Stage III - 15-30%
- Stage IV - 25-55%

- Mean time to recurrence is 5year, intra-thoracic in 70 –80%
- Treatment is re-resection, DXT, Chemotherapy, Octreotide (Somatostatin analogues) new!

Surgery: VATs or MS, VATs still experimental, Avoid injury to Phrenic nerves but may be sacrificed
Thymoma

Prognosis

Predictors of Survival

- Stage (as above)
- Histology (Epithelial worst, lymphocytic and spindle best)
- Completeness of surgical resection
- MG not prognostic factor
- Cause of death unrelated to tumour in up to 65%
Thymoma

Anterior Mediastinal Mass (Not Typical are areas of necosis)
Final Diagnosis was Stage I Thymoma (WHO AB Cell Type)
Thymoma

Final Diagnosis was Stage III Thymoma invading local structures

Controversial management
Preoperative DXT and Chemotherapy followed by surgery for debulking or Radical surgery followed by Chemotherapy and DXT
Discuss in MDT
Thymoma

Final Diagnosis was Stage IVa Thymoma intrapleural spread

Controversial management Preoperative Chemotherapy followed by surgery for debulking followed by DXT Discuss in MDT
Myasthenia

- Autoimmune neuromuscular disorder caused by antibodies to post-synaptic nicotinic Ach receptors
- Decrease in AchRs results in end-plate potentials which may fail to trigger action potentials
- Becomes manifest as weakness

**Biphasic mode of distribution**
- First peak 2\textsuperscript{nd} – 3\textsuperscript{rd} decade of life mainly women
- Second Peak 6\textsuperscript{th} – 7\textsuperscript{th} decades mainly men

**Natural History (1/3rds)**
- 1/3\textsuperscript{rd} will have spontaneous remission
- 1/3\textsuperscript{rd} stable course
- 1/3\textsuperscript{rd} deteriorate possibly life threatening

**Clinical Features**
- Ocular most common (Ultimately 90% of cases) – Ptosis and diplopia
- Generalised weakness (85%) – Muscles of facial movement, talking, mastication, swallowing
- Life threatening Crisis involves respiratory muscles – May require mechanical ventilation
- Affects motor and not sensory nerves

**Osserman Classification**
- Grade I – Focal disease restricted to ocular muscles
- Grade II – Generalised disease
  - IIa – Mild weakness
  - IIb – Moderate weakness
- Grade III – Severe generalised disease
- Grade IV – Life threatening

Prevalence of 1/100 000 in USA
Myasthenia

Diagnosis
- **Tensilon Test** – Edrophonium – Anticholinesterase improves symptoms by increasing levels of AcH
- **Jolly Test** – Repetitive stimulation of peripheral nerve results in reduced amplitude of MEP
- **Single Fibre EMG** – Increased jitter or blocking
- **Antibody assays** – Positive in 80–90 % of patients with MG

Role of Thymus Gland in MG
- 70 –80 % of patients with MG have a thymic abnormalities
- 85 % have thymic hyperplasia
- 15 % have Thymoma

Treatment of MG (4 treatments)
- Enhancement of neuro-muscular transmission (Anti-cholinesterases) – side-effects Diarrhoea, lacrimation, salivation
- Thymectomy
- Immunosuppression, Steroids, AZA, Cyclosporine
- Immuno-modulation – PLEX, IVIGs

Which Patients should undergo Thymectomy
- Patients with generalised MG from Puberty to 60 should be considered ASAP (some say Osserman grade II and above)
- Some advocate Thymectomy for Osserman class I patients refractory to medication
- Patients with associated Thymoma (15%)
- Clinical improvement after Thymectomy seen in 50 – 90% of patients
- Permanent remission in up to 50% of patients (Drug free)
- Clinical improvement may not be seen for up to 3 –5 years following Thymectomy

Surgical Approaches to Thymus
- Median Sternotomy
- Trans-cervical( up to 60% of patients following this are found to have mediastinal recurrence of Thymic tissue)
- Maximum Thymectomy (combines trans-cervical and MS)
- VATS increasingly popular

Prevalence of 1/100 000 in USA No RCT Medicine vs. Surgery
Myasthenia - Surgery

Preoperative
- RFTs, optimise steroids, consider Plasmapheresis

Operative
- Avoid neuromuscular blockers use Volatiles (Provide sufficient muscular relaxation)
- If use neuro-muscular blockers consider
  - Patients with active Myasthenia are resistant to succinylcholine
  - Use 1/10th dose of non-depolarising agents and a lot of volatile

Surgery
- Median Sternotomy
- Pericardial fat pad is divided at level of Diaphragm
- Fatty tissue around lower pole of Thymus is dissected bluntly form Pericardium
- Continue dissection to within 1 cm of Phrenics
- Open Pleura to see and preserve Phrenics
- Continue dissection Cephalad and skeletonise innominate vein (Posterior to IV leave!) leave fat around Phrenics
- Superior poles of Phrenics are identified and easily resected
- Drain or not drain Pleura if no Parenchymal lung injury

Post-operative
- Watch respiratory system
- IV steroids if on them before
- Some withhold anti-choline-esterases post operatively (we do not1)
- Wean immunosuppressive agents

Prevalence of 1/100 000 in USA No RCT Medicine vs. Surgery
Myasthenia – Surgery - Results

- Mortality should be 0%
- Phrenic nerve palsies 0%
- Respiratory complication
- Wound complications
- Symptoms improved by at least 1 Osserman grade in up to 80%
- Overall drug free remission in up to 50% of patients with reduction in AntiC & Immunosuppressive
- Long term drug-free remission seen in 70% of patients with preoperative grades I, II, III
- Long term drug-free remission seen in 29% of patients with preoperative grades IV
- Clinical benefit is not immediate and can improve from 5 – 10 years post Thymectomy

Prevalence of 1/100 000 in USA No RCT Medicine vs. Surgery
Germ Cell Tumours

- Anterior mediastinum is the most common site of extra-gonadal germ-cell tumours
- Commonly affect males aged 25–35 years
- Investigation should include serum markers and testicular examination and ultrasound
- All patients with an anterior mediastinal mass should have (AFP), (β-HCG), (LDH) levels drawn at the outset
- Hematologic malignancies may occur in conjunction with mediastinal non-seminomatous germ cell tumors (NSGCT)
- 80% of NSGT germ cell tumors, AFP is elevated. β-HCG elevated in 30% - 35% of patients (gynecomastia)
- Patients with pure seminomas should never have an elevated serum AFP
- In mature teratomas, AFP, B-HCG and LDH are normal
- Although treatment can be initiated based upon positive tumor marker results, histological diagnosis is recommended
- There is a pathologic discrepancy of 6% between histology and fine-needle aspiration (FNA)
- Core needle biopsy should be performed
- If surgical biopsy is warranted, an anterior mediastinotomy (Chamberlain) is usually the procedure of choice
Germ Cell Tumours

In most NSGCT tumours (except mature teratomas) tumour markers will be elevated. Pure Seminomas do not have elevated tumour markers.
Germ Cell Tumours

Mainstay of Treatment is Chemotherapy
Germ Cell Tumours - Treatment

- Chemotherapy is the mainstay of initial Rx and surgery should be viewed as an adjuvant to chemotherapy
- Teratomas which are encapsulated should be completely excised usually with good results
- Bleomycin, etoposide, and cisplatin (BEP) is the current standard chemotherapy
- Patients with tumor marker normalization and persistent mass on CT following chemotherapy
  - Rx with surgical resection
- Patients with a residual mass on CT and persistently elevated tumor markers
  - Either treat with repeat chemotherapy to obtain normal tumor marker levels prior to surgery
  - Resect and then more Chemotherapy

Incisions include
- Median Sternotomy
- Hemi-clamshells
- Complete Clam shells

Results
- Memorial Sloan-Kettering
  - 32 patients who underwent post-chemotherapy surgical resection of mediastinal germ cell tumors
  - Histologic analysis revealed viable tumor in 66%, teratoma in 22%, and necrosis in 12% of the specimens.
  - and teratoma with malignant transformation to non-germ cell histology (e.g. sarcoma)
Retrosternal Thyroid
Bronchogenic Cysts

- Most common primary cyst in the mediastinum (60%)
- Anomaly of Bronchial development, can occur within wall of Oesophagus. Arise from ventral foregut
- Close to trachea, main stem bronchi, often posterior to carina and right para-tracheal region.
- Communication with tracheal lumen unusual but usually adjacent to trachea with a cartilaginous tract
- Histologically composed of ciliated respiratory epithelium, mucous glands, cartilage
- 2/3rd are asymptomatic
- 1/3rd present with tracheo-bronchial or oesophageal compression symptoms and infection of TBT
- Uncommon in infancy but when occur cause severe respiratory distress due to tracheal compression
- In older child cough, dyspnoea, stridor are common
- Diagnosis usually made on CT / MRI finding of Cystic mass
- If fluid in cyst is infected making cyst more solid diagnosis may be difficult
- Bronchoscopy or OGD may be required to exclude communication with airway or Gut
- Intra-Oesophageal US may also be useful
- No role for preoperative biopsy to make diagnosis
- Surgery is advocated as soon as diagnosis is made to prevent complications and to establish diagnosis
- VATs is becoming increasingly popular

Commonest Primary Cyst of Mediastinum
Bronchogenic cyst in a 21-year-old asymptomatic woman. Transverse contrast-enhanced chest CT scan (mediastinal window settings) shows a well-circumscribed water-attenuation cyst in the middle mediastinum. Note the thin, enhancing wall medially (black arrows) and peripheral punctate calcification (white arrow).
Pericardial Cysts

- Second commonest primary cyst in the mediastinum (30%)
- Arise from mesenchymal lacunae failing to fuse with the pericardial sac
- Commonly occur in cardio-Phrenic angles 70% right, 30% left
- Classically appear as unilocular cyst with smooth border
- On CT hounsfield enhancement of almost 0
- Rarely cause symptoms
- Can be excised or aspirate for Diagnosis
- VATS increasingly used

Second Commonest Primary Cyst of Mediastinum
Lymphoma

- Rarely (5 – 10%) confined to mediastinum
- Look for Pel-Ebstein fever, chills, weight loss, night sweats “B symptoms” (50% of patients)
- Usually anterior-superior mediastinum but can be in hilar region of middle mediastinum
- Hodgkins comprises 30% of all Lymphomas
- NHL comprises 70% of all Lymphomas
- However, Hodgkins comprises 70% of mediastinal Lymphomas
- Nodular Hodgkins is the most common
- Usually involves the anterior mediastinum as well as the Thymus
- Surgery is to provide diagnosis (FNA is usually insufficient)

Treatment
- Hodgkins – Early Radiotherapy later stages Chemotherapy
- NHL – Chemo-Radiotherapy
Neurogenic Tumours

Chest roentgenogram showing the left upper mediastinal mass. (b) CT scan showing a predominantly cystic paravertebral mass. (c) MRI scan horizontal axis revealing a paravertebral mass without any extension into the intervertebral foramina and having solid and cystic areas. (d) MRI scan para-saggital axis showing a well-defined mass in the apex of the thoracic cavity.
Thyroid: Most extend into anterior mediastinum but up to 15% extend into posterior Mediastinum
A) Computed tomography scan showing contrast filled mass adjacent to the ascending aorta. This was reported as aneurysm of ascending aorta. Retrospectively the saccular aneurysm of superior vena cava (SVC) was diagnosed. A clear fat plane separates the mass from the aorta and the mass seems to arise from the SVC. (B) The venogram demonstrating the saccular aneurysm of superior vena cava.

Saccular aneurysm of superior vena cava (SVC)
Oesophagus

- Muscular epithelial-lined tube – length 40 cm from incisors to OG jnct (cervical 5, thoracic 20, abd 2)
- Derived from primitive foregut – ventral trachea, dorsal Oesophagus
- Adventitia: outer loose connective tissue containing nerves, lymphatics, blood vessels (no serosa)
- Muscularis: two layers of muscle--outer longitudinal and an inner circular
- Submucosa: connects muscularis with the mucosa
- Mucosa: Stratified squamous, then columnar; Z-line is 2 cm from OG junction if higher >3cm=Barrett
- Areas of constriction Cricopharyngeus (tightest), LMB & Arch Aorta and OG junction

Arterial: 3 sources
- 1. Inferior and superior thyroid arteries: cervical esophagus
- 2. Tracheobronchial, aortic arch and esophageal branches: body of esophagus
- 3. Left gastric and splenic arteries: GEJ

Venous drainage superiorly into thyroid veins, inferiorly via the azygous

Lymphatics Upper to Paratracheal and Jugular, Distal to Coeliac and Cisterna Chyli (Skip mets occur)

Nerves
- Sympathetic: cervical, thoracic chains; celiac plexus and ganglia
- Parasympathetic: Vagus nerve muscular plexus around circular layer of the muscularis (Auerbach's plexus)
- Submucosal plexus ganglia (Meissner's plexus) – secretory functions
- Auerbach's plexus ganglia between inner circular and outer longitudinal responsible for motor function

From incisors to Cardia = 40 cm, from incisors to Cricoid 15 cm, from Cricoid to Cardia 25 cm
**Peristalsis**
- **Primary:** normal propulsive wave in response to the stimulation of normal voluntary deglutition (30 – 180 mmHg)
- **Secondary:** normal wave without voluntary deglutition: best defense in response to dilatation
- **Tertiary:** abnormal; may occur spontaneously or following deglutition

**Upper Oesophageal Sphincter**
- Level of Cricoid cartilage C 5-6
- Composed of cricopharyngeus and inferior pharyngeal constrictors
- Remains contracted between swallows due to continuous stimulation by IX and XI
- Resting tone: 45 - 65 mmHg (range 32 -101 mmHg)
- Swallow: inhibition of all motor nerve stimulation; UES opens; closes; rebound; baseline pressures

**Esophageal Body**
- Proximal striated muscle: direct innervation to its motor end plate from nucleus ambiguous
- Smooth muscle: indirect neural input from dorsal motor nucleus (X) via myenteric plexus
- Innervation: longitudinal muscle shortens; circular muscle contracts; peristalsis
- Duration and amplitude: weaker in proximal esophagus; stronger, longer in distal esophagus

**Lower Oesophageal sphincter (LOS)**
- Specialized muscle arrangement 3 - 4 cm above gastroesophageal junction (GEJ)
- High pressure zone with resting tone 15 - 25 mmHg (24.8 mmHg)
- Influenced by neural and hormonal factors; drugs
- Relaxes at time of swallowing; closes with passage of contraction through sphincter
Oesophagus Imaging

Barium Swallow 3 different parts; full column view, mucosal relief manoeuvre, air/contrast interface

- **Full Column View**
  - 250 mls of Barium images of full column taken in the upright position and then supine for motor dysfunction

- **Mucosal Relief**
  - Once Barium has gone through and wall of Oesophagus still coated with Barium – mucosal defects can be seen

- **Air/contrast interface images**
  - Gas-producing granules added to Barium or swallowed after to create air – contrast interface. Sometimes called “fizzies”

Limitations

- Limited usefulness in diagnosing Barrett's or Oesophagitis
- Can only detect Reflux in 40%
- Limited in staging Oesophageal Cancer

Cinematographic Oesophagogram

- Videotaped contrast study
- Smaller amount of Barium is swallowed
- Used for diagnosing dysphagia when previous Barium swallow has been negative
- Particularly useful for upper Oesophageal pathology in the oropharynx
Oesophageal Manometry

- Invasive method used to measure amplitude, organisation, and rate of progression of contractions
- Easy, Short outpatient-based test to assess function of sphincters at rest and during swallowing
- Test is 2–3 hours long

Stationary
- Patient lies supine
- Five channel water perfused catheter system lower orifice at LOS & remaining channels at UOS and body of oesophagus
- Catheter is advanced into stomach and pressures are measured during pull-back 0.5–1cm intervals (station pull through)
- 5-ml water bolus

Ambulatory
- 3 microelectrodes and data storage system

Three components of LOS
- Overall Length (Normal 2–4 cm)
- Resting pressure (Normal 12–25 mmHg)
- Abdominal Length (Normal 2 cm if < 1 cm more likely incompetent)
- Intra-thoracic length (if > 1.5 cm then identifies hiatus hernia)

UOS difficult to assess due to quick pressure changes

LOS identified at point of pressure inversion from +ve intra-abdominal to –ve intra-thoracic
Castell’s Classification of Primary Oesophageal Motility (updated 1997)

- **Achalasia**

- **In-Coordinated Motility**
  - Diffuse Oesophageal spasm

- **Hypercontracting Oesophagus**
  - Hypertensive peristalsis (Nutcracker Oesophagus)
  - Hypertensive LOS

- **Hypocontracting Oesophagus**
  - Ineffective motility
  - Hypotensive LOS
Oesophageal Manometry

Functional Zones of Oesophagus
- UOS
- Oesophageal body
- LOS

Stationary
- Multi-channel probe, Water is perfused through the channels at low rate (0.6 ml/min) pressure changes recorded
- Lower orifice is positioned at LOS and remaining channels are located in the Oesophageal body and UOS
- Dry and wet swallows

LOS if resting pressure is < 6mmHg and length < 2cm more likely to be incompetent

Normal findings:
- UOS relaxation in response to pharyngeal swallowing
- Contraction of Oesophageal body increases to 25 – 35 mmHg as it travels towards stomach
- LOS relaxation in response to bolus propulsion

Achalasia
- Failure of LOS to relax
- Poor contraction and peristalsis of Oesophageal body
- Resting LOS pressure is > 35 mmHg
- Mirror-like tracings at all levels (poor peristalsis)
Oesophageal Manometry

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**Oesophageal Manometry**

### Diffuse Oesophageal Spasm
- Oesophageal body contractions are multifocal and of elevated pressures
- Contractions that do not result in peristalsis (high pressure > 180 mmHg)
- Contractions that may be multiple in response to single swallow
- Contractions with multiple peaks
- Intermittent episodes of normal peristalsis

### Nutcracker Oesophagus
- Greatly elevated contractile force
- Prolonged contractile time
- Normal peristaltic pattern
- Peak pressure can reach 400 mmHg
- Pain associated with high amplitude contractions

### Hypertensive LOS
- Increased resting pressure > 25 mmHg
- Remaider of oesophageal function is normal
- LOS relaxation in response to bolus propulsion

### Non-specific Oesophageal motility disorders
- Failure of peristalsis
- Weak disorganised oesophageal body contractions
- Normal resting pressure and relaxation of LOS
Achalasia

- **Incidence**: 1 : 100,000

- **Aetiology**
  - **Primary**
    - Degenerative nerve process with loss of Ganglion cells in myenteric plexus with reduction of vagal nerve fibres in wall of oesophagus
  - **Secondary**
    - Chagas disease
    - Amyloidosis and Sarcoidosis
    - Malignancy (Pseudoachalasia often due to gastric cardia adenocarcinoma clues with age > 55, short duration of symptoms)
    - Viral Infections
    - Diabetes

- **Symptoms**
  - Dysphagia (Liquids > Solids)
  - Reflux of undigested foods
  - Aspiration
  - Squamous cell cancer of Oesophagus on average 20 years after diagnosis

- **Investigations**
  - CXRAY – usually normal but later on Air – fluid levels, loss of gastric bubble, perhaps signs of aspiration
  - Barium – Tapering of Oesophagus “Birds Beak”, Dilated sigmoid like Oesophagus with undigested food
  - Oesophageal Manometry – Incomplete relaxation of LES, Hypertensive LES, ultimately aperistalsis body of Oesophagus
  - Vigorous Achalasia – High amplitude non-peristaltic contraction (Present commonly with chest pain)

**Oesophageal Motility Disorder** characterised by failure of LOS to relax
Achalasia

Barium swallow
"bird's beak or rat’s tail"
Esophageal dilatation; megaesophagus

Peristalsis absent in esophageal body
Contractions weak at all recording levels
Failure of relaxation of a hypertensive LOS
Mirror like activity at all recording levels

Oesophageal Hypo-Motility Disorder characterised by failure of LOS to relax
Achalasia Management

**Pneumatic Dilatation**
- Successful in 70% of cases
- The dilator is placed fluoroscopically so balloon is centred on GEJ
- Dilatation to at least 3 cm Pressure of ????????????
- Should be considered first line therapy in most patients
- Patients who fail two dilatations should be considered for surgical myotomy
- Risk of perforation during 1 – 5%

**Surgical**
- Heller's anterior and posterior Myotomy (Now modified into one single myotomy)
- Surgical transection of outer Longitudinal and inner circular muscles preserving Submucosa
- Palliation in 80 – 90% long-term
- Controversy exists as to whether anti-reflux procedure is required as well
- Oesophageal resection reserved for those failing myotomy or have end-stage mega-oesophagus
- With vigorous Achalasia myotomy may have to be extended to aortic arch!

**Medical Therapy**
- Calcium channel blockers, Nitrates, B Agonists
- Endoscopic Botulinum toxin injection (long-term as effective as Pneumatic dilatation)

**Pneumatic Dilatation Versus Surgery**
- RCT 39 treated with PD Vs 42 with surgery
- Good immediate response both but at 5yrs results sustained in 95% of surgical group vs 65% in PD
- Mayo Clinic paper Myotomy was safer than PD(Mortality 0.2 Vs 0.5%), perforation (1 Vs 4%)

Consider Myotomy as first line treatment in young patients since efficacy of pneumatic dilatation is age dependent
Oesophageal Spasm (Diffuse)

Symptoms
- Dysphagia and pain
- Pain dull or colicky and sub-ternal sometimes radiating into jaws and neck down to the arms and back
- Due to the above and the fact that it is relieved by GTN often confused with cardiac pain

Barium Swallow
- Simultaneous segmental contractions observed in smooth muscle below aortic arch down to LOS
- Typical corkscrew or rosary bead appearance

Manometry
- Oesophageal body contractions are multifocal and of elevated pressures
- Contractions that do not result in peristalsis
- Contractions that may be multiple in response to single swallow
- Contractions with multiple peaks
- Intermittent episodes of normal peristalsis

Treatment
- Initially Calcium channel blockers or nitrates
- Surgery reserved for those not responding to medical Rx
- Pneumatic dilatation can be tried
- Myotomy extending from proximal extent of smooth muscle (aortic arch) onto stomach

Triad of clinical symptoms: Dysphagia(with pain), Barium, & Manometry findings
Nutcracker Oesophagus

- Manometrically defined syndrome
- Patients present with chest pain and dysphagia
- Dyspepsia and regurgitation are usually absent
- Most common oesophageal motility disorder in patients with non-cardiac chest pain
- Barium usually normal
- OGD usually normal

**Manometry**
- Greatly elevated contractile force
- Prolonged contractile time (>6 secs)
- Normal peristaltic pattern
- Peak pressure can reach 400 mmHg
- Pain associated with high amplitude contractions

**Treatment**
- Calcium channel blockers or anti-cholinergics
- In general pneumatic dilatation or surgery are not helpful
Hypertensive LOS & Non-specific Motility disorders

- **Manometric finding**
- **Patients present with dysphagia sometimes chest pain**

- **Manometry**
  - LOS pressure > 45mmHg
  - LOS relaxation > 75%
  - Normal Oesophageal peristalsis

- **Treatment**
  - Most patients respond to medical therapy
  - Dilatation for very select group
  - Rarely Surgery

- **Non-specific Oesophageal Motility disorder**
  - Diagnosis by exclusion of all other motility disorders
  - Manometry may be abnormal but does not fit into any definite single disorder
  - May be failure of, low amplitude, or spontaneous tertiary peristalsis
  - Includes Presbyoesophagus, abnormal oesophageal motility associated with ageing
Gastro-Oesophageal Reflux GOR

Accounts for 75% of all Oesophageal disease
Prevalence of reflux Oesophagitis at endoscopy ranges from 0.5 – 25%

Aetiology & Pathophysiology

- LOS incompetence
  - Less than 1cm intra-abdominal length,
  - Disturbed angle of HIS between Oesophagus & Fundus
  - Inappropriate relaxation
  - Hypotensive LOS < 6 mmHg either as primary cause or secondary to tissue damage or inflammation

- Nature of refluxate
  - Acid or Bile

- Oesophageal mucosal resistance

- Oesophageal clearance mechanisms
  - Gravity
  - Secondary peristalsis (protective)
  - Salivation (alkaline)

- Abnormal Gastric Function
  - Delayed gastric emptying seen in up to 50 % of patients with GOR

- Miscellaneous
  - Pregnancy
  - Previous gastric surgery
  - Zollinger Ellison syndrome
  - Scleroderma
  - Treated Acahalasia
Gastro-Oesophageal Reflux GOR

Complications
- Peptic Oesophagitis
  - Inflammation / ulceration
  - Stricture
  - Barretts
- Motor Dysfunction
- Aspiration

Clinical Presentation

Diagnosis
- Radiology
  - Less than 1cm intra-abdominal length,
  - Disturbed angle of HIS between Oesophagus & Fundus
  - Inappropriate relaxation
  - Hypotensive LOS < 6 mmHg either as primary cause or secondary to tissue damage or inflammation
- Endoscopy
  - Acid or Bile
- PH studies
  - Scleroderma
  - Treated Acahalasia
Oesophageal Diverticula

Pharyngo-oesophageal (Zenker’s)
- Incidence of 2% of patients with dysphagia and 0.1% in general population
- Herniation of mucosa and sub-mucosa between
  - Oblique fibres of inferior pharyngeal constrictors & transverse fibres of Cricopharyngeus (Killians triangle)
  - Theory that the origin is due to failure of Cricopharyngeus muscle to relax on swallowing
- Presentation
  - Dysphagia, Halitosis, retrosternal pain, alterations in voice character, aspiration, neck mass
- Complications
  - Aspiration, fistula formation between Oesophagus and respiratory tract, Squamous cell carcinoma
- Diagnosis
  - Barium Swallow
  - Endoscopy with caution in order to exclude Squamous cell cancer changes
- Surgery
  - Indicated for symptomatic large Diverticula
  - Options are; Diverticulectomy, Diverticulopexy, Cricopharyngeal myotomy, Endoscopic resection of diverticula wall

Midoesophageal
- Traction diverticula as a result of adherence of oesophagus to lymph nodes after TB, Sarcoidosis
- Mostly inter-bronchial level and are true diverticula
- Usually broad based and can be manages conservatively. Surgery reserved for complications

Epiphrenic
- Pulsion diverticula hence 2/3rd have associated oesophageal motility disorder
- Present with dysphagia, chest pain, regurgitation
- Assess with Barium, Endoscopy, Possible perforation, fistula, squamous cell carcinoma
- Surgery reserved for large symptomatic cases via left P/L thoracotomy, diverticulectomy, myotomy

Pulsion are false diverticula since contain only mucosa and sub-mucosa, Traction is true contains all layers
Paroesophageal Hernia

Hiatus Hernia

4 types
- Type I Sliding – OG junction moves Cephalad and stomach follows accounts for a lot of OG reflux
- Type II rolling more uncommon OG junction remains in the right place with Gastric Fundus moving up next to Oesophagus less reflux
- Type III is a combination of types I and II and reflects prolonged type II hernias which eventually enlarges the Oesophageal Hiatus
- Type IV abdominal contents other than stomach in chest

Presentation
- Either asymptomatic diagnosis on routine CRAY
- Symptoms of GO reflux, dysphagia
- Complications such as obstruction, bleeding, Gastric Volvulus

Diagnosis
- CXRAY, Barium swallow “upside stomach” in chest

Therapy
- Indications for surgery is the presence of a hiatus hernia itself (Some say!)
- Others say that sliding are OK, Types II, III, IV require intervention even if asymptomatic
- Belsey and Skinner showed that patients treated conservatively have a 29% mortality rate
- Approach either Laparotomy, Thoracotomy, trans-thoracic laparoscopic, or Laparoscopic
- Principles are
  - Reduction of hernia
  - Excision of hernia sac
  - Closure of hiatal defect
  - Fix stomach in abdomen either Gastrostomy or Fundoplication

Protrusion of Abdominal contents through Oesophageal Hiatus into chest
Paroesophageal Hernia

**Thoracotomy**
- Better for obese patients, previous abdominal operations, ease with which to mobilise contents
- More able to mobilise Oesophagus to achieve length
- However morbidity of incision as well as more difficult to perform Fundoplication from chest

**Laparotomy**
- Chest contents easily come down
- Easier to perform Fundoplication from abdomen
- Less morbidity from incision
- Can’t get adequate length however

**Laparoscopy**
- Between legs
- Retract liver, reduce contents, excise sac, mobilise Oesophagus in chest
- If patient has had reflux preoperatively combine this with a fundoplication which would fix stoch to Oesophagus in abdomen
- Need Manometry before hand in-order to decide whether you are going to do a fundoplication procedure

Protrusion of Abdominal contents through Oesophageal Hiatus into chest
Perforated Oesophagus

**Aetiology**

- Instrumentation (60%)
- Spontaneous (15%) * (Boerhaave’s syndrome)
- Foreign body ingestion (12%)
- Trauma (9%)
- Tumour (1%)
- Operative injury (2%)

**Location**

- Killian’ triangle – Inferior constrictor Pharyngeus and cricopharyngeus (posterior oesophageal mucosa is unprotected by muscularis)
- Anatomical areas of narrowing, OG junction, aortic arch area, LMB area
- Biopsy sites just proximal to Benign or malignant strictures

**Presentation**

- Chest pain, dysphagia, dyspnea, emphysema, epigastric pain, fever, tachycardia
- Cervical perforations – Less severe, Oesophageal attachments to pre-vertebral fascia limits spread of infection
- Thoracic Perforation – Rapidly soil the mediastinum, (Left pleura more commonly involved), sepsis within hrs
- Abdominal Perforation – Early sepsis as in thoracic perforations with acute abdomen

*Mackler’s Triad: 1) Vomiting 2) Sudden Lower chest pain 3) Subcutaneous Emphysema*
Perforated Oesophagus

Diagnosis

- **CXRAY** – (lateral neck for cervical to show air in pre-vertebral plane)
  - Positive in 90% of cases
  - Pleural effusion, Pneumo-mediastinum, Emphysema, Hydro-pneumothorax, air under diaphragm (abdominal perforation)
  - 75% appear within 12 hours of perforation

- **Barium Vs Gastrograffin**
  - Barium was used in early Bronchograms therefore isn’t bad if aspirated. Gastrograffin if aspirated causes Pulmonary Oedema
  - Barium is better visualised if extravasates extraluminally as in a perforation than Gastrograffin
  - Barium however lingers in the mediastinum making follow-up swallows more difficult to interpret

- **Gastrograffin swallow** (Sensitivity 90%)
  - If Gastograffin normal try Barium

- **CT Scan**

- Analysis of pleural fluid

- **Flexible Endoscopy**
  - Recommended in acute trauma to Oesophagus is considered
  - Small mucosal tears if scoped due to air insufflation severe emphysema occurs suggesting much bigger tear

Thoracic > Cervical > Abdominal (Incidence)
Perforated Oesophagus

Determinants

- Cause
- Location
- Severity
- Interval from perforation to presentation (Early < 24 hours, Late > 24 hours)

Aims of treatment (5)

- Elimination of infection
- Prevention of further contamination
- Restoring integrity of Oesophagus
- Providing nutrition
- Correct underlying cause of perforation

Early Diagnosis Mortality 15% Late Diagnosis Mortality 35% Golden Period of closure is first 12 hours
Perforated Oesophagus

**Surgical Options (5)**

- Primary Repair re-enforced repair or not
- Resection
- Drainage alone
- T Tube drainage
- Exclusion and diversion

- Perforation in middle third of Oesophagus approach through right thoracotomy
- Perforation in lower third of Oesophagus approach through left thoracotomy
- Upper midline laparotomy for abdominal oesophagus
- Primary repair remains treatment of choice in all pts without malignancy or diffuse mediastinal necrosis even > 24 hrs
- Drainage alone is not option for thoracic and abdominal perforations only cervical
- Must correct underlying causes such as achalasia with repair + myotomy opposite the side of repair
- Oesophagectomy is a reasonable option with some advocating anastamosis at the same time in neck
- T Tube drainage for those you can not close creates entero-cutaneous fistula allowing perhaps late closure
- Exclusion and drainage – Cervical Oesophagostomy and Gastrostomy (Obviously need reversal out of vogue)

**Without reinforcement Mortality 25% Leak 39%, With reinforcement Mortality 6 % Leak 13%**
Perforated Oesophagus

Non-Operative Management

- Early diagnosis or leak contained if diagnosis delayed
- Leak contained within neck or mediastinum, or between mediastinum and visceral lung pleura
- Drainage into Oesophageal lumen as evidenced by contrast imaging
- Injury not into neoplastic tissue, not into abdomen, and not proximal obstruction
- Signs and symptoms of sepsis are not present
- Contrast imaging and experienced Thoracic surgeon is available

Non-operative management includes NBM for 48 – 72 hours

- TPN
- Broad Spectrum antibiotics
- Drainage of collections (CT guided if need be)
- Suction guided endoscopically placed catheters
- Observation and conversion into more aggressive management must be at hand if no improvement
You should still attempt primary repair of a cervical perforation approached through the left neck.
## Perforated Oesophagus

### Table 1. Outcome After Treatment of Esophageal Perforation in Series Published Between 1990 and 2003

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Number of Patients</th>
<th>Number of Deaths</th>
<th>Mortality (%) Mean (Range)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary repair</td>
<td>322</td>
<td>40</td>
<td>12 (0–31)</td>
<td>5–7, 10, 14, 16, 17, 42, 55, 90–2, 95</td>
</tr>
<tr>
<td>Resection</td>
<td>129</td>
<td>22</td>
<td>17 (0–43)</td>
<td>5–7, 12, 16, 42, 64, 90–2</td>
</tr>
<tr>
<td>Drainage</td>
<td>88</td>
<td>32</td>
<td>36 (0–47)</td>
<td>5–7, 16, 17, 42, 91</td>
</tr>
<tr>
<td>Exclusion and</td>
<td>33</td>
<td>8</td>
<td>24 (0–80)</td>
<td>5–7, 17, 76, 77, 96</td>
</tr>
<tr>
<td>Nonoperative</td>
<td>154</td>
<td>26</td>
<td>17 (0–33)</td>
<td>7, 8, 13, 14, 42, 88, 90–2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>726</strong></td>
<td><strong>128</strong></td>
<td><strong>18 (0–80)</strong></td>
<td></td>
</tr>
</tbody>
</table>

Cervical 6%, Thoracic 34%, Abdominal 29% (Mortality)
Oesophageal Trauma

- Look at this as you would for the perforated Oesophagus
- With added facts that Penetrating trauma is more common in the cervical Oesophagus
- Blunt Oesophageal trauma is very rare
- Diagnostic Pathways are same as perforated Oesophagus but sometimes other injuries will take precedence in trauma
- Surgical principles are the same 5 points:
  - Drain the sepsis
  - Prevent further spillage
  - Restore Oesophageal continuity
  - Correct any Oesophageal Pathology
  - Establish nutrition
- Emphasis that primary repair should be the goal at all times (Quote Jones & Gingsberg review) irrespective of timing
- Other Options are still there
  - Drain and Exclude
  - T tube entero-cutaneous fistula
  - Primary resection
  - Drainage
- Think about approach and must re-inforce repair, broad spectrum antibiotics
- Contrast swallow before resuming eating (around 7 days)
Oesophageal Trauma – Corrosive Injuries

- Accidental or intentional
- In children or adults by accident damage to Oesophagus is limited because they spit it out
- Deliberate causes extensive damage
- Acid causes coagulative necrosis
- Alkaline causes Liquefaction necrosis
- Gentle endoscopy to assess depth of injury and report as Burns 1st, 2nd, 3rd degree
- Treatment is usually supportive unless perforation or full thickness necrosis has occurred
- Resect all necrotic material and do not re-establish continuity until recovery has occurred

Foreign Bodies:

- PA and Lateral CXRAY
- Barium swallows will obstruct view of a foreign body
- If subcutaneous Emphysema is seen then perforation has occurred
- Rigid endoscopy is carried out to remove
- Cover sharp ends of sharp foreign body
- Pull to scope and pull both out together
Oesophageal Cancer Pathology

**Incidence:** 1.5% of cancers, 5/100,000 in USA, 500/100,000 in Iran

**Age:** 60 – 70 years rarely below 40

**Sex:** M > F

**Geography:** Striking geographical variations China, Iran, South Africa, France

**Aetiology:**

- Diet implicated with dietary carcinogens nitosamines, lack of vitamins, Betel leaf chewing, hot beverages

- Smoking and alcohol particularly with **Squamous** (SCC) pathology

- Intrinsic Oesophageal disease
  - Barrett’s – Columnar epithelium lined Oesophagus 30 – 40 times more prone to **adenocarcinoma**
  - Plummer-Vinson – Atrophy of oropharyngeal mucosa secondary to Fe and Vitamin deficiency
  - Achalasia – 1-10% of patients with Achalasia of > 20 years will develop **Squamous** Cell carcinoma
  - Erosive / caustic strictures /injuries

- Genetic - blood group A, HLA A2, B40 associations, tumour suppressor gene p53 short arm chr. 17

- Hereditary- Tylosis (Hereditary hyperkeratosis palmaris et plantaris) autosomal dominant high frequency of SCC
Oesophageal Cancer Pathology

- **Squamous**
  - Most common worldwide (80%) – 40 – 60 % in Europe and N. America (increasing incidence of Adenocarcinoma)
  - Upper Oesophagus 10 –20 % (up to 25 cm from incisors from crico-pharyngeus to aortic arch)
  - Middle Oesophagus 50% (25 – 32 cm from incisors or from aortic arch to inferior pulmonary veins)
  - Lower Oesophagus 30 –40% (33 – 42 cm from incisors)
  - Local invasion is common, submucosal spread (contiguous) is also common found at 10cm from main tumour in 6%
  - Metastasis to local lymph nodes seen in 50% major determinant of survival
  - Lung 30 – 50%, Liver 20 – 50%

- **Adenocarcinoma**
  - 40% of Oesophageal tumours in North America
  - Should be distinguished from Adenocarcinomas of Gastric Cardia
    - An associated Barrett’s Epithelium
    - Greater than 75% of the tumour mass involving body of tubular Oesophagus
    - Direct invasion of peri-oesophageal tissues
    - Minimal Gastric involvement
    - Clinical symptoms of Oesophageal obstruction

- **Uncommon Malignancies**
  - Undifferenctiated, Small Cell, Carcinoid, Melanoma

No difference in post-surgical 5 year survival by cell type
Oesophageal Cancer

Presentation

- Primary tumour
  - Dysphagia 90%
  - Pain < 20%
  - Respiratory symptoms

- Distant Metastasis
  - At least 50% of patients will have evidence of systemic disease at time of presentation
  - Of all metastasis Lung (30–50%), Liver 20–50%

Diagnosis

- Physical Examination – look for supraclavicular lymph nodes
- Routine Blood – Anaemia, LFTs, Hypercalcaemia, hypo-proteinaemia
- CXRAY – Pulmonary mets, pneumonia, air-fluid levels
- Barium Swallow – Defines fore-gut anatomy, level of obstruction
- OGD – Exact site of tumour, relationship to crico-pharyngeus and OGJ, state of stomach for conduit, Biopsies, dilatation
- CT – Good for Stage IV determination. Accuracy 60 – 90%, nodes accuracy 50 –60% (see later), not good for T1 – T3
- Endoscopic US – Excellent for T stage with 80 –95% accuracy. Can also guide node biopsy. Gold standard for T stage
- Bronchoscopy – For all tumours at or above level of Carina (25cm)
- Laparoscopy – In thing. Gold standard for M1 staging in terms of distal 1/3rd adenocarcinomas
- MRI/ PET – No evidence of superiority over CT
16. Compare the accuracy of EUS versus CT scan.

<table>
<thead>
<tr>
<th></th>
<th>EUS</th>
<th>CT SCAN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Determining tumor stage</td>
<td>80–90%</td>
<td>60%</td>
</tr>
<tr>
<td>Staging regional lymph nodes</td>
<td>80%</td>
<td>50%</td>
</tr>
<tr>
<td>Assessing early esophageal tumors</td>
<td>Good</td>
<td>Ineffective*</td>
</tr>
<tr>
<td>Assessing distant metastasis</td>
<td>Poor</td>
<td>Good</td>
</tr>
</tbody>
</table>

*CT scan is ineffective at differentiating early-stage tumors from more advanced tumors.

Figure 2. Endoscopic Image (Panel A) and Endoscopic Ultrasonogram (Panel B) showing a Transmural Adenocarcinoma of the Esophagus Associated with Barrett’s Esophagus (Short Arrows), with Lymph-Node Metastases (Long Arrow). Courtesy of John Saltzman, M.D.
Oesophageal Cancer Staging

1. **T descriptor**
   - Tis. Carcinoma in situ.
   - T1. Tumor invades lamina propria, muscularis mucosa or submucosa, but does not extend into the muscularis propria.
   - T2. Tumor invades muscularis propria.
   - T3. Tumor extends beyond the muscularis propria into the periesophageal tissues.
   - T4. Tumor invades adjacent structures.

2. **N descriptor**
   - N0. No regional lymph-node metastasis.
   - N1. Regional lymph-node metastasis.

3. **M descriptor**
   - No distant metastases.
   - Distant metastases.

For tumors of lower thoracic esophagus:
   - M1a. Metastasis in coeliac lymph nodes.
   - M1b. Other distant metastasis.

For tumors of upper thoracic esophagus:
   - M1a. Metastasis in cervical lymph nodes.
   - M1b. Other distant metastasis.

For tumors of mid-thoracic esophagus:
   - M1a. Not applicable.
   - M1b. Nonregional lymph node or other distant metastases.

**Table 1. TNM subsets for the stages of esophageal cancer**

<table>
<thead>
<tr>
<th>Stage</th>
<th>TNM subsets</th>
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<tbody>
<tr>
<td>0</td>
<td>Tis</td>
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<tr>
<td>I</td>
<td>T1</td>
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<tr>
<td>IIA</td>
<td>T2</td>
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<tr>
<td>IIIB</td>
<td>T1</td>
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<tr>
<td>III</td>
<td>T2</td>
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<tr>
<td>IV</td>
<td>Any T</td>
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<td>IVA</td>
<td>Any T</td>
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<td>IVB</td>
<td>Any T</td>
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<td>N0</td>
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<td>Any N</td>
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</tbody>
</table>

In Oesophageal Cancer Staging M stage as well as referring to distant metastasis also refers to distant nodes.
Oesophageal Cancer Staging

An adenocarcinoma of the distal esophagus invades the muscularis propria with metastases to the paraesophageal and celiac axis lymph nodes. The stage of this tumor is

A. T2 N1 M0, Stage IIB.
B. T2 N1 M1, Stage IV.
C. T3 N1 M0, Stage III.
D. T3 N1 M1, Stage IV.
E. T4 N1 M0, Stage III.

In Oesophageal Cancer Staging M stage as well as referring to distant metastasis also refers to distant nodes

Answer B
Oesophageal Cancer

Significance of Staging

- In Node negative patients T stage has big prognostic impact
  - T1 N0 5 year survival is 75–85% (Secrets in Thoracic Surgery 50%)
  - T3 N0 5 year survival is < 25%

- In Node positive patients T stage has no impact on survival
  - N1 M0 5 year survival is < 10%
  - N0 M0 5 year survival is > 25% (Note all T classifications)
Oesophageal Cancer – Adjuvant and neo-adjuvant therapy

Radiotherapy

- **Preoperative** - No evidence for its use either preoperatively in terms of improved survival
- **Postoperative**
  - 2 RCTs
  - No survival advantage (overall or disease free). **Can not be currently recommended**

Chemotherapy

- Single Agent and combination chemotherapy – Cisplatin, 5FU, Mitomycin, MTX
- Cisplatin & 5 FU now standard double agent therapy with response rates of 50% for local disease and 30% for metastasis
- Preoperative (neo-adjuvant)
  - Potentially eliminate systemic micro-metastasis and improve resectability
  - **OEO2 showed definite survival benefit**
- Postoperative chemotherapy
  - Few Trials can not currently recommend use of post-operative chemotherapy

Multi-modality therapy

- Walsh et al (NEJM 1996) Multi-modality therapy Vs Surgery alone
- RCT in patients with locally advanced invasive adenocarcinoma randomised to
  - Surgery alone or
  - 2 courses of Cisplatin, 5 FU with 40Gy of radiotherapy followed by Surgery
- Striking improvement in survival in patients receiving the multi-modality therapy
- Median survival of 16 months for the combined-modality group Vs 11 months for the surgery alone group
- After 3 years only 6% of the surgery alone group were alive Vs 32% for the combined modality group
- Critique: Surgery only group had a very bad 3 year survival why? Groups not balanced at randomisation

**CTSNET grand round Mayo clinic lecture definite survival benefit in locally advanced cancer recommends;**

Stages I – IIa Surgery alone, Stages IIb and III – Combination therapy chemoradiotherapy as well as surgery, Stages IV - Palliative
Oesophageal Cancer – OEO2 Trial

- Lancet 2002
- 800 Patients resectable Oesophageal Cancer of any cell type
- 400 Randomised to Preoperative Cisplatin-based Chemotherapy then 4 wks later surgery
- 402 Randomised to just resection
- Complication rates post-operatively the same
- More complete resection in the surgery and chemotherapy group
- Median survival 17 months in Chemotherapy/surgery group vs. 13 months for surgery
- 2 year survival was 43% in the Chemotherapy/surgery group vs. 34% in surgery group

Concluded
- Two cycles of pre-operative Cisplatin and 5FU improves survival without additional serious adverse events in patients with resectable Oesophageal Cancer
Oesophageal Cancer Surgery

- 50% of patients will be judged by preoperative staging to have resectable disease
- Early Tis, Stage I – Complete surgical resection may be curative with 5 yr survival 70%
- Surgical Mortality should be < 5%
- Morbidity 20 – 40%

Preoperative
- Correct anaemia, dehydration
- Role of preoperative correction of nutritional status is controversial
- Long-term nutritional support may correct deficiencies but places patient at risk of either aspiration or sepsis

Surgical principles
- Proximal resection margin should be 10 cm to include contiguous mucosal spread and skip lesions
- Distal resection margin 5 cm
- En bloc resection to obtain clear lateral resection margins (No evidence for role of extended lymphadenectomy in improving survival)
- 7 Approaches
  - Laparotomy first followed by Right Thoracotomy (Ivor-Lewis)
  - Right thoracotomy / laparotomy (as for transhiatal) / left neck (Middle Oesophageal tumours)
  - Left Thoraco-abdominal - Extending incision across costal margins into abdomen (Distal 1/3rd tumours)
  - Thoraco-laparotomy – Incising diaphragm (thoraco-phrenotomy) (Distal 1/3rd tumours)
  - Transhiatal – non-thoracotomy (Distal 1/3rd tumours)
  - VATS
  - Trans-sternal

Post-Surgical 5 year Survival is 20% All Stages
Oesophageal Cancer Surgery

**Objectives**
- Alleviate dysphagia
- Curative resection
- Minimise morbidity

**Cervical Vs Thoracic anastamosis**
- Cervical more chance of leak (>10%) but when leaks occur less life threatening easier to drain
- Thoracic less chance of leak (<5%) but more life threatening more difficult to drain

**Surgical approaches**
- **Ivor Lewis (Laparotomy followed by right thoracotomy)**
  - Precise oesophageal and node dissection, anastamosis in chest (less likely to leak)
  - Leaks more life threatening, pain from thoracotony
- **Right thoracotomy / Laparotomy, Cervicotomy (McKeown)**
  - Precise oesophageal and node dissection, anastamosis in neck (less life threatening if leaks)
  - More suitable for Proximal and Middle tumours
  - Pain from thoracotomy, three incisions, three position changes
- **Transhiatal (Laparotomy and Cervicotomy)**
  - Reduced pain (no thoracotomy), Anastamosis in neck, generous proximal margin
  - Imprecision in Oesophageal and node dissection
  - More RLN palsies, only for distal third tumours
- **Left Thoracotomy / thoraco-laparotomy**
  - Best suited to distal 1/3rd tumours as aorta and arch get in way
  - Less Proximal resection margins, painful incision, intrathoracic anastamosis

Post-Surgical 5 year Survival is 20% All Stages
Pectus Deformities

**Pectus Excavatum 90%**

- Sunken chest concave
- Overgrowth of costal-cartilages leading to depression of sternum
- Increased elasticity or weakness of connective tissue seen in Marfan’s (10% of Marfan’s will have Pectus Excavatum)
- Familial incidence of Pectus Excavatum
- 5:1 Male to Female ratio
- Progressive depression of sternum through childhood years with accelerated growth spurt around puberty
- Exercise intolerance, static pulmonary function may be normal, sophisticated exercise testing shows decreased ET
- Echo may show decreased ejection fraction

**Pectus Carinatum (Pigeon chest) 10%**

- Convex protuberant abnormality
- Overgrowth of costal cartilages leading to protuberance of chest wall
- Classically occurs around puberty growth spurt (Rarely before 12 years of age)
- Pain may be a feature
- No Physiological component appears with defect

Patients with Pectus Deformity have issues with body image
Pectus Deformities

**Pectus Excavatum 90%**
- Assess cardio-respiratory reserve including Echocardiogram
- Screen for possible connective tissue disorders
- Measurement of Pectus Defect
  - In past distance between back of sternum and anterior spine
  - Now CT Pectus Index Width of chest between ribs divided by AP distance between sternum and Spine (>3 then significant)
  - Callipers which measure deepest part of defect at mid-clavicular line (>2.5 cm then severe, correlates with Pectus Index of >3)

**Management**
- Optimum age for repair of a Pectus Excavatum is between 8 - 12
- Not advised for children <7
  - Fear of Jeune’s syndrome (asphyxiating thoracic dystrophy) due to damage of growth areas of rib cartilage

**Modified Ravitch Procedure**
- Transverse submammary incision (Make preoperative plan regarding how many and draw it on skin)
- Develop subcutaneous flaps
- Pectoralis Major flaps created elevating the muscle off the costal cartilage bilaterally
- Remove downcurved costal cartilages, sub-perichondrial, bilaterally
- Dissect sternum off pleura and pericardium
- Above level of excised costal cartilages, cuneiform Sternal Osteotomy
- At same level of osteotomy bevel cut the corresponding costal cartilage to help keep sternum elevated
- Sternal Bar or plate (Adkins) (Abrams) placed transversely retrosternally and rests on ribs bilaterally anchored with Prolene
- Drain everything

**Results & Complications of Modified Ravitch Procedure**
- Complications (All < 5%)
  - Haemothorax, Pneumothorax, Seroma, wound infection
- Recurrence
  - Up to 10%

Patients with Pectus Deformity have issues with body image
Pectus Deformities – Nuss Procedure

- Transverse bilateral incisions continued down to pleural space
- Incision to allow Bone hook just beneath Xyphoid to elevate sternum (not shown here)
- Vascular clamp passed from right to left to guide insertion of Lorenz Bar which is then stabilised laterally (some use VATS)
- Post-operatively significant pain, Haemothorax, Pericardial injuries seen but good results
- Bar stays for at least 2 years (Bar moves in < 2% of cases)
- Since no cutting of costal cartilages less scar which may account for less recurrence rates seen

For School Age Children and adults as well as recurrent Pectus Excavatum)
Pectus Deformities

Pectus Carinatum (Pigeon chest) 10%
- Classically occurs around puberty growth spurt (Rarely before 12 years of age)
- DO NOT OPERATE ON THEM UNTIL AFTER PUBERTAL GROWTH SPURT OTHERWISE RECURRENCE
- No Physiological component appears with defect therefore no investigations required preoperatively
- Purely a cosmetic procedure (Sometimes pain)
- Technique as modified Ravitch with
  - Osteotomy Modifications (depress the sternum) & hold it there
  - Perichondrial reefing
- Postoperative pain, wound problems, recurrence in non excised cartilages

Patients with Pectus Deformity have issues with body image
Positron emission tomography, is a diagnostic examination that involves the acquisition of physiologic images based on the detection of radiation from the emission of positrons. Positrons are tiny particles emitted from a radioactive substance administered to the patient. The subsequent images of the human body developed with this technique are used to evaluate a variety of diseases.

Similar to PET, single photon emission computed tomography (SPECT) uses radioactive tracers and a scanner to record data that a computer constructs into two- or three-dimensional images. A small amount of a radioactive drug is injected into a vein and a scanner is used to make detailed images of areas inside the body where the radioactive material is taken up by the cells. SPECT can give information about blood flow to tissues and chemical reactions (metabolism) in the body.
Positron Emission Tomography (PET)

- Non-invasive imaging technique relying on focal metabolic differences between tumour and normal cells
- Tumour cells have a higher rate of glycolysis than surrounding normal cells
- \(^{(18)}F\) Fluorodeoxyglucose is used as a glucose analogue
- Radioactive label \(^{18}F\) is Positron emitting isotope undergoing annihilation reaction with electron emitting \(\gamma\) ray
- FDG competes with glucose, is taken up, accumulates, and lights up (standardised Uptake Value (SUV))
- Most sensitive tool for detecting nodal involvement both intra- and extra-thoracic
- Best used in conjunction with CT to localise nodal involvement
- Sensitivity 88% for nodal disease (same sensitivity as Mediasinoscopy)
- Sensitivity approaches 95% for the detection of malignancy in focal lung abnormalities
- Often shows involvement of nodes that are less than 1cm in diameter
- Specificity 90% (granulomas, infection, adenocarcinomas, atelectasis, aspergillomas, mistaken for N2) *
- Used on individual basis
- Mostly in patients unable or unwilling to undergo biopsy of focal lung abnormality
- SPN surveillance
- Individual decision based on whether a missed cancer or unnecessary invasive procedure is the greatest risk

19 Causes of false positive PET scans (Bakheet and Powe)
Pneumothorax Spontaneous (SP)

Accumulation of air in the pleural space leading to lung collapse

Primary (80% of SP) – In otherwise normal lungs, sub-pleural blebs

- Males > Females
- Young people, 25 – 30 years old
- Tall thin, Marfan’s etc
- Associated with smoking
- Familial History often there
- If happened before, 90% chance that it was on that side
- Sometimes coughing or sneezing
- Aspiration is as good as drainage (Failure rate as high as 50%)

Secondary (20% of SP) – In diseased lungs

- 45 – 65 years old, 90% on right
- Can go unnoticed since mimics COPD in SOB
- Since loss of elastic recoil, lung goes down slower and less spectacularly
- COPD most common, anything that breaches visceral pleura, from infection to endometriosis to neoplasm

After first SP 33% will recur after a 2nd 60%, 3rd – 80%
Spontaneous Pneumothorax (SP)

BTS – Observation for small < 20%, closed, mildly symptomatic SP

- 1.3% (50-75ml/day) of intrapleural air will be absorbed daily therefore may take weeks to resolve
- If SP is secondary e.g. to COPD odds on patient will be symptomatic therefore ICD/Aspiration

BTS - Recommendations for intervention in SP

- Indicated after 2nd ipsilateral SP or first contralateral SP
- After 1st episode if after prior Pneumonectomy
- After 1st episode in Airline pilots, divers, individuals in Wales!
- Complications of SP – Haemothorax, empyema
- Massive air leak preventing lung re-expansion
- Large cysts

Bedside pleurodesis is more effective at preventing recurrence than simple ICD

VATs superior over ICD at preventing recurrence

VATs vs. Limited Thoracotomy

- VATS recurrence 2-5% vs. Limited Thoracotomy 1-2%

*Number of cm that lung is down from apex, how far down the lateral wall is the lung retracted, or distance from lateral chest wall at mid thorax
**Persistent Air Leak**

- Definition of persistent air leak is very variable some quoting air leak after 3 days as persistent
- Some advocate surgical intervention for persistent air leak (USA) particularly if Primary SP
- In Secondary SP probably give them longer to resolve (>3 days) before considering operating
- Most would recommend that if after 14 days of persistent air leak then should intervene either
  - Surgery (VATS vs. Limited Thoracotomy)
  - Chemical Pleurodesis
- Consider if lung is up and not very symptomatic home with flutter bag
- If patient is not fit for surgery then providing lung is up chemical pleurodesis can be tried

**Consider Early CT more in Secondary Spontaneous Pneumothorax**

- Giant Bullae
- Other Pathology

*Number of cm that lung is down from apex, how far down the lateral wall is the lung retracted, or distance from lateral chest wall at mid thorax*
Spontaneous Pneumothorax (SP)
Spontaneous Pneumothorax (SP)

Deep Sulcus Sign
Spontaneous Pneumothorax (SP)

Pneumomediastinum
Pulmonary Metastasis

**Diagnosis**

- Only 15% are symptomatic
- Usually detected on CXRAY
- Xray detects them when > 8mm in dimension
- CT will demonstrate metastasis when only 3mm

**Previous history of Sarcoma or Melanoma increases the probability of a pulmonary lesion as being a metastasis 10X**

**Previous history of colorectal/genitourinary malignancy increases probability of pulmonary lesion being 2 by 50%**

**Previous history of Head/neck squamous cell cancer suggest that a pulmonary lesion is a second primary by 2 fold**

**3 Preoperative aspects of assessment before metastectomy:**

1. Look at primary site for any residual disease
2. Look for other likely secondary sites e.g. Sarcoma bone scan, Melanoma, Liver, Adrenals, Brain
3. Patients cardiovascular and respiratory reserve

**Criteria that all patients must meet**

- Primary Cancer is under control
- No other secondaries outside Lungs
- It is possible to excise all lung metastasis. Namely multiple nodules in different lobes precludes this. Use lung-sparing techniques
- Patient is fit to tolerate planned resection
- Histology shows that surgery is the only cure as opposed to either Chemo- or radiotherapy
Pulmonary Metastasis

Prognostic Factors

- Disease free interval the longer the better
- Number and anatomic location of the metastasis
- Tumour doubling time the longer the better the prognosis
- Response to Chemotherapy
- Histology
  - Germ cell do best 68% 5 year survival following resection (from gonads not mediastinal)
  - Melanomas do worst 21% 5 year survival
  - Osteogenic better than soft tissue sarcomas (35% vs. 25% at 5 years)
  - Epithelial (Carcinomas) up to 37% 5-year survival

Surgical Approaches

- Posterolateral thoracotomy
  - Advantage: Good visualisation
  - Disadvantage: Access to one hemithorax only. May need staged thoracotomies with 1-2 week interval for bilateral disease

- VATS
  - Advantage: Good visualisation minimal morbidity
  - Disadvantage#: Can miss lesions not on CT due to lack of ability to palpate lungs & may seed tumour in port sites

- Median Sternotomy
  - Advantages - Both lungs at same time, less patient discomfort
  - Disadvantages - Access to posterior thorax, especially left lower lobe, may be difficult
At rest the normal excursion of the diaphragm is about 1.5 cm and the normal intrapleural pressure change is minus 4 – 9 mmHg (or 5 – 10 cm H2O)
Pulmonary Physiology

- Static refers to study of forces bringing about equilibrium
- Normally lungs have a natural tendency to recoil inwards
- Chest Wall has natural tendency to move outwards
- Because of these two opposing forces the lungs are at their resting volume (Functional Residual Capacity)
  - Inward recoil of lungs = outward force of the chest wall
  - Is the volume remaining in the lungs when the recoil pressure cancels the outward chest wall pressure
- Lung Compliance is Change in Volume per unit of pressure Normal 200ml / 1cm H2O(lungs only)
- That is every time that trans-pulmonary pressure increases by 1cm of water the lungs expand by 200ml
- Elastance is the opposite of Compliance
- Negative intrapleural pressure is more negative in apex of lung
- Hence Alveoli at apex are more distended and working less efficiently at upper end of their compliance curve
- Hence ventilation is said to be less effective at the apex of the lung
- Intrapleural pressure is always below atmospheric pressure throughout a normal ventilatory cycle
- Intrapleural pressure is always below alveolar pressure

At rest the normal excursion of the diaphragm is about 1.5 cm and the normal intrapleural pressure change is minus 4 – 9 mmHg (or 5 – 10 cm H2O)
Pleural Space Physiology

- Intrapleural negative pressure is due to tendency of elastic lung to collapse and rigid chest wall to expand.
- Maintained by continual absorption of fluid from intrapleural space by pleural capillaries.
- Measured using intra-oesophageal Balloon.

At rest the normal excursion of the diaphragm is about 1.5 cm and the normal intrapleural pressure change is minus 4 – 9 mmHg (or 5 – 10 cm H2O).
Lung Compliance (ΔV/ΔP)

- Change in Pressure relates to changes in Trans-pulmonary Pressure (Difference between Alveolar and Pleural)
- Change in lung Volume (L)
- Normal compliance is 200ml/1cm H2O change for the lungs alone (1/2 of that for entire lung/chest wall 100ml/cm)

Inversely related to lung volume, decreasing at end-inspiration
Characteristics of compliance curves are determined by the elastic forces of lungs (2):

- Elastic forces of lung itself to recoil (Loss of elasticity as in COPD improves compliance more V for same P)
- Elastic forces caused by surface tension of the fluid that lines the inside walls of the Alveoli

By filling the lungs with fluid there is no air-fluid interface hence the surface tension effect is abolished.

This shows that surface tension accounts for 2/3rd of elastic forces and tissue elastic forces for 1/3rd.
Lung Compliance ($\Delta V/\Delta P$) – Emphysema Vs Fibrosis

![Graph showing the comparison of lung compliance between Emphysema and Fibrosis](image)
Pulmonary Physiology – Ventilation difference between Apex & Base

Weight of lung accounts for this

Poor Ventilation at Apex

Better ventilation at base

Figure 2–30. Intrathoracic pressure gradient in the upright position. The negative intrathoracic pressure is normally greater in the upper lung regions as compared to the lower lung regions. Because of this, the alveoli in the upper lung regions expand more than the alveoli in the lower lung regions. This condition causes alveolar compliance to be lower in the upper lung regions, and ventilation to be greater in the lower lung regions.
Lung Zones of West

PA = Alveolar pressure does not change between different zones but is constant

Pa (and Pv) = Pulmonary artery pressure which does change

- 15mmHg (20cm/H2O) at level of Main PA (Average Pulmonary artery Pressure)
- Add 15 cm of Hydrostatic Pressure to the main PA level for Zone 3 (= 35 cmH2O at base)
- Subtract 15 cm of Hydrostatic pressure from the main PA level for Zone 1 (=5cmH2O only)

Zone 1

- Upper 1/3rd of lung PA > Pa > Pv (Pa = 5cm H2O)
- Alveolar pressure (PA) greater than pulmonary artery pressure (Pa) which is greater than pulmonary vein (Pv)
- Therefore poorly perfused throughout respiratory cycle

Zone 2

- Middle 1/3rd
- Pa > PA>Pv (Pa = 20 cm H2O)
- Perfusion only when intravascular pressure exceeds alveolar pressure i.e. inspiration

Zone 3

- Lower 1/3rd
- Pa>Pv>PA (Pa = 35 cm H2O)
- Perfusion throughout respiratory cycle
Figure 5-18. Relationship between gravity, alveolar pressure (PA), pulmonary arterial pressure (Pa), and pulmonary venous pressure (Pv) in different lung zones. Note: The +2 cm H₂O pressure in the alveoli (e.g., during expiration) was arbitrarily selected for this illustration.
Ventilation and Perfusion Matching

Last 2 slides have shown that

1) Ventilation is best at lung bases

2) Perfusion is best at lung bases (Zone 3)

Therefore Ventilation and Perfusion are matched
 Spirometry

With exception of RV, All volumes can be obtained by Spirometry
FRC Determination

With exception of RV, All volumes can be obtained by Spirometry
FRC Calculations - Helium

- Breathing at rest known concentration of Helium (%) (In this case 6%)
- With gentle breathing the alveoli equilibrate with system containing Helium arriving at a new concentration (3%)
- From this you know the volume added to the system hence the FRC concentration and you the concentration
- If you have bullae that do not participate in ventilation (air trapping) FRC will be under-estimated by Helium
- In above scenario Body Plethysmography will be a better measure of FRC and hence TLC
FRC – Body Plethysmography

- Based on Boyle’s Law $P_1 \times V_1 = P_2 \times V_2$
- Pressure is inversely proportional to Volume at constant temperature
- Pressure transducers in box and mouthpiece; hold cheeks in breathe normally then shutter comes down and pant
- Pressure changes and hence volume FRC is obtained
Flow-Volume Loops

Peak Flow (10 l/sec)

Top is Dry Rolling Seal Spirometer, Bottom is Flow measuring Pneumotachograph
Flow-Volume Loops - Examples

A. Normal
B. Emphysema
C. Unilateral main-stem bronchial obstruction
D. Fixed UAO
E. Variable extrathoracic UAO
F. Variable intrathoracic UAO
G. Restrictive parenchymal lung disease
H. Neuromuscular weakness
Forced Expiratory Flow

Figure 4–15. The effort-dependent and effort-independent portions of a forced expiratory maneuver in a flow-volume loop measurement. FVC = forced vital capacity.

**Forced Expiratory Flow**$_{200–1200}$ (FEF$_{200–1200}$)

The average rate of airflow between 200 and 1200 ml of the FVC (Figure 4–6); formerly known as the maximum expiratory flow rate (MEFR). The first 200 ml of the FVC is usually exhaled slower than the average flow rate because of (1) the inertia involved in the respiratory maneuver, and (2) the general unreliability of the equipment response time. Because the FEF$_{200–1200}$ measures expiratory flows at high lung volumes (i.e., the initial part, or the effort-dependent portion,* of the FVC), it is a good index of the integrity of large airway function. The average FEF$_{200–1200}$ for the healthy male between 20 and 30 years of age is about 8 L/sec (480 L/min), and for the female between 20 and 30 years of age is about 5.5 L/sec (330 L/min). In obstructive lung disease, however, flow rates as low as 1 L/sec (60 L/min) have been reported. The FEF$_{200–1200}$ decreases with age and in obstructive lung disease. Conceptually, the FEF$_{200–1200}$ is similar to measuring, and then averaging, the flow rate from a water faucet when 200 ml and 1200 ml have accumulated in a measuring container (Figure 4–7).

*See The Effort-Dependent Portion of a Forced Expiratory Maneuver, later in this chapter.
Figure 5.12 a) A patient with chronic obstructive pulmonary disease showing early collapse of large airways and a sudden drop in flow early in the expiratory part of the manoeuvre. The inspiratory limb is unaffected as the airways are being opened up by transmural pressure; b) example of a flow volume loop from an elderly subject showing the curvilinearity in the latter part of the expiratory limb; c) a patient with asthma shows a smooth curvilinear drop in flow with respect to volume indicating intrapulmonary airflow limitation. The inspiratory limb is relatively unaffected; d) variable extrathoracic upper airway obstruction due to goitre showing decapitation of the expiratory part of the loop with more extreme limitation of the inspiratory limb due to collapse of the trachea during inspiration; e) intrathoracic central airway obstruction showing decapitation of the expiratory limb of the loop but little, if any, reduction in the inspiratory limb. This was due to an intrathoracic retrosternal goitre.
GAS DIFFUSION

FICK'S LAW
The diffusion of gas takes place according to Fick's law, which is written as follows:

\[ V_{\text{gas}} \propto \frac{A \cdot D \cdot (P_1 - P_2)}{T} \]

where \( V_{\text{gas}} \) is the amount of gas that diffuses from one point to another, \( A \) is surface area, \( D \) is diffusion constants, \( P_1 - P_2 \) is the difference in partial pressure between two points, and \( T \) is thickness.

The law states that the rate of gas transfer across a sheet of tissue is directly proportional to the surface area of the tissue, to the diffusion constants, and to the difference in partial pressure of the gas between the two sides of the tissue and is inversely proportional to the thickness of the tissue (Figure 3-7).

The diffusion constants (\( D \)) noted in Fick's law are determined by Henry's law and Graham's law.

HENRY'S LAW
Henry's law states that the amount of a gas that dissolves in a liquid at a given temperature is proportional to the partial pressure of the gas. The amount of gas that can be dissolved by 1 ml of a given liquid at standard pressure (760 mm Hg) and specified temperature is known as the solubility coefficient of the liquid. At 37°C and 760 mm Hg pressure, the solubility coefficient of oxygen is 0.0244 ml/mm Hg/ml H₂O. The solubility coefficient of carbon dioxide is 0.592 ml/mm Hg/ml H₂O. The solubility coefficient varies inversely with temperature (i.e., if the temperature rises the solubility coefficient decreases in value).

On the basis of the solubility coefficients of oxygen and carbon dioxide, it can be seen that in a liquid medium (e.g., alveolar-capillary membrane) carbon dioxide is more soluble than oxygen:

\[
\begin{align*}
\text{Solubility CO}_2 &= 0.592 \\
\text{Solubility O}_2 &= 0.0244
\end{align*}
\]

GRAHAM'S LAW
Graham's law states that the rate of diffusion of a gas through a liquid is (1) directly proportional to the solubility coefficient of the gas and (2) inversely proportional to the square root of the gram-molecular weight (GMW) of the gas. In comparing the relative rates of diffusion to oxygen (GMW = 32) and carbon dioxide (GMW = 44), it can be seen that, since oxygen is the lighter gas, it moves faster than carbon dioxide:

\[
\begin{align*}
\text{Diffusion rate for CO}_2 &= \frac{\sqrt{\text{GMW O}_2}}{\sqrt{\text{GMW CO}_2}} = \frac{\sqrt{32}}{\sqrt{44}} \\
\text{Diffusion rate for O}_2 &= \frac{\sqrt{\text{GMW O}_2}}{\sqrt{\text{GMW CO}_2}} = \frac{5.6}{6.6}
\end{align*}
\]

By combining Graham's and Henry's laws, it can be said that the rates of diffusion of two gases are directly proportional to the ratio of their solubility coefficients, and inversely proportional to the ratio of their gram-molecular weights. For example, when the two laws are used to determine the relative rates of diffusion of carbon dioxide and oxygen, it can be seen that carbon dioxide diffuses about 20 times faster than oxygen.

\[
\begin{align*}
\text{Diffusion rate for CO}_2 &= \frac{5.6 \times 0.592}{6.6 \times 0.0244} = \frac{20}{1} \\
\text{Diffusion rate for O}_2 &= \frac{5.6 \times 0.592}{6.6 \times 0.0244} = \frac{20}{1}
\end{align*}
\]

To summarize, the diffusion constant (\( D \)) for a particular gas is directly proportional to the solubility coefficients (\( S \)) of the gas, and inversely proportional to the square root of the gram-molecular weight (GMW) of the gas:

\[ D = \frac{S}{\sqrt{\text{GMW}}} \]

Mathematically, by substituting the diffusion constants,

\[ D = \frac{S}{\sqrt{\text{GMW}}} \]

into Fick's law:

\[ V_{\text{gas}} \propto \frac{A \cdot D \cdot (P_1 - P_2)}{T} \]
Diffusion Capacity of CO

CO has 210 greater affinity for Hb than O2.

Gases that combine with Hb do not exert a significant partial pressure.

Normal is 25 ml / minute / mm Hg.
Normal is 25 ml / minute / mm Hg
Diffusion Capacity of CO

After inspiring a gas mixture containing CO the alveolar CO fraction or pressure decreases with time as CO diffuses in.

If alveolar CO fraction is known at the beginning and at the end of a time interval it is possible to calculate the decay.

**Single Breath method**

Patient is breathing via a 2 way valve system.

After maximal Expiration the subject is asked to inspire deeply a gas mixture of 0.3% CO and 5 – 10 % Helium.

Flows are measured with a flow transducer.

After a breath hold of 10 seconds at total lung capacity the subject exhales.

The first 750 mls are discarded.

Alveolar gas sample is the next 750 mls is analysed.
Pleural Effusions

- Approximately 3.5ml/Kg of fluid is produced and absorbed daily
- Only 0.1 - 0.3 ml/Kg is present normally in space
- Symptoms usually when there is 300 – 500 mls present
- 175mls blunts Costo-phrenic angles
- Classified in to either Transudates or Exudates
  - Transudates altered Osmotic Hydrostatic pressures
  - Exudates altered pleural membranes or Lymphatics
- Analysis of Pleural Fluid
  - pH
  - Gram Stain, Culture
  - Protein
  - LDH
  - Glucose
  - Amylase
  - Cytology

By Definition Exudate = > 3g / dl (30g/L)
Pleural Effusions

- Net hydrostatic pressure moving fluid from parietal pleura into pleural space = Sys. Cap. Pres. + negative pl pres = 30 + 5 = 35 (out)
- Opposing this is colloid osmotic pressure of blood – colloid pressure in pleural fluid = 34-8=26 (in pleural space)
- Net is 9 cm H2O favouring fluid out of parietal fluid into pleural space
- Opposing this is Visceral Pleura production and absorption of fluid:
  - Net hydrostatic pressure moving fluid from visceral pleura into pleural space = Pul. Cap. Pres. + negative pl pres = 11 + 5 = 16 (out)
  - Opposing this is colloid osmotic pressure of blood – colloid pressure in pleural fluid = 34-8=26 (in) (same as parietal pleura)
- Net is 10 cm H2O favouring absorption of pleural fluid in to visceral pleura
- Hence fluid flows from systemic capillaries in parietal pleura, into pleural space, into pulmonary capillaries in visceral pleura (5–10L)

This is a contentious theory! 30 and 11 are at arterial end what about venous end of Capillary? You should average whole systemic capillary at 17.5 and Pulmonary capillary at 7 mmHg
Pleural Effusions

- Normal pleural fluid has protein content of 1.5 – 3 g/100 ml

Transudate
- Systemic factors influence absorption and formation of fluid
- Decrease in plasma colloid osmotic pressure
- Increase in Pulmonary Hydrostatic pressure
- Increase in Systemic Hydrostatic pressure

Exudate
- Disease of the pleural surface or involving lymphatics
- Increase in permeability of capillaries to proteins, Rheumatoid, Wegeners
- Causes include infection, Neoplasia, Haemothorax, Chylothorax

If fluid has any of these three criteria then it is called Exudate if none Transudate
- Pleural fluid Protein / Serum Protein = > 0.5
- Pleural fluid LDH / Serum LDH = > 0.6
- Pleural fluid LDH > 2/3 \textsuperscript{rd} normal serum LDH

Not so useful anymore since relates to sensitivity and specificity
- 43% of exudates = Malignancy
- 83% of Transudates = CCF

By Definition Exudate = > 3g / dl (30g/L)
Management Algorithm for Malignant Pleural Effusions

1. Proven malignant effusion
   - Recurrence/symptomatic?
     - NO: Observe
     - YES: Seek specialist opinion from a member of the thoracic malignancy multidisciplinary team
       - Intercostal tube insertion and drainage
         - Chest radiograph: complete lung re-expansion?
           - NO: Consider:
             - 1. Thoracoscopy (section 4.4)
             - 2. Long term indwelling catheter (section 4.5)
             - 3. Pleuroperitoneal shunt (section 4.6)
           - YES: Chemical pleurodesis
             - Recurrence of effusion?
               - YES: Consider:
                 - 1. Repeated pleurodesis (section 4.3)
                 - 2. Thoracoscopy (section 4.4)
                 - 3. Long term indwelling catheter (section 4.5)
                 - 4. Pleuroperitoneal shunt (section 4.6)
                 - 5. Palliative repeated thoracentesis (section 4.2)
               - NO: STOP
         - YES: STOP
Starling Fluid Forces – Oedema

Starling averaged the whole capillary pressures to a mean of 17.3 mmHg and found that a near equilibrium exists with a slight favour in ultrafiltration.

Causes of Oedema: Increase in capillary pressure, interstitial colloid osmotic pressure or decrease in plasma colloid pressure.
Starling Fluid Forces – Pulmonary Oedema

- Note pulmonary capillary pressure is low 7 mm Hg comparing with systemic capillary pressure of 17 mmHg
- The pulmonary capillaries are more leaky to proteins so that colloid osmotic pressure of pulmonary int. is high
- The interstitial fluid pressure is more negative than in peripheral interstitium
  - Can be looked at promoting Oedema but this is essential at keeping Alveoli dry
  - If Interstitial fluid pressure becomes even slightly positive alveoli will become flooded with fluid
  - Alveolar walls are extremely thin and will rupture by any positive interstitial pressure
- Causes of Pulmonary Oedema
  - Left-sided heart failure or mitral valve disease with consequent increase in pulmonary capillary pressures
  - Damage to pulmonary capillary membrane infection
two types of abnormalities:

I. Increased Capillary Pressure
   A. Excessive kidney retention of salt and water
      1. Acute or chronic kidney failure
      2. Mineralocorticoid excess
   B. High venous pressure
      1. Heart failure
      2. Venous obstruction
   C. Failure of venous pumps
      (a) Paralysis of muscles
      (b) Immobilized parts of body
      (c) Failure of venous valves
   C. Decreased arteriolar resistance
      1. Excessive body heat
      2. Insufficiency of sympathetic nervous system
      3. Vasodilator drugs

II. Decreased Plasma Proteins
    A. Loss of proteins in urine (nephrotic syndrome)
    B. Loss of protein from denuded skin areas
       1. Burns
       2. Wounds
    C. Failure to produce proteins
       1. Liver disease
       2. Serious protein or caloric malnutrition

III. Increased Capillary Permeability
    A. Immune reactions that cause release of histamine and other immune products
    B. Toxins
    C. Bacterial infections
    D. Vitamin deficiency, especially vitamin C
    E. Prolonged ischemia
    F. Burns

IV. Blockage of Lymph Return
    A. Cancer
    B. Infections (e.g., filaria nematodes)
    C. Surgery
    D. Congenital absence or abnormality of lymphatic vessels
What I think is happening!

Note Averaged Hydrostatic pressures are higher on systemic side, more negative interstitial fluid pressure and higher oncotic pressure of interstitial fluid on pulmonary side.

Net out is 0.3

However: Visceral pleura is relatively impermeable so current theory is that pleural fluid is predominantly produced and re-absorbed at the Parietal Pleura level.

Net Out is 1

Pleural Space itself has a negative pressure of 5 therefore efflux of fluid from both sides of pleural space into pleural space.
Control of ventilation

Medulla & Pontine respiratory centres receive information from Central & Peripheral Chemoreceptors

In medulla dorsal and ventral respiratory groups

- Dorsal group inspiratory exhalation is then passive – involved in passive quiet breathing
- Ventral only involved in strenuous exercise

Pontine centres

- Apneustic
- Pneumotaxic
- Function in man not really clear

Central chemoreceptors are located in the medulla and are sensitive to H+ changes in the CSF

- CO2 sensitive
- Blood-brain barrier is only permeable to CO2 not H+
- As CO2 rises (eg hypoventilation) CO2 diffuses into brain and makes H+
- Increase in H+ is not buffered therefore pH is reduced and ventilation is stimulated

Peripheral Chemoreceptors

- Carotid and Aortic Bodies and are O2 sensitive
- Activated when PO2 < 60 mmHg (Sats 90%)
- Chronically elevated CO2 turns off CSF sensitivity and control of ventilation
- These patients therefore rely of the hypoxic drive from peripheral chemoreceptors
Pulmonary Sequestration

- Congenital malformation embryonic lung tissue that derives its blood supply from an anomalous systemic Artery

- Two different forms Intra-lobar and extra-lobar very different presentations & management

- Accessory lung bud forms caudal to normal lung if early before pleura has formed then intra-lobar if after extra-lobar

**Extra-lobar**
- Very rare, mainly boys
- Associated with other congenital abnormalities in 50% of cases as well as diaphragmatic hernias
- L > R with total pleural covering
- Systemic artery as well as systemic venous drainage
- No Bronchial communication
- Presents in neonate with IRDS with triangular wedge opacity

**Intra-lobar**
- Still rare, but more common than extra-lobar, Girls = boys
- Not Associated with other congenital abnormalities
- L > R within lobe
- Systemic artery but usually normal venous drainage in to left atrium
- Occasional Bronchial communication
- Presents in adolescent young adult with cough, fever, sputum, possibly abscess

Congenital malformation embryonic lung tissue deriving blood supply from anomalous systemic Artery
Pulmonary Sequestration

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**Extra-lobar**
- Very rare, mainly boys
- Associated with other congenital abnormalities in 50 % of cases as well as diaphragmatic hernias
- L > R with total pleural covering
- Systemic artery as well as systemic venous drainage. If right sided blood supply from abdominal aorta below diaphragm
- No Bronchial communication
- Presents in neonate with IRDS with triangular wedge opacity. 15% are asymptomatic. Remove it but beware of sys. artery

**Intra-lobar**
- Still rare, but more common than extra-lobar, Girls = boys
- Not Associated with other congenital abnormalities
- L > R within lobe. Often indistinguishable from pyogenic abscess or bronchiectasis. (Abscess in upper lobe latter in L. lobe)
- Systemic artery but usually normal venous drainage in to left atrium
- Usually present Bronchial communication hence propensity for getting infections
- Presents in adolescent young adult with cough, fever, sputum, possibly abscess

Congenital malformation embryonic lung tissue deriving blood supply from anomalous systemic Artery
Pulmonary Sequestration

Presentation
- Extra-lobar presents in the neonate perhaps at time of having diaphragmatic hernia repair or if large IRDS
- Intra-lobar because of bronchial communication presents in the young adult with recurrent chest infections
- Intra-lobar sometimes indistinguishable from a lung abscess or bronchiectasis
- Aspiration type lung abscess usually occurs in upper lobes sequestrated lung almost occurs in the lower lobes
- High output failure due to large systemic blood from anomalous artery draining into pulmonary circulation

Investigations
- Bronchoscopy usually of little help but required
- Evaluate Upper GI tract to look for foregut communication
- CT
- Aortogram has been advocated by some

Treatment
- Resection for both
- For intra-lobar bearing in mind its inflammatory nature do it in quiescent phase of disease
- Beware of systemic artery

Congenital malformation embryonic lung tissue deriving blood supply from anomalous systemic Artery
Pulmonary Sequestration

Congenital malformation embryonic lung tissue deriving blood supply from anomalous systemic Artery
Congenital malformation of pulmonary vasculature in which there is a persistent communication that bypasses the pulmonary capillary bed
SVC Syndrome

- Aetiology has changed from TB, Aortic Syphilis
- Divide them into Extrinsic compression, Direct Invasion or thrombosis
- Now Mostly Malignant (lung cancer 70 – 80%)
- Nodal compression from Small cell Cancer being common (30%) of small cell (70%) in NSCC
- Other tumours of Mediastinum, Lymphomas, Thymoma, Substernal Goitres
- Thrombosis from lines (Note thrombosis is associated with SVC obstruction)
- Types I – IV described with relation to patency and direction of flow in the Azygous vein

**Symptoms**
- Dyspnoea, Cough, facial or arm swelling

**Signs**
- Oedema, prominent venous pattern of head, neck, arms, with cyanosis and plethora, Horners, Vocal cord probs

**Diagnosis**
- CXRAY – Anterior mediastinal mass
- CT – With contrast, Mass, venous collaterals
- MRI – For vascular structures
- Serum markers for germ cell tumours
- Tissue Diagnosis
- Venogram
SVC Syndrome

Management

Medical
- Elevation of head, diuretics, Oxygen, Steroids, Anticoagulation if venous thrombosis
- Consider thrombolytic therapy if fresh line thrombus

Radiotherapy
- For NSCC as well as Lymphomas

Chemotherapy
- Lymphoma or SCCC

Interventional Radiology
- Stents

Surgery
- Bypass for benign disease with spiral venous autograft
**Stridor**

- **Stridor is inspiratory wheeze and relates to large airway obstruction**
  - Larynx, Trachea, Hypo-Pharynx

- **Wheeze usually described in the expiratory phase and relates to smaller airways**
Solitary Pulmonary Nodule (SPN)

- Lesion measuring < 3cm in maximum diameter surrounded by lung parenchyma
- If it is a cancer it is T1NO = Stage IA with post-resection 5 year survival of 75% (CANCER UNTIL PROVEN OTHERWISE)

Early Lung Cancer Action Project
- 1000 symptom free smokers > 60 years of age
- 68/1000 on CXRAY found to have a pulmonary nodule 7/68 of these nodules were malignant (10%)
- 233/1000 on CT found to have non-calcified nodules 27/233 of these nodules were malignant (10%)
- Concludes that approximately 10% of such solitary nodules are malignant and therefore not to subject 90% of pts to unnecessary operations

General Approach
- Previous CXRAY comparison is vital
- If on previous CXRAY same nodule is seen just observe
- Add sputum cytology to look for any malignancy

How long to observe for? Quaterman Study
- No difference in survivorship in patients with early stage NSCLC if resected within 90 days of diagnosis Vs After 90 days
- Hence rational for 3/12 (90days) interval CT

Position of SPN
- If centrally placed in lobe would probably require Lobectomy
- If peripherally located may be removed by wedge resection
- Therefore Lobectomy for a potentially benign lesion is more egregious

New SPN in a middle-aged smoker is a primary lung cancer until proven otherwise
Chances of SPN being a cancer is approximately the patients age
SPN Differential Diagnosis (link with History taking)

- **Inflammatory**
  - Granulomas from prior infection, TB, histoplasmosis (History of previous TB, recent travel, Pets)
  - Round Pneumonia
  - Wegener’s Granulomatosis, Sarcoidosis, Rheumatoid nodules (other evidence of joint involvement)

- **Benign tumours**
  - Lung Carcinoid
  - Hamartoma
  - Haemangioma

- **Malignant Tumour**
  - Primary Lung Cancer (Smoking history, Asbestos exposure, COPD)
  - Secondary lung metastasis (Prior history of malignancy colorectal, gastric..)
The Solitary Pulmonary Nodule (SPN)

**CXRAY**
- Often incidental finding on CXRAY
- Risk factors for malignancy
  - Smoking history
  - Previous Malignancy
  - Age > 35

**CT scan (5 Properties)**
- **Calcification**
  - Most commonly seen in benign lesions
  - Granulomas more central solid calcification
  - Hamartomas “popcorn calcification”
  - However 6 – 14 % of malignancies show calcification “stippled”
- **Edge Characteristics**
  - Spiculated margin with fine linear strands has a high (up to 94%) positive predictive value for malignancy
  - However smooth borders are seen in up to 20% of malignancies
- **Internal Characteristics**
  - Size of nodule; > 2 cm majority are malignant; < 2 cm 50% malignant
  - Cavitation is most commonly seen in malignancy – wall thickness in this setting is important >15mm wall thickness 84% malignancy
  - Nodules displaying fat are most likely benign, most usually Hamartomas
- **Enhancement**
  - Enhancement < 15 Hounsfield units (HU) with contrast is strongly predictive of Benign lesions
  - Enhancement > 20 – 60 (HU) with contrast is strongly predictive of Malignant lesions
  - False positives with central necrosis of benign tumours
- **Rates of Growth**
  - Doubling time for lung cancers between 20 – 400 days
  - Hence 2 year stability is equated with benign lesions

As a general Rule Malignant tumours are not calcified
The Solitary Pulmonary Nodule (SPN)

**PET (Positron Emission Tomography)**
- 18 fluorodeoxyglucose (18-FDG) is taken up by cells for glycolysis but cannot be utilised and accumulates
- Meta-analysis of 40 studies 2001
  - Sensitivity 97% Specificity of 78% for malignancy
  - Sensitivity 96% Specificity of 88% for Benign lesion
  - Limitations; resolution of 8mm hence less than 1 cm is unreliable
  - False negatives Carcinoïd, broncho-alveolar cancers, and Adenocarcinomas (Birmingham results)
  - False positives seen in infection and inflammatory lesions

**Bronchoscopy**
- “Bronchus Sign” on CT scan – fourth order bronchus or higher can be seen within or leading to nodule
- Diagnostic yield 60% if bronchus sign present Vs only 18% if not present
- Can be aided by Fluoroscopic guidance

**CT guided Biopsies**
- Sensitivity ranges between 60 – 100 % according to which series
- Feasible even in 1cm nodules
- <0.8cm unlikely
The Solitary Pulmonary Nodule (SPN)

Figure 1. Flow diagram to show the diagnostic pathway for a solitary pulmonary nodule.
# The Solitary Pulmonary Nodule (SPN)

## Recommendations

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Evidence level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with a solitary pulmonary nodule should have a CT scan to characterise the nodule. Assessment of size, calcification, edge and internal characteristics can be useful in identifying nodules that are more likely to be benign. CT also plays an important role in staging.</td>
<td>IIb/B</td>
</tr>
<tr>
<td>Benign or indeterminate nodules should be followed-up with a repeat CT scan and volumetric assessment. An increase in volume should prompt further investigation or excision.</td>
<td>IIb/B</td>
</tr>
<tr>
<td>PET scanning should not be used in the assessment of solitary pulmonary nodules &lt;1cm diameter. PET scanning may provide additional information in nodules thought to be at a low-risk of malignancy. In nodules at a higher risk of malignancy, a definitive tissue diagnosis will be needed.</td>
<td>Ib/A</td>
</tr>
<tr>
<td>Bronchoscopy is of value in obtaining a tissue diagnosis in larger lesions that are centrally placed and have a “bronchus sign”. Multiple sampling techniques including washings, brushings, biopsy and needle aspiration should be used.</td>
<td>IIb/B</td>
</tr>
<tr>
<td>Transthoracic needle biopsy provides the best means to obtain a tissue sample for an indeterminate solitary pulmonary nodule.</td>
<td>IIb/B</td>
</tr>
</tbody>
</table>
The Solitary Pulmonary Nodule (SPN)

Figure 1. Images of Solitary Pulmonary Nodules.

The CT scan in Panel A shows a nodule with a corona radiata. Multiple fine striations extend perpendicularly to the surface of the nodule, which is surrounded by a radiolucent halo formed by emphysematous lung tissue. The diagnosis was non-small-cell lung cancer. The CT scan in Panel B shows a spiculated lesion that is highly suggestive of cancer; the diagnosis was adenocarcinoma. In Panel C, the CT scan shows a lesion with a scalloped border, a finding associated with an intermediate probability of cancer. In Panel D, the radiograph shows a smooth, well-marginated nodule in the left lung with dense central calcification (left), a pattern typical of hamartomas; a resected specimen helped confirm the presence of central calcification and laminated fibrous tissue (right). The CT scan in Panel E shows a well-demarcated lesion with both calcification and fat, findings that are typical of a hamartoma. The patient was asymptomatic. In Panel F, the CT scan shows an air-bronchus sign, which suggests that bronchoscopy will have a high yield. The diagnosis was bronchoalveolar carcinoma.
Thoracic Trauma – Tracheal

- Blunt injury is rare deceleration, hyper-extension type injuries
- Blunt injuries may involve Larynx, cervical trachea, mediastinal trachea, intrathoracic bronchi
- Trachea protected by SCM laterally, C spine posteriorly, Mandible anteriorly (not when neck extended)
- Membranous tears can be seen
- Carina and Cricoid cartilage are fixed
- 80% of tears are located 2 – 3 cm from trachea
- Massive surgical Emphysema
- After placement of ICD for Pneumothorax failure of lung to expand and brisk airleak may indicate tracheobronchial inj.
- Note if significant air leak suction on chest drain may exacerbate the patients condition

Management

- Will be dictated by patients condition follow ATLS ABC
- Establish and protect airway is paramount, Xrays later
- Fibreoptic Bronchoscopy is essential to guide intubation beyond a disrupted airway
- State of Larynx will dictate whether to attempt Oro-tracheal intubation or to go for Surgical airway, Cricothyrotomy may save the day
- Cricothyrotomy is contraindicated however in setting of cricoid cartilage injury and is difficult in a neck with haematoma & emphysema
- Steroids to reduce oedema

Surgery

- Injuries to > 1/3rd of tracheal circumference should be repaired surgically to avoid later stenosis
- Collar incision for cervical Trachea 1-2 cm above jugular notch and flaps developed laterally and superiorly
- Mediastinal trachea and bronchus best approaches through a postero-lateral thoracotomy
- Trachea, Carina, RMB, proximal left main stem Bronchus best through right chest
- Single Lumen ET tube passed through tare under direct vision
- Intercostal muscle pedicle in chest, Strap muscles in cervical region
- Care not to mobilise trachea too much & should be performed in AP plane to avoid lateral vascular blood supply
- Bronchoscopy for clearance of secretions
Thoracic Trauma – Tracheal
Thoracic Trauma

- Pneumomediastinum
  - Respiratory tract, larynx, hypopharynx, trachea, main stem bronchi
  - Oesophagus

- Can be post-trauma or Spontaneous

- Half of childhood pneumothoracic, pneumomediastinum, are spontaneous coughing etc..

- Symptoms / Signs
  - Substernal pleuritic chest pain radiating to back, dyspnea, cough
  - High pitch voice
  - ‘Hamman’s crunch “crepitant sound heard at auscultation that varies with heart beat resulting from air around/in pericardium

- In children immaturity of fascial planes allows dissection in to pericardium pneumo-pericardium

- Investigations
  - CXRAY – V sign of Boerhaave, Sail sign of Thymus, Fallen lung
  - CT, Neck, Chest, abdomen, jubicious oral contrast
  - Triple endoscopy, Laryngoscopy, Bronchoscopy, Oesophagoscopy (Latter sometimes makes things worse!)
  - OGD should be only done intraoperatively
  - Gastrograffin and Barium swallows

- Treatment
  - In a ventilated patient may result in tension Pneumothorax therefore if you see small Pneumothorax drain immediately
  - Treat Oesophageal tears see Oesophageal perforation slide
  - Tracheobronchial rupture
    - When greater than 1/3rd of circumference of airway, unresolving pneumothorax, worsening Pneumomediastinum then surgery
Thoracic Trauma

Flail Chest
- A segment of chest wall that moves independently from the rest of the chest wall as a result of multiple ribs being fractured in at least 2 places
- Mechanics of respiration are altered paradox during inspiration (If patient is ventilated this may not be apparent therefore palpate)
- Force the caused it is severe therefore look for other injuries

Treatment (Pain Relief and Pulmonary support)
- Pain management is paramount
- Pain reduces tidal volume and ability to cough, atelectasis, hypoxia, and later Pneumonia develop
- Oral Narcotics
- If no other spinal trauma consider thoracic epidural
- Look for Po2 and Pco2 if Hypoxia or Hypercarbia then PPV

Consider early Tracheostomy if likely to be ventilated for a long time

Pulmonary Contusion
- After Blunt of penetrating trauma, patchy opacity which blossoms over next several days
- If Isolated treatment is conservative with pulmonary toilet, incentive spirometry
- Strict attention to volume resuscitation CVP, Swan-Ganz etc.
Blunt injuries to the heart are associated with **stenal fractures** in 25% of cases.

**Gibson 1962** 80 patients with fractured sternum 11 year series
- 19/80 deaths (24%) most from head injuries
- Concluded that the heart was relatively spared of any injuries

Diagnosing sternal fracture depends on lateral CXRAY.

Association between sternal fractures and Myocardial Contusion depends on definition of latter.

Surgeon treating a patient with anterior chest trauma asks 7 questions:
- 1. Is the patient haemodynamically stable
- 2. Does patient have cardiac muscle dyskinesia, Coronary artery insufficiency, hemothorax, VSD, valve tear
- 3. What test to do in stable and unstable patient
- 4. What are the associated injuries
- 5. Do they need to be hospitalised for monitoring
- 6. Is there any bony lesions that require fixation
- 7. If there are timing of fixation

**Scoring of Blunt Cardiac Injury**
- 1 Point for each of No ECG abnormality, minor, major ECG abnormality, cardiac enzyme rise
- 2 Points for each of free wall hematoma and septal hematoma
- 4 Points for each of VSD, Valvular insufficiency
- 5 Points for each of free wall rupture, Coronary artery injury, cardiac herniation
Thoracic Trauma – Blunt Cardiac Trauma

Investigations

- As per ATLS guidelines with Primary and secondary surveys
  - Lateral CXRAY is part of secondary survey
  - ECG changes are very non-specific
- CK-MB is found in skeletal muscle so raised levels are not diagnostic of cardiac contusion
- FAST (Focused Abdominal Sonogram Trauma) and, TTE, TEE

Treatment

- Sternal fractures – USA say fix for pain relief and cosmesis
- BCT with abnormal ECG – Admit and Monitor treat arrhythmias
- Cardiac Rupture
  - Haemopericardium detected by FAST treat with surgery (? Anterolateral thoracotomy)
  - RA and RV most commonly affected but very rare to need surgery for blunt trauma to heart
- VSD
  - Operate on CPB if significant
- Valvular insufficiency
  - Operate on
- Pericardial tear with herniation
  - Operate on

Results

- Mortality / Morbidity after BCT with blunt cardiac manifestation such as ECG changes as well as enzymes rise is extremely low
- Mortality and morbidity after septal, free wall, valvular lesions are very high up to 50%
Thoracic Trauma - Haemothorax

- Laceration of lung parenchyma or chest wall vessels (intercostal of Internal mammary artery)

- In 90% of blunt and 80% of penetrating injuries ICD placement is the only management required

- Large ICD 32 – 40 Fr MAL 5th ICS with CXRAY after t to check that cessation of blood loss isn’t due to kinked drain

- Eastern Association for surgery of trauma group recommend antibiotics for 24 hours following ICD in trauma

- If you drain immediately 1500 mls of blood or if loss is > 200mls/hr for 2-4hrs after insertion is an indication for surgery

Residual Haemothorax

- If CXRAY suggest on day three following trauma that there may be a retained haemothorax then CT is advised

- If CT evidence that there is retained Haemothorax then VATs is superior to thoracotomy

Treatment

- If from Pulmonary Parenchyma then either Oversew, wedge resection or anatomical resection

- Tractotomy is when the stapling device is placed through the tract of a bullet and stapled revealing the bleeding points for the surgeon to then over-sew.

- Tractotomy avoids resection of any lung tissue
Thoracic Trauma – Diaphragmatic Injuries

- Blunt or penetrating account for 95% of injuries
- Other causes: Operative, effort-related etc.
- Blunt injuries are usually result of deceleration type injuries when excessive pressure on inflexible central tendon
- 95% of injuries occur on the left, if on right more severe general injuries
- Suspect penetrating injuries if entry wound is below 4th ICS to Umbilicus
- Can also be classified according to timing: Acute, Latent, Obstructive phases
  - Acute - from time of injury to recovery of other injuries. Symptoms are acute, SOB, Pain etc.
  - Latent – Compensatory period when patient adjusts to abdominal contents being in the chest, variable symptoms
  - Obstructive – Ranges from 20 days to 28 years. Obstructive symptoms
- Grading – 1–5, 1 being contusion only, 2, small < 2cm laceration to 5 which is > 25 cm² tissue loss
- Diagnosis can be missed in up to 66% of cases with serious late consequences
  - CXRAY – 75% non-diagnostic may show indistinct costo-phrenic angle, air fluid levels and NG tube going into intrathoracic stomach
  - Owing to difficulty in diagnosing particularly on R side some surgeons advocate mandatory Laparotomy for penetrating lower chest inj.
- Surgery mostly through abdomen in blunt injuries since they will commonly have intra-abdominal injuries
- Latent and Obstructive presentation some advocate better go through chest since they will have adhesions
Thoracic Outlet Syndrome

Clinical Presentation
- Nerve compression
  - Pain / parathesias along Ulnar nerve (T1)
  - Motor Weakness again T1 muscles of the hand
- Arterial Compression
  - Coldness, weakness, fatigability of hand
  - Raynoud’s disease
  - Arterial Occlusion
- Venous compression
  - Venous occlusion or obstruction is infrequent
  - Effort thrombosis (Paget-Schroetter syndrome) Oedema, venous distension
  - Motor Weakness again T1 muscles of the hand

Examination
- Physical manoeuvres (Adson, hyper-abduction, modified Roos test to reproduce symptoms

Investigations
- Clinical sensory testing
- EMG
- CXRAY for cervical rib
- CSPINE XRAY
- CT
- MRI

Differential Diagnosis
- Neurological disorders herniated Cervical disc. Brachial plexus, Pancoasts tumour, Peripheral nerve entrapment
- Vascular both arterial and venous
Thoracic Outlet Syndrome

Management

- Non-Operative
  - Patient education waffle
  - Physiotherapy
  - Subclavian vein thrombosis, thrombolytic therapy, compression garments

- Operative
  - Indicated when failed medical therapy (<5% of cases)
  - Trans-axillary approach for excision of first rib with division of Fibrous Band
  - Supra-clavicular approach
  - Posterior approach
Tracheo-Oesophageal Fistula

Congenital
- With or without Oesophageal atresia

Acquired
- Inflammatory / Infective
  - TB, Histoplasmosis
- Traumatic
- Post-intubation
  - ET tube and NG tube pincer
- Neoplastic
  - Tracheal tumours or Oesophageal tumours

Clinical Presentation
- Gastric Contents in airway or marked tracheal secretions
- In ventilated patient if Cuff of ET tube is above fistula gastric distension will occur
- Diagnosis by Bronchoscopy or Oesophagoscopy
- If Oesophageal tumour during Barium Swallow you may see Barium in airway (better than Gastrograffin)

Management
- Benign pathology (e.g. Intubated patient)
  - Conservative at first with Gastrostomy and Feeding Jejunostomy until patient is stable and extubated
  - Tracheostomy with cuff below fistula
  - Surgical repair is then tracheal repair / resection with Oesophageal defect closure (vascular pedicle inbetween suture lines)
- Malignant Pathology
  - Stent Oesophagus
  - Rarely diversion with extra-anatomic bypass

No Role For Initial Diversion Of Oesophagus in Early Management
Tracheo-Oesophageal Fistula

Basics of Preoperative Management

- Delay operative repair until patient is off ventilatory support
- Haemodynamically stabilise the patient
- Remove NG
- Place a new low pressure, high-volume (long) Tracheostomy with cuff below fistula
- Draining Gastrostomy
- Feeding Jejunostomy
- Nutrition, Hydration, antibiotics

No Role For Initial Diversion Of Oesophagus in Early Management
Trachea

- The blood supply to the trachea is segmental and comes into the trachea laterally.
- The lateral pedicles induct vessels from the:
  - Inferior thyroid
  - Subclavian
  - Supreme intercostal
  - Internal thoracic
  - Innominate
  - Superior and middle bronchial arteries
- In cervical region, inferior thyroid is the principle artery:
  - Supplies branches that run medially behind the lower part of the common carotid artery.
- The vessels are interconnected along the lateral surfaces of the trachea.
- From lateral longitudinal anastomoses:
  - The anterior and posterior walls of the trachea receive their blood supply.
  - Through transverse segmental vessels that run in the soft tissues between the cartilages.
- Resection of the trachea proximal and distal ends can safely be mobilized anteriorly and posteriorly.
- Can resect up to 50% of trachea without a release procedure.
- Most common Tracheal tumour is Adenoid Cystic Carcinoma (Cylindroma).
- Second most common is Squamous cell Carcinoma.
- Surgical resection followed by Radiotherapy is now advocated with post-operative Radiotherapy.
Tracheostomy

Indications

- Upper Airway Obstruction
  - Congenital
  - Trauma
  - Infection
  - Neoplasms
- Control of secretions
  - Following neurological injuries
  - Acute / Chronic Pulmonary disease
- Ventilatory Support
  - Weaning from ventilator
  - Guillain – Barre syndrome
- Innominate
- Superior and middle bronchial arteries

Tubes

- Shiley
  - Size 6 / 8 == 30 / 36 Fr = outside diameters of 10 / 12 mm inside diameters 7 / 8.5 mm
- Portex
  - Size 6 / 8 == 30 / 36 Fr = outside diameters of 10 / 12 mm inside diameters 7 / 9 mm

Inner tubes for cleaning

Fenestrated for talking

Cuffs should be large volume low pressure

Between 2\textsuperscript{nd} / 3\textsuperscript{rd} rings – above glottis problems, below TIF

Keep cuff pressure below 25 cm H2O – Pressure at which capillaries close
Tracheostomy

Shiley
Tracheal Stenosis

- **Acquired Intrinsic**
  - Post-intubation
  - Inflammatory
  - Idiopathic
  - Neoplastic

- **Acquired Extrinsic**
  - Goitres
  - Nodes
  - Vascular Rings

- **Mechanism**
  - Pressure from Cuff of ET tubes or Tracheostomy causes Ischaemia and healing by full thickness Fibrosis
  - Leverage of Tracheostomy tube is very important hence tubing set-up is very important

- **Presentation**
  - Suspect in any previously ventilated patient who shows signs of upper airway obstruction
  - Stridor (Inspiratory)

- **Diagnosis**
  - Bronchoscopy
  - CT for extrinsic compressive causes – Spiral CT reconstruction
  - Tomograms of Trachea with measurements and relations to Vocal cords and Carina

- **Indication for Surgery**
  - Depends on degree of symptoms
  - Failure of Dilatations or recurrence after Dilatations
  - For Extrinsic causes these need to be addressed
Tracheal Stenosis

- **Surgery**
  - Get them off ventilator first if on it
  - Treat infection
  - Get them off steroids

- **If present as an emergency consider**
  - Dilatation
  - Tracheostomy
  - T Tube (Montgomery)

- Preoperative assessment carefully with Bronchoscopy with all measurements

- Position patient neck hyper-extended collar cervical incision flaps developed

- **Apply Basic principles of Tracheal Surgery**
  - Keep close to trachea
  - Avoid lateral dissection
  - Resect the bare minimum

- **Post-operative**
  - Tracheostomy may be required
  - Watch for Stridor
  - Chin Stitch
  - Complications include the usual + Tracheo-Innominate fistula
Tracheal Stents

Indication for Stents
- Malignant Intrinsic Obstruction after airway patency has been re-established (Dilation, Laser)
- Airway Malignant Fistula
- Benign Intrinsic Stenosis not amenable to surgery
- Extrinsic Obstruction
- Tracheo-Malacia

Silicone vs. Metal
- Silicone must be placed with Rigid Bronchoscopes, migrate but can be easily removed even after long periods
- Metal can be placed with flexible Bronchoscopy, mould easily to tracheal wall, difficult to remove

Patients with Benign Pathology present Challenge
- Life expectancy is long
- Complications with stents are present and inherent in their use
- Limited use of metal stents because of difficulty in removing them

Most common Silicone stent is the Dumon stent

Montgomery Tube
- Sub-glottic or high tracheal obstructions or very long segment stenosis that can not be resected
- Proximal and distal ends can be cut to size to fit patient
- Leave enough distance to vocal cords to ensure good voice

Metal Stents
- Gianturco is thing of the past
- Most are made from metal alloys (Nickel or Titanium)
- Covered or uncovered
  - Covered with membrane to prevent tumour ingrowth into stent or use in fistulas
  - Uncovered cannot therefore be used in fistula controls
Vascular Rings

- Collection of congenital vascular anomalies that encircle and compress the Oesophagus and Trachea

- 1,2,5 Involute as part of normal development

- If development stops here a double arch is formed

- If the right 4th involutes a normal left arch is formed

- If the left 4th Involutes a right arch is formed

- Classic symptom of a child with a vascular ring is the “seal-bark” cough

- Other symptoms are Stridor, Asthma, wheeze, recurrent pneumonia, Dysphagia

- Barium swallow – PA sling is the only vascular ring that causes anterior compression with no posterior element of compression

- Bronchoscopy demostrates the classical tear drop compression of the Trachea

- Double arch causes both anterior and posterior compression
Vascular Rings

Figure 3
A, Pulmonary artery sling. RPA, right pulmonary artery; LPA, left pulmonary artery; MPA, main pulmonary artery. Inset shows anterior compression of esophagus. B, Repaired pulmonary artery sling. Left pulmonary artery has been anastomosed to main pulmonary artery anterior to trachea.
WHO Performance State

- **0** Normal activity without restriction
- **1** Strenuous activity restricted, light house work
- **2** Up and about > 50% of waking hours, all self care, no work
- **3** Bed / Chair bound > 50% of time, limited self care
- **4** Confined to Bed / Chair, no self care, completely disabled