

- 1) Aetiology of disease
  - I) Congenital: ASD, patent Ductus
  - II) Hereditary:
  - III) Metabolic:  
→ Gout (purine), Osteoporosis(calcium)
  - IV) Endocrine:  
→ Cushing syndrome, Acromegaly
  - V) Immunological:
  - VI) Degenerative:  
→ Atherosclerosis
  - VII) Neoplastic
  - VIII) Infective
  - IX) Iatrogenic (人造)
- 2) Ways of effect on the host by disease
  - a) **Derangement of functions**
    - I) Disturbance of internal environment  
→ homeostasis of the body fluid  
→ e.g. hypercalcaemia
    - II) Disturbance of physiological function  
→ Will gives rise to **symptoms**
  - b) **Derangement of structures**
    - ⇒ Derangement of normal anatomical structure of the body  
→ Will gives rise of **signs**
- 3) History taking
  - I) **Name, Age, Sex**
  - II) **Chief Complaint (主訴)**
  - III) **History of present illness (現病史)**
  - IV) **Past History, Family history, obstetric history(既往史, 家族史, 婚產史)**
  - V) **Physical examination**
    - 1) **Special signs**  
→ E.g. Finger Clubbing
    - 2) **Normal procedures**  
→ Cardiovascular, respiratory, CNS, Abdominal  
→ Musculo-skeletal, Urinalysis
  - VI) **Investigation**  
→ Function tests, imaging, blood and fluid tests

- 4) 4 Principles in clinical bedside diagnosis
- I) **Commence** with a process of exclusion and inclusion
  - II) **Symptoms**
    - Clinical manifestations of disturbance of internal environment of the body and normal physiological functions
  - III) **Signs**
    - Derangement of normal anatomical structure of body
  - IV) **Common** diseases always come commonly
- 5) Stigmata of liver disease
- I) **Jaundice**
    - Not at medial and lateral corners of sclera
  - II) **Spider Angioma \*\*\***
    - At superior vena cava drainage area
  - III) **Scattered telangiectasia \*\*\***
    - Dilated capillaries
  - IV) **5 signs of hand**
    - 1) **Palmer erythema**
    - 2) **Clubbing of fingers and toes**
    - 3) **Dupuytren's contracture**
      - Loss of tissue in the palm
      - First affects 4<sup>th</sup> & 5<sup>th</sup> tendons
    - 4) **White nails**
    - 5) **Flapping tremor**
      - Cannot hold the hand steadily
  - V) **Fetor hepaticus**
    - Special smell of breathe
    - Severe hepatocellular decompensation
  - VI) **Changes of body hair distribution**
  - VII) **Gynaecomastia**
  - VIII) **Ankle pigmentation + leg ulcers**
    - Associate with splenectomy
  - IX) **Signs of fluid retention**
  - X) **Easy bruising, purpura (皮下出血)**

6) Examination of the abdomen

a) **Inspection**

I) Shape

- 1) Scaphoid
- 2) Distended  
    → Fat, flatus(氣), faeces, fluid    fetus

II) Umbilicus

- 1) Depressed  
    → In fat abdomen
- 2) Bulging / everted  
    → Increased intra-abdominal pressure (Ascites)

III) Movement

- 1) Pulsation  
    → Thin patients
- 2) Visible peristalsis  
    → Intestinal obstruction

IV) Hernial orifices (疝氣)

→ ASK PERMISSION

b) **Palpation**

→ START FROM Lower Right Quarter

I) Gross abnormalities

II) Tenderness

III) Liver

- Upper border: 5<sup>th</sup> intercostal space + mid clavicular line
- Lower border: 7cm under costal margin + mid clavicular line
- Palpable ?
- Characteristic of edge and surface
- Sharp ? Nodular ? Hard ?

IV) Spleen

→ Along line joining UMBILICUS and Left anterior axillary fold

→ Enlarged spleen have notches \*\*\*

→ Method

- 1) Turn patients towards right
- 2) Hook spleen forward with hand at renal angle
- 3) Spleen may float to a more lateral position in presence of ascites

V) Kidneys

→ Right kidney lower than left

→ \*\*\*Differentiation between spleen and left kidney

- 1) Spleen is anterior, kidney bimanual palpation
- 2) Subcostal gap absent for spleen
- 3) Percussion dull for spleen
- 4) Notches for spleens > 10cm

c) Percussion

I) Ascites

- a) Shifting dullness at flanks
- b) Fluid thrill

d) Auscultation

I) Bowel sounds

II) Splashing

→ Pyloric stenosis

7) Way to measure arterial blood gas

→ Use a fine needle and syringe to take blood from artery (**RADIAL ARTERY**)

→ Normal range

→ PO<sub>2</sub>: 80-100mmHg

→ PCO<sub>2</sub>: 36-44mmHg

→ pH: 7.36 – 7.44

→ HCO<sub>3</sub>: 22-29

8) Gas exchange and transport

a) Background

→ Respiratory quotient = 0.8

→ Every 250ml of O<sub>2</sub> is used, 200ml of CO<sub>2</sub> is produced

b) Oxygen transport

I) Form

→ Majorly bind to hemoglobin (1.39ml/g of Hb)

→ 20mL O<sub>2</sub> will be carried by 15gram of Hb/100mL blood

→ S shape Hb-O<sub>2</sub> dissociation curve

→ Slight decrease in saturation at high PO<sub>2</sub>

→ Great decrease in saturation at low PO<sub>2</sub>

→ PO<sub>2</sub> 100mmHg (X axis) = 97% saturation (Y axis) = Artery

→ PO<sub>2</sub> 60mmHg = 90% saturation

→ PO<sub>2</sub> 40mmHg = 75% saturation = Venous

**II) Factors affecting the affinity of hemoglobin for O<sub>2</sub>**

- a) Shifting the curve to the right
  - Favors dissociate
    - 1) Decrease in pH
    - 2) Increase in PCO<sub>2</sub>, temp, 2,3 Diphosphoglycerate
- b) Shifting the curve to the left
  - Favors carrying, Tends not to dissociate
    - 1) Decrease in 2,3 Diphosphoglycerate
      - This chemical binds more readily to deoxy. RBC
    - 2) Increase in pH

**c) CO<sub>2</sub> transport**

**I) Form**

→ Majorly in form of HCO<sub>3</sub> (85%)

→ 5% in physical solution, 10% binds to Hb / plasma proteins

**II) Factors affecting the affinity of hemoglobin for O<sub>2</sub>**

→ Dissociation is usually linear over limited range b2 vein / artery  
→ Dissociation curve is steep

**9) Mechanisms of hypoxemia**

**I) Decreased of PiO<sub>2</sub>**

→ Lower atmospheric pressure with normal fraction of inspired O<sub>2</sub>  
→ High altitude

**II) Alveolar hypoventilation**

- 1) Reduced respiratory drive
- 2) Diseases of respiratory muscles
  - E.g. motor neuron disease, cervical spine injury
- 3) Disease of chest wall or rib cage
- 4) Disease of upper airways
- 5) COPD

**III) Ventilation-perfusion mismatching**

→ Asthma, COPD, pulmonary embolism  
→ Acute respiratory distress syndrome

**IV) Shunt**

→ Right-to-left shunt in congenital heart disease

**V) Diffusion defect**

## 10) Respiratory failure

- a) **Type 1 respiratory failure / oxygenation failure**
  - ➔ \*\*Hypoxemia predominance, NO retention of CO<sub>2</sub>
    - Defined by PO<sub>2</sub> < 8kPa (Normally 10-13kPa)
  - ➔ Due to
    - 1) Gas exchange units defect
      - Severe pneumonia
    - 2) Distal conducting airways defect
      - Asthma
    - 3) Pulmonary vasculature defect
      - Pulmonary Embolism
- b) **Type 2 respiratory failure / ventilation failure**
  - ➔ \*\*Hypercarbia predominance + hypoxemia
    - PCO<sub>2</sub>> 6.5kPa, PO<sub>2</sub>< 8kPa (Normally PCO<sub>2</sub> 4.5-6.0kPa)
  - ➔ Due to
    - 5 reasons causing hypoventilation (9, II) \*\*

## 11) Acid base balance

- ➔ Closely interrelated to respiratory status
- ➔ **Equation:** CO<sub>2</sub> + H<sub>2</sub>O ⇌ H<sub>2</sub>CO<sub>3</sub> ⇌ H<sup>+</sup> + HCO<sub>3</sub><sup>-</sup>

- a) 2 Types of acids in body
  - I) Carbonic acid
    - ➔ Can be effectively removed by the lung as CO<sub>2</sub>
    - ➔ Measured by PaCO<sub>2</sub>
  - II) Other Metabolic acids
    - ➔ Excreted by kidney
    - ➔ Measured by HCO<sub>3</sub>
- b) Type of diseases
  - I) **Respiratory acidosis**
    - ➔ Due to respiratory failure (COPD / SEVERE ASTHMA)
    - ➔ Increase in PCO<sub>2</sub>, drives the equation to the right, increase H<sup>+</sup>
    - ➔ Compensated by
      - Kidney excrete H<sup>+</sup> and increase HCO<sub>3</sub> by renal retention

- II) **Metabolic acidosis**
  - ➔ Due to renal failure, lactic acidosis, diabetic ketoacidosis
  - ➔ Increased H<sup>+</sup>, drives equation to left, consuming more and thus lowering HCO<sub>3</sub>
  - ➔ Compensated by
    - Increased ventilation to get rid of more CO<sub>2</sub>, **restoring HCO<sub>3</sub>- / PCO<sub>2</sub> ratio**
- III) **Respiratory alkalosis**
  - ➔ Due to hyperventilation in early asthma, pulmonary embolism
  - ➔ Decreased in pCO<sub>2</sub> **drives the equation to the left**, decrease H<sup>+</sup>
  - ➔ Compensated by
    - Decreased HCO<sub>3</sub>- by retention, **restore HCO<sub>3</sub>- / PCO<sub>2</sub> ratio**
- IV) **Metabolic alkalosis**
  - ➔ Due to vomiting, retention of HCO<sub>3</sub> associated with diuretic therapy
  - ➔ **Decrease in H<sup>+</sup> drives the equation to right**, increasing HCO<sub>3</sub>
  - ➔ Compensated by
    - Decrease in ventilation to increase pCO<sub>2</sub>, **restoring HCO<sub>3</sub>- / PCO<sub>2</sub> ratio**

## 12) Physical examination of the chest

### a) Normal clinical pulmonary anatomy and physiology

- I) **Angle of Louis**
  - ➔ 2<sup>nd</sup> intercostal space
- II) **Horizontal fissure**
  - ➔ 4<sup>th</sup> intercostal space
  - ➔ Separate the right middle lobe and right upper lobe of lung
- III) **Right Oblique/Greater fissure**
  - ➔ A curve, starts at spinous of T4,
  - ➔ Cross 5<sup>th</sup> intercostal space laterally
  - ➔ Follows the contour of 6<sup>th</sup> intercostal space
  - ➔ Right superior part separate right upper lobe and lower lobe
  - ➔ Right inferior part separate right middle lobe and lower lobe
- IV) **Left Oblique/Greater fissure**
  - ➔ A curve, starts at spinous of T3/T4,
  - ➔ Cross 5<sup>th</sup> intercostal space laterally
  - ➔ Follows the contour of 6<sup>th</sup> intercostal space
  - ➔ Separate left upper and lower lobe

**V) Mediastinal outline structures**

- 1) Superior vena cava  
→ Right upper mediastinum
- 2) Right Atrium  
→ Right middle mediastinum
- 3) Inferior vena cava  
→ Right lower mediastinum
- 4) Aortic Arch  
→ Left upper mediastinum
- 5) Pulmonary trunk  
→ Left upper mediastinum
- 6) Left ventricle  
→ Left lower mediastinum
- 7) Cardiac apex  
→ 5<sup>th</sup> intercostal space ----- left mid-clavicular line

**VI) Others points**

- I) Left lung is smaller under X-ray  
→ Heart is there
- II) The medial parts of anterior rib is not seen under X-ray  
→ Made of Cartilage
- III) **PaO<sub>2</sub>** = partial pressure of oxygen in the arterial blood
- IV) **PaCO<sub>2</sub>** = partial pressure of CO<sub>2</sub> in arterial blood
- V) **Pneumonia** = infection of lung parenchyma
- VI) **Bronchiectasis** = dilatation of bronchi
- VII) **Lung abscess** = pneumonia with form of cavities
- VIII) **COPD** = chronic obstructive airways disease
- IX) **Chronic bronchitis / emphysema**  
→ Part of COPD

**b) Information at the bedside**

- I) **Oxygen supplementation**  
→ Oxygen mask . Nasal cannula
- II) **Inhalers**  
→ Deliver drugs
- III) **Oxygen concentrators**  
→ Long term low dose oxygen delivery

**IV) Ventilators**

→ For pulmonary failure patients

**V) Peak expiratory flow meter (PEFR meter)**

→ Assess air flow rate, but NOT volume

→ Monitor disease severity in Asthma

→ **Asthma patients normally have a reduced PEFR at night**

→ **Procedure**

- 1) Set zero of the meter
- 2) Stand up straight
- 3) Take a deep breathe
- 4) Place the meter in mouth and blow out as hard, fast as possible
- 5) Repeat 2 more times, write down the **HIGHEST** number
- 6) If cough, then repeat again
- 7) Same time doing it each day

→ **Limitations**

- 1) Effort dependent
- 2) Not useful for patients with very poor lung function

**VI) Pulse oximeter**

→ Check the ratio of Oxy- to deoxy- haemoglobin

→ **SaO<sub>2</sub> %**

→ **Affected by**

- 1) Skin colour
- 2) Circulatory state
- 3) Hb concentration

→ **Limitations**

- 1) <70%
- 2) Presence of abnormal Hb
- 3) Presence of dye
- 4) Increased bilirubin
- 5) Shock

c) **Bedside skill**

I) **General examination**

- 1) Tachypnea
- 2) Oxygen supplementation, sputum collection
- 3) **Breathing pattern**  
→ Breathlessness ?? (**Normal 12-18 / min in adult**)
- 4) **Cyanosis**
- 5) Clubbing  
→ Loss of angle  
→ Floating sensation  
→ Drumstick appearance
- 6) Swelling of head and neck  
→ **Superior vena cava obstruction**

II) **Inspection**

- 1) Asymmetry, scars, scoliosis & kyphosis(脊柱側彎/後突)
- 2) Flattening / over-inflation
- 3) Respiratory movements  
→ Upper chest + Lower zones 前後都要

III) **Palpation**

- 1) Cervical lymph nodes
- 2) Position of the mediastinum  
→ Heart apex
- 3) Chest expansion
- 4) Mass /Pain / Tenderness

IV) **Percussion**

- 1) Resonance  
→ All resonant **EXCEPT liver and heart**  
→ Percuss each intercostal space
- 2) Lower border  
→ 6<sup>th</sup> intercostal space between sternum to the mammalian line  
→ 8<sup>th</sup> i.s. at midaxillary line  
→ 10<sup>th</sup> at scapular line  
→ 11<sup>th</sup> near the vertebrae

V) **Auscultation**

- 1) Breath sounds arise from turbulence in larynx and central airways
- 2) Added sound  
→ Wheeze  
→ Creptitation

### 13) Fever – Pyrexia

- ⇒ Usually >38.3°C over 1h
- ⇒ Normal diurnal variation (6am lowest, 4-6pm highest)
  - ± 0.5°C
- ⇒ **Cause**
  - I) **Exogenous Pyrogens**
    - Microbes and their products
  - II) **Endogenous Pyrogens**
    - Macrophage or monocyte derived chemicals
      - TNF, IFNs, IL-6

### 14) Hyperthermia

- Imbalance between production and loss
- No diurnal variation
- Usually very high temperature (>42.5°C)
- a) Cause
  - I) **Exercise + Environmental**
  - II) **Endocrine:** Rarely Thyrotoxicosis
  - III) **Failure of sweating**
  - IV) **Special syndrome**
    - Malignancy / Neuroleptic
- b) Signs
  - I) **Very high core temp.**
  - II) **Hallucination**
  - III) **Dry skin**
  - IV) **Muscle rigidity**
- c) Treatment
  - I) **Cease exposure**
  - II) **Antipyretic**
  - III) **Treat cause**

### 15) Women B.T

- 2 weeks post ovulation to menses core temp rises ± 0.6°C

**16) Fever of unknown Origin (FUO) \*\***

**a) Basic background**

- I) T>38.3
- II) Several times over 3 weeks
- III) No diagnosis after inspection in hospital for 1 week

**b) Nosocomial FUO**

⇒ Originate in hospital, not diagnosed after inspection for 3 days

- I) Drugs
- II) Vascular access sites and Foreign bodies
- III) Transfusions of
- IV) Thrombophlebitis

**c) Neutropenic FUO**

- Neutrophil count <500
- Very urgent

**d) HIV-associated FUO**

- TB, SALMONELLA, FUNGI, LYMPHOMAS, ETC.

**17) Chill**

⇒ Sensation of feeling cold

**18) Rigors**

⇒ Shivering + Chattering teeth

→ 80% patients with rigors may have malaria, pneumonia, biliary and renal infection

**19) Pros of fever**

- I) Reduced microbe virulence
- II) Increased Phagocytic activity
- III) Increased in animals survival

**20) Cons of fever**

- I) O<sub>2</sub> consumption increase
  - 13% for every 1°C increase
  - Stressful in cardiac and CV ischaemia and pregnancy
- II) Reduced appetite and mental slowing in Cachexia
- III) Seizures

## 21) Diagnosis of fever

- I) History
  - Travel, Pets, Hobbies, Drugs (antibiotics, steroids, NSAIDs)
  - Past surgery, medical, menstrual, sexual, family history
- II) Patterns of Fever
  - a) Intermittent, Sustained, Hectic(Septic)
    - Septic (Great variation)
  - b) Tertian, Quartan
    - Tertian: Every third day (P.ovale or vivax) (Malaria)
    - Quartan: Every first and forth day (Malaria)
    - Relapsing: 3-10days have, then 3-10days not (neutropenia)
  - c) Reversed Diurnal Variation
    - Typhoid, Miliary TB
  - d) Intermittent, Remittent
  - e) Drops by Lysis or Crisis
    - Drop by Lysis = Dropping gradually
    - Drop by Crisis = Dropping rapidly and suddenly
- III) Thermometry
  - a) Oral
  - b) Axillary
  - c) Remote aural / tympanic infrared sensor
  - d) Repeated Physical examination
  - e) Rectal examination
  - f) Genital examination
- IV) Lab tests
  - a) WCC increase
    - Due to acute stress, subside in 24h
    - \*\* Increase WCC count do not equal to infection
  - b) Result of smear / culture
    - Blood culture for 2-3 samples, taken out from different sites
    - If inflammatory endocarditis, 3 is normally needed
    - In Complex fever, serum sample is needed

## 22) Special investigational approaches

- I) Direct visualization of inaccessible part
  - Bronchoscopy, Sigmoidoscopy & Colonoscopy, Endoscopy, Cystoscopy, Laparoscopy
- II) Diagnostic Medical imaging
  - X-rays, ultrasound, CT, MRI
- III) Aspiration or Surgical exploration

## 23) Important points about fever

- I) Fevers not due to infection
  - Lymphomas, malignancies, Acute MI, Post Op, arthritis, skin diseases, drug interaction, Cardiac Infarction
- II) Steroids and NSAIDs can control fevers
  - But may help spread of infection
  - ONLY GIVEN when the origin is known
- III) Very ill point
  - Empirical anti-infective Rx (經驗藥)
- IV) Infective control is also important
  - Sterile, hand washing, antibiotics

## 24) Fever patterns

- I) **Intermittent**
  - Touch / not touching baseline (E.g. TB, Viral)
- II) **Hectic**
  - Exaggerated Diurnal variation (E.g. Daily hectic fever spikes)
- III) **Tertian**
  - Alternative day fever spike (E.g. P.malariae 1 & 4<sup>th</sup> day)
- IV) **Relapsing**
  - 3-10 days febrile and then 3-10 days afebrile (E.g. Borrelia recurrents)
- V) **Reversed Diurnal Variation**
  - Typhoid, miliary TB
- VI) **Pulse-temperature deficit**
  - Drugs, typhoid, heart involved

25) Basic Work-up for PUO

- I) Infection
- II) Autoimmune
- III) Neoplasm

26) Key points about FUOs

- I) In resource poor areas, mainly due to infection
- II) Increasing proportions are now due to connective tissue diseases, or remain undiagnosed
- III) In older patients, malignancy is the usual cause
- IV) The longer an FUO remains undiagnosed, the better the prognosis

27) Loss of consciousness

- I) Syncope
- II) Seizures (Epilepsy)

28) Syncope 眩暈

- ⇒ Overall
- ⇒ Failure of cerebral perfusion with reduction in cerebral O<sub>2</sub> availability
- ⇒ Loss of consciousness briefly

a) Simple Causes

- I) **Cardiac cause**
  - ⇒ Arrhythmias, Structural (Cardiomyopathy)
- II) **Hypotension cause**
  - ⇒ Drugs, Neuropathy (Dysautonomia)
- III) **Cerebrovascular ischemia**
  - ⇒ Embolism, occlusive disease, atherosclerosis
- IV) **Hypovolemia**
  - ⇒ Blood loss ,dehydration
- V) **Metabolic disorders**
  - ⇒ Anoxia, hypoglycemia, hyperventilation-induce alkalosis

**b) Multifactorial causes**

**I) Vasovagal syncope**

- ⇒ Presyncopal symptoms
    - ➔ Fatigue, nausea, weakness, sensation of impending faint
  - ⇒ Signs of autonomic hyperactivity
    - ➔ Pallor, nausea, sweating
  - ⇒ Enhanced by hot and crowded environment
- II) Situation syncope**
- ⇒ Reflex syncope
    - ➔ E.g. Cough syncope, Micturition syncope

**c) Symptoms of syncope**

**I) Premonitory symptoms**

- ➔ Palpitation, fluttering sensation in cardiac syncope
- ➔ Lightheadedness, diaphoresis 發汗 in vasodepressor and hypotensive syncope

**II) Presence of precipitating factors**

- ➔ Emotion, heat, crowds in vasovagal syncope
- ➔ Exertion, exercise in cardiac syncope

**III) Duration of faint**

- ➔ Rapid onset in cardiac syncope, may be more prolonged than syncope from other causes

**IV) Relation to posture**

- ➔ Cardiac causes occur in any position
- ➔ Upright position in orthostatic hypotension and in vasovagal syncope

**d) Investigation of Syncope**

- I) Normal Stats
- II) ECG
- III) Tilt table test

## 29) Epilepsy

- ⇒ Results from hypersynchronization of neuronal network in the cerebral cortex
- ⇒ **Defined as** recurrent unprovoked seizures

### a) Identified causes

- I) Brain tumors
- II) Stroke
- III) Neurodegenerative process
- IV) CNS infection
- V) Head trauma
- VI) Metabolic disturbance
- VII) Substance abuse, drug withdrawal

### b) History

- I) Aura
  - ⇒ E.g. flashing light, psychic experience
- II) Other ppl witness

### c) Types of seizures

- I) Absence seizures (Generalized seizures)
  - ⇒ Sudden staring with impaired consciousness, lasting for seconds
- II) Generalized tonic-clonic seizures
  - ⇒ stiffening of arms and legs and trunk
  - ⇒ jerking or twitching for a few minutes
- III) Post-Ictal state
  - ⇒ Follows an end of seizure
  - ⇒ Confusion, suppressed, alertness, focal neurological signs

### d) Investigation

- I) Liver function test
- II) Lumbar puncture
- III) Electrolytes + glucose

### 30) Syncope VS seizure

Features	Syncope	Seizure
Relation to posture	Common	No
Precipitating factors	Emotion, pain, crowds, specific situations	Sleep loss, alcohol, drugs
Skin color	Pallor	Normal or cyanosis
Aura or premonitory symptoms	Longer duration	Brief
Convulsion	Rare	Common with convulsive seizures
Urinary incontinence	Rare	Common
Post-event confusion	Rare	Common
Focal neurological signs	No	Occasional

### 31) Impaired consciousness

- I) Delirium 發狂
- II) Coma 昏迷

### 32) Delirium

⇒ acute mental status change characterized by abnormal and fluctuating attention

#### a) Clinical features

- Over hours to day
- I) Disturbance of consciousness
  - II) Change in cognition
  - III) EEG change almost always present

#### b) Pathophysiology

- Likely a result of widely distributed neurological dysfunction  
 → Can occur on top of dementia (癡呆)

**c) Cause**

- I) **Metabolic**  
→ E.g. Hypoxia, electrolyte disturbance
- II) **Drug related**  
→ E.g. Withdrawl syndrome, cocaine, amphetamines
- III) **CNS**  
→ E.g. Stroke, subdural hematoma, epilepsy
- IV) **Infections**  
→ Encephalitis,

**d) Differential Diagnosis**

- I) **Stroke with aphasia (失語)**  
→ Sudden onset  
→ Chronic and stable deficit  
→ Normal perception, no hallucination
- II) **Psychiatric condition**  
→ E.g. Depression  
→ Normal EEG

**e) Prevention and management**

- I) Attention to glucose, electrolyte, hydration
- II) Watch out for sepsis
- III) Environment intervention
- IV) Haloperidol, Risperidone

**33) Coma**

⇒ A state of complete unresponsiveness to arousal, with loss of the sleep-wake cycle

**a) Non-structural causes**

**→ More than half are due to diffuse / metabolic brain dysfunction**

- I) Toxins: CO, Cyanide, Methanol
- II) Drugs: Sedative, alcohol
- III) Metabolic: Hypoxia, lactic acidosis
- IV) Infection: Sepsis, Meningitis, encephalitis
- V) Psychiatric
- VI) Others

b) **Structural causes**

- I) **Supratentorial**
  - ➔ Hemispheric mass with herniation
- II) **Infratentorial**
  - ➔ Brain stem tumour, pontine hemorrhage
- III) **Subarachnoid hemorrhage**
- IV) **Subdural hematoma**

c) **History**

- I) **Headache, chest pain, Falling, certain cause**
- II) **Psychiatric history**
- III) **Background of coronary artery disease**

d) **Neurological examination**

- I) **State of consciousness**
  - ➔ Glasgow Coma Scale
- II) **Pupil size and reactivity**
  - ➔ Dilated pupil
    - ➔ 3<sup>rd</sup> nerve palsy
- III) **Ocular examination**
  - ➔ Eye displaced downward and laterally in unilateral 3<sup>rd</sup> nerve palsy

e) **Motor examination**

- I) **Muscle tone**
  - ➔ Usually Symmetrical and normal / decreased
- II) **Motor restlessness , tremors, spasms**
- III) **Myoclonic jerks**

f) **Investigation**

- ➔ Arterial Blood gas, ECG, EEG, Cortisol level

g) **Brain Death**

- ➔ No spontaneous respiration
- ➔ Absence of brainstem reflexes
- ➔ Absence of cerebral blood flow
- ➔ Electrocerebral silence

34) Dizziness with normal consciousness

a) **Vertigo 眩晕**

→ Sensation of spinning

→ Due to a lesion within vestibular pathways, either peripheral / central

→ Cause (Peripheral)

I) **Vestibular neuritis \*\*\***

→ Severe or rapid onset, with nausea, vomiting, imbalance

→ Likely viral cause

II) **Benign paroxysmal positional vertigo (BPPV)**

→ Brief episodes vertigo

→ Caused by calcium carbonate debris in semicircular canal

→ Most common

III) **Meniere's disease**

→ Recurrent attacks of vertigo associated with auditory symptoms

→ Abnormal fluid and ions in inner ear

Two spontaneous episodes of rotational vertigo lasting at least 20 mins

- Audiometric confirmation of sensorineural hearing loss
- Tinnitus and/or a perception of aural fullness

→ Cause (Central)

→ Stroke (Brainstem / cerebellar ischemia)

→ Neurodegenerative disorders

b) **GAIT**

→ Patients with acute peripheral vestibular lesion typically can stand but may veer towards the side of lesion

→ Patients with vertigo of central origin are often unable to stand without support; may have associated neurologic signs such as dysarthria, ataxia, hemiplegia

### 35) Physical examination of CVS

#### a) Terminology and background

##### I) Aortic Stenosis

→ Obstructed forward flow from left ventricle during systole

##### II) Mitral stenosis

→ Obstructed forward flow from left atrium during diastole

##### III) Aortic Regurgitation

→ Backflow

##### IV) Conduction system of heart

- 1) Resting polarization
- 2) Contracting depolarization
- 3) Recharging repolarization

##### V) Fetal special structures

###### 1) Foramen ovale

→ Connect Left and right atrium

→ If not close, Patent foramen oval (PFO)

###### 2) Ductus arteriosus

→ Connect proximal descending aorta to pulmonary artery

###### 3) Ductus venosus

→ Connect aorta and pulmonary artery

→ If not close, Patent ductus venosus (PDV)

##### VI) Heart Sound

###### 1) HS1 Lubb

→ Longer and lower pitch

→ M1 and Tricuspid valve contraction (Normally M1 faster)

###### 2) HS2 Dubb

→ Shorter and higher pitch

→ Aortic and pulmonary valve contraction (Normally aortic faster)

→ \*\*If split

I) Right bundle branch block

II) Pulmonary stenosis

III) Atrial septal defect

###### 3) Murmur (Swishing)

**b) General examination**

➔ Cyanosis, Cachexia, Oxygen, Pallor

**c) Inspection**

**I) Face**

**1) Pull down Eyelid:** Pallor?

**2) Cheeks:** Mitral facies (Rosy cheeks with bluish tinge)

➔ Severe mitral stenosis

**3) Corneal arcus and xanthalasma**

➔ Hypercholesterolemia

**4) Conjunctival hemorrhage**

➔ Infective endocarditis

**5) Tongue**

➔ Central cyanosis

**6) Turner Syndrome:** Coarctation of aorta

**7) Down Syndrome:** Atrial septal defect / Ventricular septal defect

**8) Williams Syndrome:** Left and right heart obstruction

**II) Hand**

**1) Clubbing**

**2) Peripheral cyanosis**

**3) Marfans syndrome**

➔ Elongated finger and arm bones + Hyperflexibility

➔ Prolapse of mitral valve

➔ Aortic regurgitation

**4) Nicotine staining**

## 5) Radial pulse

→ Rate, rhythm, character, volume,

### I) Collapsing pulse (洪脈)

→ High volume pulse hits and falls suddenly quickly

→ Aortic regurgitation \*\*\*

### II) Bounding pulse

→ Strong and powerful

→ CO<sub>2</sub> retention / Liver failure / sepsis 腫毒症

### III) Radio-femoral delay

→ Aortic coarctation

### IV) Radio-radial delay

→ aortic Coarctation 收窄/ dissection 撕裂

### V) Regularly irregular

→ 2<sup>nd</sup> degree heart block

### VI) Irregularly regular

→ Atrial fibrillation

## III) Neck

### 1) Right internal jugular vein pulsation

→ Check pressure 5CM up from sternal angle

→ If elevated, then HEART FAILURE

### 2) Hepatojugular Reflex

#### I) Open mouth, lie, breathe normally

#### II) Right hand over RUQ and apply pressure 10s

#### III) Elevated JVP = sustained hepatojugular reflex

→ Right ventricular failure

**3) Carotid pulses**

→ Palpate and auscultate for carotid bruits

I) If **weak and small**

→ Heart failure, aortic stenosis

II) If **large and bounding**

→ Fever, hyperthyroidism, bradycardia

III) **Bisferiens**

→ Increased arterial pulse with double systolic peak

→ Aortic stenosis, regurgitation

IV) **Pulsus alternans**

→ Amplitude varies from peak to peak

→ rhythm regular

→ Left ventricular failure

V) **Pulsus paradoxus**

→ Pulse smaller on deep inspiration

→ Constrictive pericarditis

**IV) Others**

**1) \*\*Leg BP > arm**

→ Coarctation of Aorta

**2) \*\*Asscess circulation**

→ Temperature

→ Colour

**3) Scars / Pacemakers**

**4) Chest wall deformity**

**5) Non-pitting Edema**

→ Lymphedema

**6) Ruptured baker's cyst**

**d) Palpation**

I)      **Apex beat**

- ➔ Displaced ?
- ➔ **Heaving?** (Sharp and firm = Aortic stenosis)
- ➔ **Tapping?** (Quick and light = mitral stenosis)
- ➔ **Thrusting** (Diffuse and long = Mitral regurgitation)

II)     **Thrill**

- ➔ Palpable murmur over valvular regions
- ➔ Mitral 5<sup>th</sup> lateral
- ➔ Tricuspid 5<sup>th</sup> medial
- ➔ Aortic 3rd medial left and right

**e) Auscultation**

I)      **Heart sounds**

- ➔ 1st sound = Close of A-V valve
  - If weak
    - 1) **Mitral regurgitation**
    - 2) **Calcified mitral valve**
    - 3) **Severe heart failure**
  - ➔ 2nd sound = Close of Aortic valve + pulmonary valve
    - If weak
      - 1) **Aortic stenosis**
      - 2) **Pulmonary hypertension**

II)     **Additional sounds**

- ➔ 3<sup>rd</sup> sound = only normal in child
- ➔ 4<sup>th</sup> sound = Physiologic various diseases

### **III) Murmurs**

→Narrowing / incomplete closure

→Site

→Tricuspid area, Aortic area, Pulmonary area

→intensity,

→timing

I) **Systolic Ejection \*\***

→Just after HS1 and ends before HS2

→Aortic stenosis, pulmonary stenosis

II) **Pansystolic \*\***

→Throughout Between HS1 and HS2

→Mitral regurgitation

III) **Systolic click late systolic**

→Mitral Valve prolapse

IV) **Early Diastolic**

→Starts immediately after S2

→Aortic regurgitation

V) **Mid diastolic**

→Mitral stenosis

VI) **Opening snap diastolic rumble**

→Mitral stenosis

VII) **Continuous Murmur →Patent ductus arteriosus**

→radiation, pitch

→\*\*\*All right side murmur louder with inspiration

IV) **Percardial rub**

V) **Carotid bruits**

→Artery stenosis

### **f) Pulmonary and liver congestion examination**

I) **Back**

→Percussion and auscultation of the lung bases

→Crepitations (Pulmonary edema)

→Sacral edema

→Dull percussion / decreased air entry (pleural effusion)

II) **Abdomen**

→Ascites, tender hepatomegaly, pulsatile liver, ascites, splenomegaly

### **36) Infective endocarditis signs**

- I) **Splinter haemorrhage**  
→ Tiny blood clots tend to run vertically under nails
- II) **Conjunctival haemorrhage**
- III) **Petechiae on the palate**
- IV) **Osler's nodes** =  
→ tender lumps in pulp of fingertips
- V) **Janeway lesions** =  
→ red macules on wrist and hand

### **37) Hypertension**

#### **a) Classification (The higher category should guide classification)**

- I) **Normal** : systolic <120, diastolic <80
- II) **Prehypertension** 120-139, 80-89
- III) **Stage 1:** 140- 159, 90-99
- IV) **Stage 2:** >159, 99

#### **b) Causes**

- I) **Essential**  
→ No defined cause
- II) **Secondary**
  - 1) **Endocrine**  
→ Cushing diseases (Cortisol)  
→ Conn's syndrome (Aldosterone)  
→ Phaeochromocytoma (Adrenaline)
  - 2) **Chronic Renal Diseases**
  - 3) **Vascular**  
→ Coarctation of aorta, renal artery stenosis

#### **c) Measurement**

- I) Support arm at the level of the heart
- II) Lower mercury level slowly until a sound (Systole)
- III) Use phase V to detect diastole (Disappearance of sound)
- IV) 2 Measures each

#### **d) Non-pharmacological therapy**

- I) Low salt, fat diet
- II) Stop smoking, drinking
- III) Aerobic exercise at least 30mins/day

**e) Pharmacological therapy**

- I) Diuretics
- II) Beta blockers
- III) Calcium channel blockers
- IV) Vasodilators
- V) Angiotensin converting enzyme inhibitor (ACE inhibitor)

38) Heart failure

**a) Signs**

- I) Ankle edema
- II) Increased Jugular venous pressure
- III) Tachycardia
- IV) Gallop rhythm
- V) Crepitation 撥發音

**b) Symptoms**

- I) **Dyspnoea** (Shortness of breathe)
- II) **Ankle swelling**
- III) **Orthopnoea** (平臥氣促)
- IV) **Paroxysmal Nocturnal dyspnoea** (Sudden and severe dyspnea at night)

**c) Treatment**

- I) **Reduction of cardiac work**  
→ Bed rest + mental rest + small meal
- II) **Treat the cause \*\***
  - 1) Cardiomyopathy
  - 2) Hypertension
  - 3) Vulvar diseases
  - 4) Coronary artery diseases
  - 5) Cardiac arrhythmias
  - 6) Atrial fibrillation
- III) **Strengthening of myocardial contraction**  
→ Cardiac glycosides (**Digoxin**)  
→ Direct inotropic effect
- IV) **Removal of XS sodium and water and prevent their re-accumulation**  
→ Diuretics + K supplements

39) Valvular heart disease

a) Left heart disease

I) **Mitral stenosis**

- ➔ Mid diastolic murmur
- ➔ Predispose to Atrial fibrillation

II) **Mitral regurgitation**

- ➔ Pan-systolic murmur

III) **Aortic stenosis**

- ➔ Ejection Systolic murmur
- ➔ Small volume pulse

IV) **Aortic regurgitation**

- ➔ Early blowing diastolic murmur
- ➔ Collapsing pulse

b) Right heart disease

➔ Less common (Pulmonary / Tricuspid)

➔ Stenosis, Regurgitation, Incompetence

40) Cardiac Arrhythmias

a) **Bradyarrhythmias**

➔ Mainly due to **AV node block**

➔ 1<sup>st</sup>, 2<sup>nd</sup>, 3<sup>rd</sup> degree

b) **Tachyarrhythmias**

➔ Mainly due to **atrial fibrillation(AF)**

I) **Cause of AF**

1) Pericarditis	2) Cardiomyopathy	3) Idiopathic
4) Thyrotoxicosis	5) Ischemic heart disease	6) Hypertension

II) **Treatment**

1) **Rate control**

➔ Beta blocker, digoxin, Ca2+ channel blocker

2) **Rhythm conversion**

➔ Class 1C, III Antiarrhythmics

➔ Propafenone + Amiodarone

3) **Prevention of Cerebrovascular accident (CVA)**

➔ Stroke

➔ Aspirin, Warfarin

## 41) Ischemic Heart Disease (Acute myocardial infarction)

### a) Criteria

- I) Chest pain
- II) ECG changes
- III) Cardiac Enzymes rises  
→ CPK, Troponin, AST, LDH
- IV) MRI Cardiac perfusion abnormal

### b) Treatment

- I) Bed Rest
- II) Oxygen
- III) Medicine: Aspirin, Nitrates, Beta blocker
- IV) Calcium channel blockers
- V) Thrombolytic Agents  
→ TNK, Streptokinase

## 42) Dyspnoea (Breathlessness)

### a) Background

- Abnormally uncomfortable awareness of breathing
- Involves perception of sensations, subjective
- A sign of serious diseases of the airways lungs or heart

### b) Causes

- I) **Airway diseases**  
→ E.g. Asthma, COPD, Abnormal dilatation (Bronchiectasis)
- II) **Pulmonary Parenchymal diseases**  
→ Pneumonia, fibrosis
- III) **Pulmonary vascular diseases**  
→ Pulmonary embolism, primary pulmonary hypertension
- IV) **Pleural / Chest wall diseases**  
→ Pneumothorax, kyphoscoliosis
- V) **Cardiac Causes**  
→ Cardiac arrhythmia, heart failure, ischaemic heart disease
- VI) **Central Nervous system diseases**  
→ Hemorrhage, abscess, central tumour
- VII) **Neuromuscular**  
→ Myasthenia gravis (重症肌無力)
- VIII) **Others**

**c) Classification**

- I) Acute (Within minutes)
- II) Non- acute (Within hours – weeks)
- III) Chronic and progressive (months – years)

**d) History**

- I) Onset and duration
- II) Respiratory, cardiac, systemic assessment result
- III) Change in exercise tolerance
- IV) Smoking history
- V) Occupational exposure (May lead to asthma)
- VI) **Chest pain \*\*\***
  - ➔ Pneumothorax, embolism
  - ➔ Aortic dissection
  - ➔ Acute myocardial infarction with heart failure
- VII) **Orthopnoea \*\*\***
  - ➔ Heart failure
  - ➔ Bilateral diaphragmatic paralysis

**e) Physical Examination**

- I) **Clubbing**
  - ➔ Cancer, bronchiectasis, pulmonary fibrosis
- II) **Cervical Lymph node Enlargement**
  - ➔ Lung cancer
- III) **Raised JVP + Ankle edema**
  - ➔ Heart failure
- IV) **Unilateral diminished breath sound**
  - ➔ If + stony dullness = pleural effusion
  - ➔ If + Hyper resonance = pneumothorax
- V) **Expiratory wheeze**
  - ➔ Asthma / COPD
- VI) **3 Depression Signs for inspiratory dyspnea \*\*\***
  - 1) Suprasternal fossa
  - 2) SuprACLAVICULAR fossa
  - 3) Intercostal space

**f) Investigation**

- ➔ ECG, chest x-ray, Lung function test, Oximetry, blood tests, exercise test

**g) Treatment**

- I) Oxygen therapy + Non-invasive positive pressure ventilation
- II) Treat specific underlying cause
- III) Exercise training
- IV) Narcotics \*\*\*

**43) Dyspnoea (Breathlessness)**

**a) Causes**

- I) Post-bronchitis syndrome
- II) Lung cancer
- III) Drug-induced
  - E.g. Angiotensin converting enzyme inhibitor
- IV) Interstitial lung diseases

**b) Classification**

- I) Acute / Chronic
  - >3 weeks = chronic
- II) Characteristic
  - Dry cough
  - Productive cough = infectious , edema
- III) Attack
  - 1) Time
  - 2) Season
  - 3) Motivation
    - Fume inhalation, exertion
- IV) Tone
  - 1) Hoarseness 沙啞
  - 2) High pitch
  - 3) Weak
  - 4) Brassy 刺耳的

**c) History**

- I) Fever
- II) Chest pain
- III) Dyspnea
- IV) Wheezing
- V) Clubbing of fingers
- VI) Hemoptysis

**d) Physical Examination**

I) **Chest**

1) **Expiratory wheeze**

→Asthma, COPD

2) **Inspiratory crackles**

→Bronchiectasis, interstitial lung diseases

II) **ENT**

→Examine throat for evidence of postnasal drip 流水倒流

→**Cobblestone appearance \*\***

- 1) Nasal discharge → 2) Swollen turbinates → 3)Post-nasal drip

**e) Investigation**

→ chest x-ray, Lung function test, Bronchial challenge test

**f) Treatment**

⇒ Treat specific underlying cause

→Anti-tussive agents (Narcotics to suppress cough)

I) **Asthma**

→Inhaled bronchodilator + steroid

II) **GERD**

→Anti-reflux surgery

→Lifestyle modification (no meal before bedtime)

III) **Postnasal drip syndrome**

→Nasal steroid + antihistamine

44) Dyspnoea (Breathlessness)

**a) Causes**

I) **Pulmonary:** Cancer, embolism, haemorrhage, pneumonia

II) **Cardiac:** pulmonary edema, mitral stenosis

III) **Others:** Epidermic hemorrhagic fever, trauma

**b) History**

I) **Vs hematemesis**

- 1) Vomiting + Nausea ?
- 2) Frothy ?
- 3) Colour ?
- 4) pH ?
- 5) Sputum or food ?
- 6) History of lung / gastric diseases
- 7) Significant blood loss

**c) Physical Examination**

- I) Clubbing
- II) Cervical LN enlarge
- III) SVC obstruction  
→Lung cancer
- IV) Calf swelling  
→DVT with pulmonary embolism
- V) Chest signs  
→Consolidation, collapse, coarse crackles
- VI) CVS: Features of mitral stenosis

**d) Investigation**

- I) chest x-ray, Lung function test
- II) Sputum analysis  
→Fungal culture, cytology, AFB smear
- III) Bronchoscopy
- IV) CT thorax

**e) Treatment**

- I) **Secure airway**
- II) **Maintain blood pressure**
- III) **Stop bleeding**
- IV) **Treat underlying causes**

## 45) Vomiting and nausea

### a) Introduction

→ **Vomiting:** Forceful expulsion of the contents from stomach through the mouth/nose

→ **Nausea:** Feeling about to vomit, but not always lead to vomiting

### b) Causes

I) GI disease	II) Brain & Sensory
1) Gastritis	1) Motion sickness
1) Gastroenteritis	2) Brain tumour
1) Food poisoning	3) Pregnancy
2) Bowel obstruction	4) Hypercalcemia

### c) Mechanism

#### 1) Stimulation

⇒ **Stimulation of receptors on the floor of the 4<sup>th</sup> ventricle \*\*\***

- I) Dopamine D2, Serotonin, Opioid and Acetylcholine receptors
- II) Vestibular system via cranial nerve VIII
- III) Cranial Nerve X (Gag reflex)
- IV) Enteric nervous system in the GI tract
- V) Stress from the higher centres

#### 2) Common Actions

- I) **Increased salivation**
- II) **Deep breath to increase aspiration + Closure of vocal cord**
- III) **Retroperistalsis from jejunum + Relax of pyloric sphincter**
- IV) **Contraction of abdominal muscle**
  - Lower intra-thoracic and Increase abdominal pressure
- V) **Relax of pyloric sphincter and lower esophagus sphincter**
- VI) **Initiate sympathetic response**
  - Sweating and increase pulse

### d) **Contents of reflux**

- I) Acidic gastric secretion with food
- II) Hematemesis
- III) Coffee ground
- IV) Bile from duodenum
- V) Fecal vomiting (In intestinal obstruction)

**e) Complications**

- I) Aspiration Pneumonia
- II) Dehydration and electrolyte imbalance
- III) Mallory-Weiss Tear (Tear in esophagus)
- IV) Dentistry problem

**f) Treatments (Antiemetics)**

- I) **5HT3 antagonists**  
→ Ondansetron, Granisetron
- II) **Dopamine antagonist**  
→ Domperidone, Metoclopramide
- III) **Antihistamine**  
→ chlorphenamine

46) Diarrhea

**a) Introduction**

- **Definition**
- 3 or more loose or watery stools per day
  - **Acute:** <14days, mostly viral and bacterial, self-limiting
  - **Persistent:** >14days,
  - **Chronic:** >30days: non infectious
  - \*\*\*Dilemmas: when to test and to initiate therapy

- I) **Non-inflammatory diarrhea**  
→ Watery stools, large volume (>1L/day)  
→ Without blood, pus, severe abdominal pain, fever
- II) **Inflammatory diarrhea**  
→ Frequent, small-volume  
→ Mucoid / bloody stools  
→ Leucocytes in stool (With fever, tenesmus)

**b) Etiology**

- I) **Viruses**  
→ Self-limiting and non-inflammatory  
→ E.g. Calciviruses, Rotavirus, Astrovirus, Ebola virus  
→ \*\*Cytomegalovirus (CMV) colitis  
→ Immunocompromised / organ transplant / elderly

- II) Bacteria**
  - ➔ Inflammatory
  - ➔ E.g. *Salmonella*, *Shigella*, *Campylobacter jejuni*, *Clostridium difficile*
- III) Protozoa**
  - ➔ Inflammatory
  - ➔ E.g. *Cryptosporidium*, *Microsporidium*, *Entamoeba histolytica*
- IV) Atypical micro-organism**
  - ➔ E.g. *Mycobacterium avium-intracellulare*
  - ➔ Associated with
    - ➔ AIDS, IgA deficiency, Immunosuppressive therapy
- V) Non-infectious**
  - I) Ulcerative colitis
  - II) Ischemic colitis
  - III) Crohn's disease

- c) Risk group**
  - I) **Antibiotic associated diarrhea**
    - ➔ *C. difficile* infection
  - II) **Children**
    - ➔ E.g. *ETEC*, *EHEC*, *C. jejuni*, *Giardia lamblia*
  - III) **Children <1**
    - ➔ *Salmonella* spp.
  - IV) **Children 6 months to 4 years**
    - ➔ *Shigella* spp.

- d) Clinical features**
  - I) **History**
    - ➔ Degree of severity, high fever, systemic toxicity, bloody diarrhea
  - II) **Diagnostic testing**
    - ➔ Dehydration, outbreak of food poisoning, recent antibiotics
  - III) **Examination of stools**
    - ➔ Grossly bloody or mucoid-inflammatory, leucocyte
    - ➔ **Stool for bacterial:** For ova and cysts

#### **IV) Clinical manifestation**

- 1) Food poisoning:
  - ➔ Clostridium perfringens
  - ➔ Severe Abdominal pain, cramps and diarrhea (longer incubat.8hrs)
- 2) Enterotoxin (Secereted by S aureus, Bacillus cereus)
  - ➔ Fever uncommon (1-6hrs)
- 3) Yersinia and Salmonella
  - ➔ Infect terminal ileum, cecum
  - ➔ Present with RLQ pain and tenderness
  - ➔ Acute Appendicitis
- 4) Extra-intestinal manifestation
  - ➔ Skin lesion, ocular symptoms, inflammatory bowel disease
  - ➔ May associate with Shigella, Salmonella, Yersinia, and C.difficile

#### **V) Lab Diagnosis**

- I) **Stool culture**
- II) **Isolation of C.jejuni onto selective growth medium at 42oc**
- III) **C.difficile toxin**
  - ➔ Recent antibiotics
  - ➔ Need hospitalization, day-care exposure or chemo
- IV) **AIDs**
  - ➔ Multiple stools for ova and parasites
- V) **Imaging**
  - ➔ CT scan with oral contrast

#### **e) Diagnostic procedures**

- I) **Colonoscopy + Diagnostic biopsies + Histology**
  - ➔ Double balloon enteroscopy
- II) **Small intestinal Biopsy**
  - ➔ Whipple's and M.avium complex
- III) **Duodenal aspirates / biopsy**
  - ➔ Protozoa
- V) **Capsule endoscopy**

**g) Complication**

- I) **Shigellosis and EHEC may lead to hemolytic-uremic syndrome**
  - ➔ Only in very young and old
- II) **Yersinia and other enteric bacterial infection**
  - ➔ May lead to HUS also
  - ➔ May lead to Reiter's Syndrome, thyroiditis, pericarditis

**h) Treatment**

- I) **Rehydration**
  - ➔ Fluid replacement (IV therapy)
- II) **Antidiarrheal agents**
  - 1) Loperamide (Opiates)
  - 2) Bismuth subsalicylate
- III) **Avoid milk and lactose-containing products**
  - ➔ Transient lactase deficiency
- IV) **Empirical antibiotics**
  - ➔ Fluoroquinolones 5days + Macrolide 3days

**f) Prevention**

- I) Improvement in hygiene
- II) Limit fecal-oral route spread of enteric pathogens
- III) Safe water and sanitation

**47) Basics about Liver Function Test**

- ⇒ Divided into 3 aspects
  - ➔ Cellular integrity + Protein synthesis + Excretory markers

**a) Evaluation of liver function test**

- I) Pattern of alteration
  - ➔ Hepatocyte predominant / Cholestasis predominant
- II) Magnitude of alteration
  - ➔ Mild / Moderate / Marked
- III) Nature of alteration
  - ➔ Increase / Decrease
- IV) Timing of alteration
  - ➔ Medications, Age
- V) Location of alteration

b) **Cellular integrity -- ALT and AST elevation**

I) **Introduction**

- ➔ **ALT:** 100% cytosolic enzyme, more specific to liver
- ➔ **AST:** 20% cytosolic enzyme, 80% mitochondrial enzyme
  - Also found in muscles, kidneys, pancreas, leucocytes, red cells
- ➔ **3 aspects**
  - Level of elevation
  - Pattern of elevation
  - Subsequent profile
- ➔ AST is also high in concentration in heart, muscle and kidney
- ➔ ALT in kidney

II) **Common cause**

- I) **Alcohol**
- II) **Drugs:** Paracetamol, NSAIDs
- III) **Autoimmune hepatitis**
- IV) **Congestive heart failure and ischemic hepatitis**
- V) **Viral hepatitis**

III) **Pattern**

⇒ Usually ALT level >> AST level in diseases primarily affecting the liver

➔ **EXCEPTS \*\*\*\***

- 1) **Alcoholic hepatitis**
  - ➔ AST > ALT, ratio > 2:1
  - ➔ Serum AST almost NEVER > 500U/L
- 2) **Hepatocellular carcinoma**
- 3) **Congestive Heart failure**
- 4) **Ischaemic hepatitis**

⇒ Usually ALT and AST will decline gradually \*\*

➔ **EXCEPTS\*\*\*\***

- 1) **Acute ischaemic hepatitis**
- 2) **Paracetamol overdose**
- 3) **Cholangiohepatitis (Hepatitis due to blockage of stone)**

c) **Protein synthesis – Albumin and PT**

I) **Albumin**

→ Approximately 10g is synthesized and secreted daily

→ Can be tested as **LIVER SYNTHETIC CAPACITY**

→ Factors make albumin level difficult to interpret \*\*

1) **Normally Liver can synthesize albumin at twice the healthy basal rate**

→ May not decrease significantly although impaired function

2) **Albumin half-life of 20days**

→ Level changes slowly in response to reduction in production

3) **Other contributed factors**

→ Nutritional, GI loss, Urine Loss

II) **PT (Prothrombin time)**

→ Liver synthesis Clotting Factors 2,5,7,9, 10

→ Half life of **FACTOR 7** is only 6 hours

→ Very sensitive to rapid changes in liver function

d) **Excretory markers – Bilirubin, ALP and GGT**

I) **Bilirubin**

→ Formed from lysis of red cells within reticuloendothelial system

→ Normally, capacity to conjugate bilirubin **far exceeds** the need

→ Most are associated with conjugated hyperbilirubinaemia

→ 3 situation leading to elevated unconjugated bilirubin(insoluble)

1) **Increased production of bilirubin**

→ Haemolysis, ineffective erythropoiesis

→ Resorption of haematoma

2) **Decreased hepatic uptake**

→ Drugs (E.g. Rifampicin)

3) **Decreased conjugation**

→ Gilbert's syndrome

→ Criggler-Najjar syndrome

→ Decrease bilirubin level \*\*

II) **ALP**

→ Bone, placenta can also produce

→ **Isolated high ALP** = bone disease / Skeletal growth, pregnancy

**III) GGT (Microsomal Enzyme)**

→**BEST marker** for excretory function of liver

→But also high in concentration in pancreas, kidney, prostate

→**Isolate high GGT**

→**Fatty liver, alcohol or Drug** (Barbituates, phenytoin)

**IV) Elevation of All Bilirubin + ALP + GGT + AST + ALT**

→Blockage of bile ducts by gallstone

**V) Elevation of ALP + GGT**

1) Primary biliary cirrhosis

2) Cholestasis

3) Infiltrative disease

→HCC, TB

4) Liver abscess

5) Hyperthyroidism

**e) Reason for initiating a lab test**

1) Confirm diagnosis

2) Aid diagnosis

3) Refine diagnosis

4) Monitor progress, therapy

5) Assess severity

6) Detect complication

48) Clinical features about Liver function test

**a) Hyperbilirubinaemia**

	<b>Haemolytic</b>	<b>Cholestasis</b>	<b>Hepatocellular</b>
<b>1) Level of bili.</b>	< 75umol/L	Dramatic increase	Delayed increase
<b>2) Urine bili</b>	No	Yes	Yes
<b>3) Other result</b>	Decreased haemoglobin  LDH may increase	Alk. Phos 3x increase  AST and ALT increase modestly  LH may increase	Alk. Pho delayed increase  AST and ALT increase greatly  LDH Increase

**b) Hepatocellular Pattern**

- I)      **ALT and AST** : Predominantly increase
  - Significant damage to hepatocytes
  - Release of AST and ALT into circulation
  
- II)     **ALP and GGT**: Normal / mild increase

\*\*\*Cause of damage to hepatocytes\*\*\*

→ Viral hepatitis, drugs, herbal toxins, shock,

**c) Cholestasis pattern**

- I)      **ALT and AST** : Normal / mild increase
  - Significant damage to hepatocytes
  - Release of AST and ALT into circulation
  
- II)     **ALP and GGT**: Predominantly increase
  
- III)    **Conjugated Bilirubin**: Predominantly increase
  - If **completely** obstructed: Predominantly increase
  - If **partially** obstructed: Mildly elevated

\*\*\*Cause of cholestasis or biliary obstruction\*\*\*

→ Gall stone, cholangitis, cholangiocarcinoma, biliary cirrhosis

**d) Mixed pattern**

- All increased
  
- I)      Prolonged biliary obstruction leading to significant liver cell destruction
  
- II)     Acute Hepatitis may give rise to severe cholestasis

**e) Comparison Between viral hepatitis with complete obstruction**

	<b>Acute hepatitis</b>	<b>Obstruction</b>
<b>1) Symptoms</b>	Painless jaundice	Pain jaundice
<b>2) %Conju bilirubin</b>	50-80	50-80
<b>3) AST, ALF</b>	>10X	<10X
<b>4) Alka. Phos.</b>	<3X	<3X early >5-10X normal latr
<b>5) Bile duct imaging</b>	Normal	Dilated

**f) Comparison Between Different hepatitis**

	Viral	Alcoholic	Toxic/Ischaemic
<b>1) Chronicity</b>	Variable	5-10%	Non
<b>2) AST/ALT ratio</b>	< 1	Usually >2	>1 (Transient)
<b>3) Peak AST</b>	10-100	1-10	>100
<b>4) Peak Bilirubin</b>	85-340	51-340	Usually 85
<b>5) PT</b>	Normal	Normal / Increase	Usually >15S
<b>6) LDH</b>	1-2	1-2	10-40

**g) Other hints**

- I) **Myopathy / muscle injury**  
→ Mild increase in ALT (<1.5X)
- II) **Alcoholic hepatitis**  
→ Minimal elevations of AST and ALT
- III) **Acetaminophen toxicity (Heavy drinker)**  
→ AST > 500U/L
- IV) **Common bile duct stone**  
→ AST and ALT elevated immediately  
→ ALP and GGT delay
- V) **Alcohol + Aromatic medications**  
→ Isolated GGT increase
- VI) **Bone growth / injury / pregnancy**  
→ Isolated ALP increase
- VII) **Gilbert syndrome / haemolysis**  
→ Isolated increase of unconjugated bilirubin

**49) Basics about of ECG**

- a) **Direction of electrical waveform**  
→ Anything moving away from lead: -ve  
→ Anything moving towards lead: +ve
- b) **Amplitude of electrogram**  
→ The larger the muscle, the larger the amplitude  
→ The farther away the lead from the heart, the smaller the amplitude
- c) **Cardiac muscle contraction**  
→ Action potential maintained by Na+ / K+ gradient  
→ Contraction is initiated by infiltration of Ca2+ into the muscle cells

**d) Electrical conduction**

**I) Origin of electrical**

→ Different types of cardiac cells spontaneously depolarize at differ.

Rate

→ \*\*The fastest pacemaker controls the heart rate

→ SA node 60-100 / min

→ Atrial cells 60-75/min

→ AV node 40-60/min

→ Ventricular cells 35-40/min

**II) Normal conduction cardiac cycle \*\*\***

1) SA node discharge

→ No deflection

2) Atrial muscle cells depolarization

→ \*\*\*P wave\*\*\*

3) AV node activation

→ No deflection

4) Bundle of His activation

→ No deflection

5) Septal activation (Ventricular contraction)

→ \*\*\*Onset of QRS complex\*\*\*

6) Free wall activation (Ventricular contraction)

→ \*\*\*Accomplishment of QRS complex\*\*\*

7) Full ventricular activation

→ No deflection

8) Ventricular repolarization

→ \*\*\*T wave\*\*\*

9) Late ventricular activation

→ \*\*\*U wave\*\*\*

→ Not seen in healthy person

**e) Approach to ECG**

- I) Determine rate and rhythm
- II) Look for any **fast / slow arrhythmia** (Normally 60-100)
- III) Look for **spot diagnosis**
  - ➔**AMI, LVH, BBB**
- IV) If not obvious, look for
  - ➔**ST-T Changes**
  - ➔**Pulmonary embolism**
  - ➔**Electrolyte and metabolic changes**
- V) Go through
  - ➔**Axis**
  - ➔**P-Q-R-S-T**

**f) Causes of cardiac arrhythmia**

- I) **Hypoxia**
- II) **Ischaemia**
- III) **Sympathetic Stimulation**
- IV) **Drugs**
- V) **Electrolyte disturbances**
- VI) **Bradycardia**
- VII) **Stretch**

**g) Symptoms of Arrhythmia**

- I) Asymptomatic
- II) Palpitations
- III) Dizziness, syncope
- IV) Angina
- V) Congestive heart failure
- VI) Sudden death

## **h) The 12 ECG leads and axis**

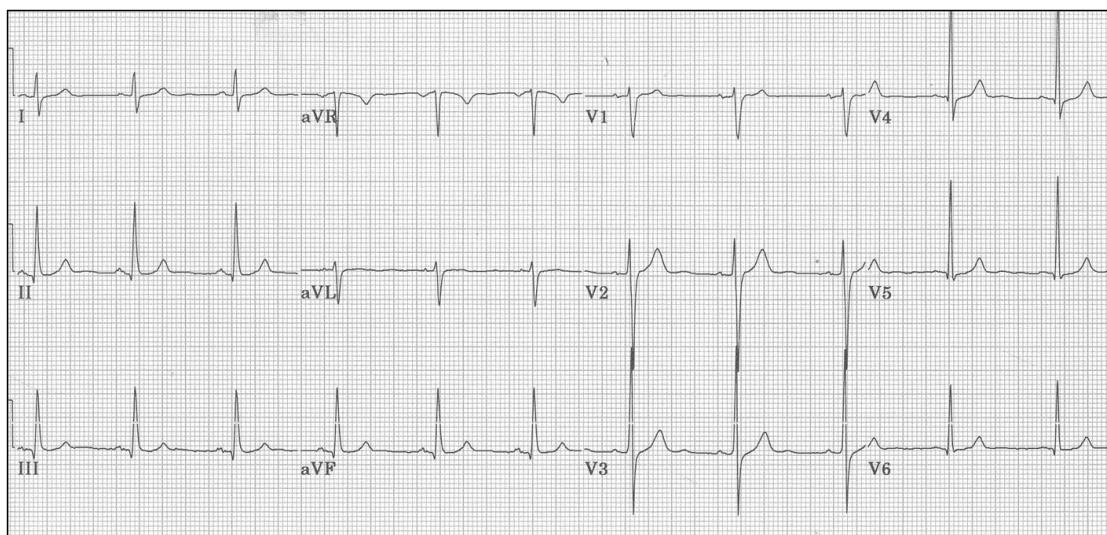
### **I) 6 Limb leads**

- 1) **I:** 0 degree (left horizontal line)
- 2) **II:** 60 degree
- 3) **III:** 120 degree
- 4) **aVR:** -30 degree
- 5) **aVL:** -150 degree
- 6) **aVF:** 90 degree (Vertical)

### **II) 6 precordial (Chest)**

- 1) **V1:** 4<sup>th</sup> intercostal space, Right side of sternum
- 2) **V2:** 4<sup>th</sup> intercostal space, Left side of sternum
- 3) **V3:** 5<sup>th</sup> intercostal space, between 2 and 4
- 4) **V4:** 5<sup>th</sup> Intercostal space, mid-clavicular line
- 5) **V5:** 5<sup>th</sup> intercostal space, between 4 and 6
- 6) **V6:** 5<sup>th</sup> Intercostal space mid-axillary line

50) A normal ECG



### **a) Normal conduction**

- I) **Heart Rate:** 60–90 bpm
- II) **PR interval:** 0.12 – 0.20 sec  
→ AV conduction
- III) **QRS duration:** 0.06 – 0.10 sec  
→ Intraventricular conduction
- IV) **QT interval:** < 0.40 sec  
→ Intraventricular conduction

### **b) Normal Sinus Rhythm**

→ P Waves in lead I and lead II must be positive

c) Waveform Descriptions

	P wave	QRS complex	ST segment and T wave	U wave
1) Duration	< 0.12 sec	< 0.10 sec	Smoothly from ST segment to T wave	
2) Amplitude	< 2.5mm	Variable → Proximity of electrodes to the ventricle → Size of ventricle	?	< 1/3 of T wave In the same lead
3) Frontal plane axis (positive)	0 to 75°	90° to -30° → Most positive in lead II	Same direction as QRS → Except right precordial lead  Always upright in → Lead I,II, V3-6  Always inverted in → Lead aVR	Same as T wave In the same lead
4) Other features	-----	-----	-----	More prominent in slow heart rates  Best seen in right precordial leads

51) ECG on bradycardia

a) Sinus node disease (SAN)

- I) Sinus bradycardia:  
→ Rate < 60/min
- II) Sinus arrest  
→ Suddenly stop for several cycles
- III) Sinoatrial block  
→ 1<sup>st</sup> degree, 2<sup>nd</sup> degree, 3<sup>rd</sup> degree

**b) Atrioventricular node Disease (AVN)**

- I) **1<sup>st</sup> degree AV heart block**
  - ➔ PR interval > 0.2 sec
- II) **2<sup>nd</sup> degree AV heart block (Wenckebach)**
  - ➔ Progressive lengthening of PR interval until a QRS is dropped \*\*
- III) **2<sup>nd</sup> degree AV heart block (Mobitz II)**
  - ➔ Sudden QRS drop without prior PR interval lengthening
- IV) **3<sup>rd</sup> degree AV heart block**
  - ➔ Regular P wave and regular QRS complexes
    - BUT no relationship between them
    - Because no beats are conducted to the ventricles
  - ➔ \*\*Cannot increase heart beat although in exercise

**c) Bundle Branch block (RBBB / LBBB)**

- I) **RBBB**
  - ➔ M shape in V1 and V2
    - Late abnormal electrical vector block
- II) **LBBB**
  - ➔ Deepened and lengthen QRS in V1, V2, V3

52) ECG on tachycardia

**a) Supraventricular tachyarrhythmia**

- I) **Sinus tachycardia**
- II) **Atrial flutter (Atrial rate = 300/min)**
  - ➔ Regular Saw-toothed appearance \*\* (鋸齒型)
  - ➔ **Symptoms**
    - Fast palpitations, dizziness, heart failure
  - ➔ **Underlying heart disease**
    - Valves lesion
- III) **Atrial fibrillation (Atrial rate = 450 / min)**
  - ➔ Irregular saw-toothed appearance
  - ➔ Heart rate dependent on
    - 1) AV node conduction
    - 2) ANS

#### IV) Paroxysmal Supraventricular Tachycardia PSVT (間歇性, 室上性)

⇒ \*\*\*Inherited disease\*\*\*

##### I) AV nodal reentrant tachycardia (AVNRT)

→ Amplitude reduce, P = QRS

→ Mechanism

→ Recirculation of the impulse within the AV node

##### II) AV reentrant tachycardia (AVRT)

→ Wolff-Parkinson-White syndrome (WPW-AVRT)

→ If orthodromic (Bypass and normal pathway same direction)

→ VERY short PR interval + Delta wave following P

→ If antidromic (Opposite direction)

→

→ Mechanism

→ Recirculation of impulse involving AV accessory pathway

### b) Ventricular tachyarrhythmia

#### I) Ventricular tachycardia

→ If monomorphic (Come from 1 site)

→ 140-200bpm

→ Regular rhythm, only widened QRS complex

→ If polymorphic (Come from more than 1 sites)

→ 160-240bpm

→ Regular rhythm, only great increase in amplitude in QRS complex

#### II) Ventricular flutter

→ 240-320bpm

→ Regular QRS complex only

#### III) Ventricular fibrillation

→ Irregular QRS complex only

#### IV) Torsades de pointes

→ Only QRS complex, spinning around the baseline

→ Keep changing their axis and amplitude

**c) Ectopics (Premature contractions)**

- I) **Atrial:** P wave contour slightly different
- II) **Junctional:** Nearly Without P waves, start at AV nodes
- III) **Ventricular (Premature ventricular complexes)**
  - ➔ Inversion of T waves + Widened QRS complex
  - ➔ Monomorphic: unifocal
  - ➔ Polymorphic: multifocal
  - ➔ \*\*\*R on T phenomenon
  - ➔ **PVCs** falling on the T wave of the previous beat

**d) Summary of tachycardia**

⇒ **By comparing the rate of p and QRS**

- I) If p < QRS = junctional tachycardia
- II) If P = QRS = PSVT
- III) IF p > QRS = AF / Aflu, Irregular

⇒ **Irregularly irregular tachycardia**

➔ AF, Aflu with variable block, Multifocal atrial tachycardia

⇒ **Regularly irregular tachycardia**

➔ PSVT

**e) General management of arrhythmia**

- I) Pacemaker implant
  - ➔ Will give to spikes in ECG
- II) Drug treatment
- III) Radiofrequency ablation
- IV) Cardioversion

53) ECG on other conditions

**a) Acute myocardial infarction (AMI)**

- I) Q wave > 25% of R wave in amplitude
- II) ST segment elevation (2 leads and > 1mm)
- III) T wave inversion
- IV) Site
  - ➔ Anterior AMI: V1 – V4
  - ➔ Posterior AMI: tall R wave in V1
  - ➔ Lateral AMI: V5, V6, I, avL
  - ➔ Inferior AMI: II, III, avF

**b) Left Ventricular hypertrophy**

- I) S in V1 (**7 big squares**)
- II) R in V5 and V6 > 35mm (**7 big squares**)
- III) Left axis deviation

**c) Right Ventricular hypertrophy**

➔ **Caused by**

→ Pulmonary embolism, primary pulmonary hypertension / Valve stenosis

- I) Dominant R in V1 \*\*\*\*\*
- II) Right axis deviation

**d) Pulmonary Embolism**

➔ Right axis deviation + S1Q3 pattern

**e) Hypothermia**

➔ Osborne waves (J shape) \*\*

➔ Prolonged intervals

**f) Acute Pericarditis**

➔ Great ST elevation

**g) Hyperkalemia**

- I) Peaked T wave
- II) Prolonged PR interval, flat P waves
- III) QRS widening

**h) Hypokalemia**

- I) ST segment depression
- II) Flattening of T wave
- III) Appearance of a U wave

**i) Hypocalcemia**

➔ QT interval slightly prolonged

➔ A PVC falls on the prolonged T wave

→ Set for a run of torsades de pointes

**j) Digoxin Overdose**

➔ Reversed tick (Inversion of T wave)

54) CNS examination – Cranial Nerve

I) **Olfactory Nerve (1<sup>st</sup> nerve)**

→ Lay below the frontal lobe

→ If dysfunction, can lead to frontal lobe depression

→ Causing low limb dysfunction

→ **Examination**

→ Each nostril individually

→ Use **aromatic** but **no irritant agents**

II) **Optic nerve (2<sup>nd</sup> nerve)**

→ Quantitative testing

→ 20 / 60 = normally can see 60 feet away, but now only 20

→ Test for

1) **Visual acuity**

→ Eye by eye counting fingers

2) **Visual field**

→ Eye by eye

→ Confrontation test (by comparing the field between patient and examiner)

a) **Total dysfunction**

→ Defect in optic nerve

b) **Lateral half in both eyes**

→ Defect in the junction of 2 optic nerves (Optic chiasm)

c) **Lateral half of one and medial half of another**

→ Defect in optic tract

→ **Homonymous hemianopsia**

d) **Quarter of both eyes**

→ Defect in near optic radiation

e) **Lateral half of one and medial half of another + central dots**

→ Defect

3) **Direct light reflex**

→ If shine to left eye, left constrict

4) **Consensual light reflex**

→ When shine to left eye, both pupils should constrict

5) **Accommodation reflex**

→ Accommodate looking at distant and near object

6) **Fundus**

**III) Ocular movements (Oculomotor, Trochlear, Abducent 3,4,6)**

→ H test \*\*

→ Examine 6 muscles

→ 1 hand length

→ Ask the patient to follow your finger

**IV) Trigeminal Nerve (5<sup>th</sup> nerve)**

1) Motor function

→ Muscle of mastication

2) Sensory Division

1) **Ophthalmic division**

→ Forehead, nose, upper eyelid

2) **Maxillary division**

→ Cheek, lower eyelid, upper lip (Cheek)

3) **Mandibular division**

→ Lower lip, lower face and jaw (Chin)

3) Corresponding Reflex

→ Jaw jerk

→ Corneal reflex

→ Afferent – Ophthalmic division of V

→ Efferent – Facial Nerve (VII)

**V) Facial (VII)**

1) Muscles of facial expression

I) Frowning (Upper face)

→ **Bilateral innervation \*\*\***

→ So, if upper motor neuron lesion,

→ Only contralateral lower face is affected

II) Blowing cheek (Lower face)

→ **Only unilateral innervation \*\*\***

→ So, if lower motor neuron lesion,

→ Whole of ipsilateral face weak

2) Taste of anterior 2/3 of tongue

3) Salivary secretion

\*\*Commonly, block by\*\*

→ Parotid gland tumour

→ Trauma in the face under ear lobe

## **VI) Vestibulocochlear Nerve (Deafness) (VIII)**

⇒ **Rinne's Test \*\***

→ Normal Air conduction >> Bone conduction

- I) Put the tuning folk onto mastoid until no sound is heard
- II) If you hear by normal hearing afterwards, then it is proved that air >>bone

⇒ **Weber's test \*\***

→ Put tuning folk onto the top of cranial

→ The defected side will hear soft sounds

→ EXCEPTION \*\*

→ Conductive Deafness will hear louder sound in the defected side

→ Absence of noises

### **a) Conductive Deafness**

→ Sound not transmitted to cochlear

→ Bone conduction >> Air conduction

### **b) Sensorineural Deafness**

→ Sound Transmitted to cochlear

→ Both diminished

## **VII) Glossopharyngeal nerve (IX)**

1) Position and movement of soft palate and uvula

→ **Gag reflex (Afferent part)**

→ By touching the back of tongue, initiating the swallowing reflex

2) Taste and sensation of posterior 3<sup>rd</sup> of tongue

## **VIII) Vagus nerve (X)**

→ Autonomic

→ E.g. Heart beat

→ E>g. GI motility

→ **Gag reflex**

→ Efferent part

→ Pharynx, larynx

→ Motor and sensory

**IX) Accessory nerve (XI)**

**1) Sternocleidomastoid muscle**

→ Rotate head to the opposite side

**2) Trapezius muscle**

→ Shrug shoulders against resistant

**X) Hypoglossal Nerve (XII)**

→ Motor to intrinsic and extrinsic tongue muscle

→ Test for

- I) Atrophy
- II) Fibrillation
- III) Deviate on protrusion
- IV) Tongue movement
- V) Power

\*\*\*\*E.g. Right XII pulsy\*\*\*\*

55) CNS examination – Upper limb

a) **Motor System**

**I) Appearance**

- Symmetry, Deformity
- Muscle wasting / Hypertrophy
- Muscle fasciculation

**II) Muscle Tone**

- Increased tone
  - Cog-wheel type
  - Lead-pipe type
  - Clasp-knife type
- Decreased tone

### **III) Muscle Power**

- 1) Serratus anterior** (Looking for winging of scapula)  
→C5,C6,C7 root, long thoracic nerve
- 2) Shoulder abduction**  
→Deltoid, C5-C6 root, Axillary nerve
- 3) Elbow**  
→**Extension:** Triceps, C6,C7,C8, Radial nerve  
→**Flexion:** Biceps, C5, C6, Radial + Musculocutaneous nerve
- 4) Finger**  
→**Extension:** Extensor digitorum, C7,C8, Posterior interosseous ner.  
→**Flexion::** Flexor digitorium profundus, C7, C8  
→2-3 finger: Median nerve  
→4-5 finger: Ulnar nerve  
→**Abduction:** C8 T1, Ulnar nerve
- 5) Thumb**  
→**Extension:** C7, C8, posterior interosseous nerve  
→**Opposition:** C8 T1, Median nerve

### **b) Reflex System**

- I) Bicep Jerk:**  
→C5, C6, Musculocutaneous nerve  
→Palpate and strike the bicep tendon with tendon hammer  
→Bicep contraction and elbow flexion
- II) Tricep jerk**  
→C6,C7,C8 Radial nerve  
→Strike a few inches above the olecranon process  
→Tricep contraction and elbow extension
- III) Supinator Jerk**  
→C6, C7, Radial nerve  
→Strike lower end of radius  
→Elbow and finger flexion
- IV) Hoffman reflex**  
→C7,C8, Flick the terminal phalanx  
→Thumb flexion indicates hyperreflexia

c) **Sensory System**

- ⇒ Consider root or peripheral nerve
  - I) **Sharp pain (Pin prick)**
  - II) **Dull pain (Pin prick)**
  - III) **Temperature**

56) CNS examination – Lower limb

a) **Motor System**

- I) **Appearance (same)**
- II) **Muscle Tone (same)**
  - Alternatively flexing and extending the knee and hip
  - Rolling from side to side
- III) **Muscle Power**
  - 1) **Hip**
    - **Flexion:** L1, L2, L3, Femoral nerve, ilio-psoas
    - **Extension:** L5, S1, S2, Inferior gluteal nerve, Gluteus maximus
    - **Abduction:** L4, L5, S1, Superior, gluteal nerve
      - Gluteus minimus, medius and tensor fasciae latae
    - **Adduction:** L2, L3, L4, Obturator nerve, Adductors
  - 2) **Knee**
    - **Flexion:** L5, S1, S2, Sciatic Nerve, Hamstring
    - **Extension:** L2, L3, L4, Femoral Nerve, Quadriceps
  - 3) **Ankle**
    - **Dorsiflexion:** L4, L5, Deep peroneal nerve, Tibialis anterior
    - **Plantarflexion:** S1, S2, Tibial nerve, Gastrocnemius, soleus
  - 4) **Toe**
    - **Extension:** L4, L5, Tibial nerve, Tibialis posterior
    - **Inversion:** L4, L5, Tibial nerve, Tibialis posterior
    - **Eversion:** L5, S1, Superficial peroneal nerve
      - Peroneus longus and brevis

**b) Reflex System**

- I) Knee jerk:
- II) Ankle jerk
- III) Plantar response
  - ➔ Babinski sign

**c) Sensory System**

⇒ Consider root or peripheral nerve

- IV) Sharp pain (Pin prick)
- V) Dull pain (Pin prick)
- VI) Temperature