



**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009

1.2.2

Add-on Certificates Details with Report

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Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
Dean

Velammal Medical College Hospital  
and Research Institute  
"Velammal Village"  
Madurai-Tuticorin Ring Road  
Anuppanadi, Madurai-625 009, T.N.



**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009

Department of Anatomy

Velammal Medical College Hospital And Research Institute, Madurai

**Report**

**Topic-** Basic Genetics For Undergraduates

**Date –** 18<sup>th</sup> October 2017

**Time –** 8am – 4pm

**Duration-** 8 Hours

**Venue-** Department Of Anatomy

**Organizer Of Program-** Dr. P Sasikala (Professor & Hod)

**Program Coordinator-** Dr. Parineeta Suman

**Target Audience-** 1<sup>st</sup> Mbbs Students

**Total Registered Participants-** 145

**Total Students Speaker-**

**Participated-** 150

Dr. Parineeta Suman

Mr. David Ebenezer

**Mode of Teaching -** Classroom



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**Basic Genetics for Undergraduates**

1.	Chromosomes	8-9am	Dr. Parineeta Suman
2.	Karyotyping	9-10am	Dr. Parineeta Suman
3.	Chromosomal Abnormality	10.15-11.15am	Mr. David Ebenezer
4.	Genes and genetic codes	11.15-1pm	Dr. Parineeta Suman
5.	Pedigree chart	2-3 pm	Mr. David Ebenezer
6.	Genetic counselling	3-4pm	Dr. Parineeta Suman

  
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# VELAMMAL MEDICAL COLLEGE HOSPITAL AND RESEARCH INSTITUTE MADURAI - 625009

Velammal Medical College Hospital & Research Institute  
**DEPARTMENT OF ANATOMY**  
**CERTIFICATE COURSE: BASIC GENETICS FOR UNDERGRADUATES**  
Speakers:  
Dr. Parineeta Suman  
Associate Professor of Anatomy  
Mr. David Ebenezer  
Assistant Professor of Anatomy  
DATE - 18.10.2017  
1<sup>st</sup> MBBS Students 2017-18 Batch

Vellammal medical college, madurai  
chinthamani.  
Department of Anatomy, VMCH&RI  
BASIC genetics for under graduates  
2017-10-18(Wed) 10:13(am)  
29°C  
84°F

Genetic counseling:

- Genetic counseling is the process of advising individuals and families affected by or at risk of genetic disorders to help them understand and adapt to the medical, psychological and familial implications of genetic contributions to disease.
- Sheldon Reed proposed the terminology "Genetic Counseling" in 1947.



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Example of Pedigree Charts

Is it Autosomal or X-linked?

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Samanatham  
Kammal Kovil  
Ayya  
Rana  
Google

FLOW OF INFORMATION: GENE → PROTEIN

The Central Dogma of Molecular Biology

Replication → DNA → Transcription → RNA → Translation → Protein

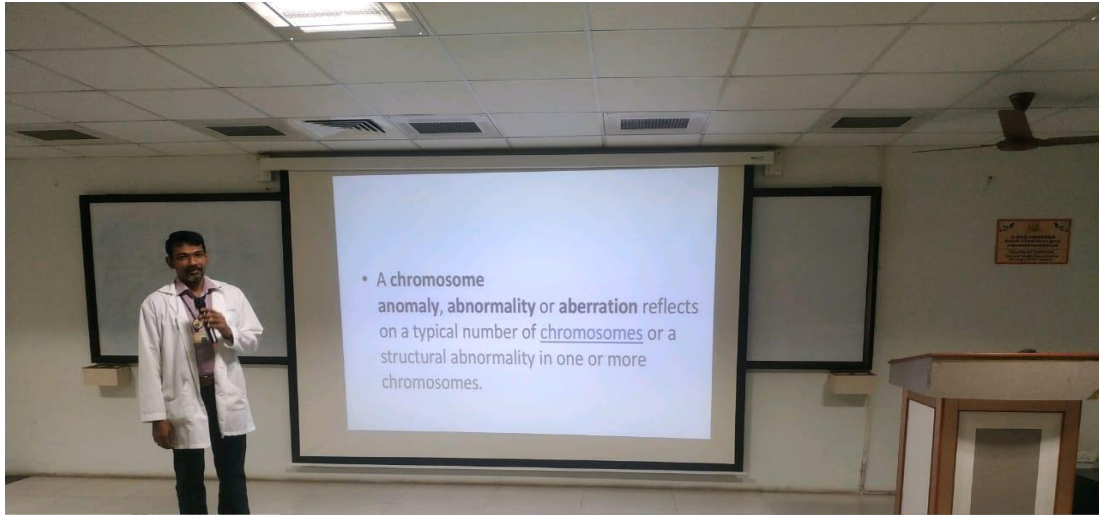
Proteins do most of the work in the cell.

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• A chromosome anomaly, abnormality or aberration reflects on a typical number of chromosomes or a structural abnormality in one or more chromosomes.



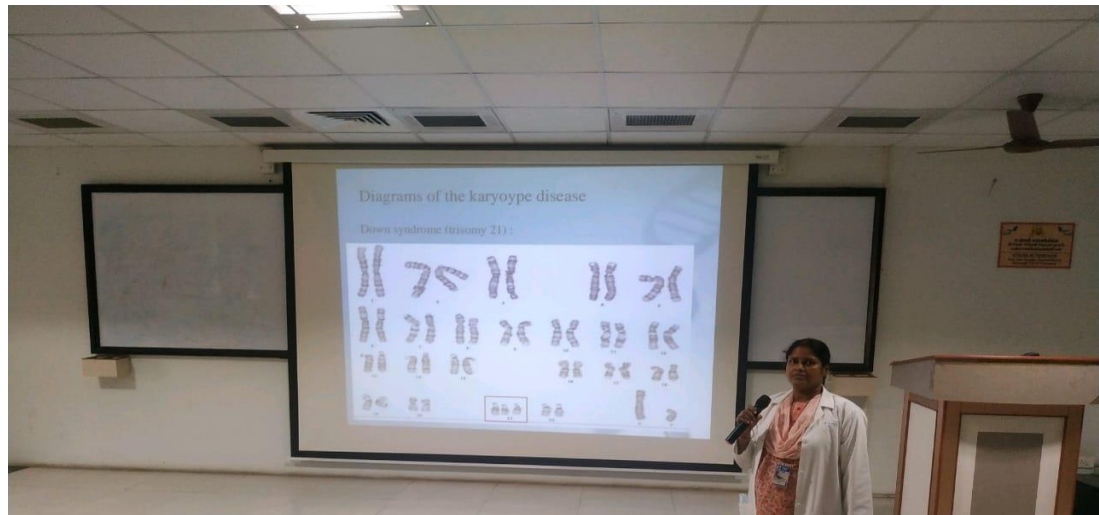
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Department of Anatomy, VMCH&RI  
BASIC genetics for under graduates

2017-10-18(Wed) 10:15(am)

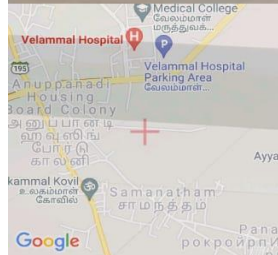
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Diagrams of the karyotype disease

Down syndrome (trisomy 21):



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**Types of chromosome**

- According to the relative position of centromere chromosomes are divided into four types

Centromere Localizations

metacentric    sub-metacentric    acrocentric    telocentric

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## Certificate Sample

Velammal Medical College Hospital and Research Institute, Madurai, TN




# CERTIFICATE Of Participation

THIS CERTIFICATE IS PRESENTED TO

**NAME**

For participating in the certificate course on “**Basic genetics for undergraduates**” conducted on 18 October 2017 by Department of Anatomy

  
Dr. Parineeta Suman  
Coordinator

  
Dr. P Sasikala  
Head of Department



**VELAMMAL MEDICAL COLLEGE**  
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Department of Anatomy

**Report of Certificate Course**

**Topic-** Histotechniques

**Date** – 23<sup>rd</sup> January 2018

**Time** – 8am – 4pm

**Duration-** 8 Hours

**Venue-** Department Of Anatomy

**Organizer Of Program-** Dr. Sumana R (Professor & Hod)

**Program Coordinator-** Dr. Parineeta Suman

**Target Audience-** 1<sup>st</sup> Mbbs Students

**Total Registered Participants-** 150

**Total Students Participated-** 149


**Speaker-**

Mr. David Ebenezer

Mr. M Ram Kumar

**Mode of Teaching** - Classroom

1.	Introduction	8-9am	Mr David Ebenezer
2.	Types of Histotechniques	9-10am	Mr. Ram Kumar
3.	Collection of tissue	10.15-11.15am	Mr David Ebenezer
4.	Sectioning and mounting	11.15-1pm	Dr. Parineeta Suman
5.	Staining and fixing	2-3 pm	Mr David Ebenezer
6.	Frozen section and decalcification	3-4pm	Mr. Ram Kumar

  
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**HISTOTECHNIQUES**

- Paraffin embedding technique
- Parlodion embedding technique
- Exfoliative cytology
- Fine-needle aspiration cytology
- Fine-needle aspiration biopsy
- Ground section
- Frozen section
- Immunohistochemistry (or IHC)
- Fluorescence technique
- Tissue microarray
- Molecular techniques

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**HISTOTECHNIQUES**  
2018-01-23(Tue) 09:30(am)

**COMMON FIXING AGENTS**

- Numerous substances or chemicals are used singly or in combination:
  - have ability to preserve tissue
  - allow demonstration of every tissue component.

Organic fixative	Inorganic fixative
Alcohol	Formal
Acetone	Osmium tetroxide
Acetic acid	Potassium dichromate
Mercuric Chloride (formalin solution)	Glutaraldehyde
Platin acid	
Trichloroacetic acid	

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**HISTOTECHNIQUES**  
2018-01-23(Tue) 11:05(am)



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**Certificate sample-**





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VMCH/Physio/CC/2017

**Circular**

**To First MBBS students of 2017-18 batch**

**Date: 1.12.2017**

There will be a certificate course on **Aging** for first MBBS students from 9am to 4pm on Friday, 8-12-2017. All the students are expected to attend the course without fail.

**Date: Friday, 8<sup>th</sup> December 2017**

**Time: 9am to 4pm**

**Venue: Lecture Hall 1 First floor**

Dr.S.Anu

Professor and Head

Department of Physiology

Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
Dean

Velammal Medical College Hospital  
and Research Institute  
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Departments of Physiology  
Velammal Medical College Hospital and RI

Madurai

invite you for

*Certificate course in*

**AGING**

PATRON

**Shri.M.V.Muthuramalingam**, Chairman, Velammal Educational Trust

ADVISORS

**Dr.R.M.Raja Muthaiah**  
Dean

**Dr.P.K.Mohanty**  
Vice Principal

Organising Secretary  
**Dr.S.Anu**, Professor and Head,  
Department of Physiology

8 December 2017 • 9:00 am onwards

**For First MBBS Students of 2017-18 batch**

Venue

Lecture Hall 1, Velammal Medical College

## Course Objective

To help the students to discover the science behind the ageing process and to give them an overall picture of Physiology, Pathophysiology and graceful aging process through scientific lectures.

## AGENDA

9.00 am	Pretest
9.30 am	Introduction By <b>Dr.S.Anu</b> , Professor and Head, Physiology
10.00 am	Physiological changes in aging By <b>Dr.M.Saravanan</b> , Associate Professor
10.45 am	Break
11.00 am	Theories of aging By <b>Dr.M.Shanthi</b> , Professor
12 noon	Pathophysiology of aging By <b>Dr.A.S.Kanietha Priya</b> , Assistant Professor
1 pm	Lunch break
2 pm	Graceful aging By <b>Dr.Keba</b> , Assistant Professor
3 pm	Stress and aging By <b>Dr.K.Rekha</b> , Assistant Professor
3.30 pm	Vote of thanks By <b>Dr.John Rajpathy</b> , Professor
4 pm	Post test

## Physiology of Aging

### Ageing

- a natural process
- Biological ageing  $\neq$  chronological age
- Characterised by a gradual and progressive constriction of the homeostatic reserve of every organ system
- Rate and extent varies with the individual
- Influenced by genetic, diet, environmental factors and personal habits
- Gerontology : science of aging
- Geriatric medicine: specialized form of medicine dealing with aging related illness

### AGE RELATED CHANGES IN THE DIFFERENT SYSTEMS

#### CARDIOVASCULAR SYSTEM

- Deposition of lipofuscin pigments and amyloid
- Capillary density is decreased
- Valves thickened  $\rightarrow$  mitral valve may be slightly incompetent
- Heart rate and cardiac output response to exercise is decreased
- Baroreceptor sensitivity is decreased therefore, impaired response to standing and volume depletion
- Decrease in the elastic fibres in the blood vessels
- Deposition of calcium salts in the major branches
- Systolic pressure is raised
- Blood flow to the organs may be decreased
- Distensibility of the blood vessels decreased

#### CHANGES IN RESPIRATORY SYSTEM

- Alveoli become flatter and shallow
- Loss of inter alveolar septa leading to decrease in the number of alveoli
- Compliance of thoracic cage and lungs decreased
- increased wall thickness of the capillaries
- FRC increases by 50%; Residual volume increases by 100%
- Airways are susceptible to collapse
- Response to hypoxia and hypercapnia is decreased
- There is an increase in physiological dead space
- Bronchiolar escalator function decreased

#### CHANGES IN GIT

- Masticatory efficiency decreased
- Weakness of pharyngeal musculature  $\rightarrow$  difficulty in swallowing
- Disordered oesophageal motility at the lower end
- Reduction in gastric acid secretion above 60 years of age
- Reduction in villus height and reduction in the lactase activity in the brush border
- Decrease in number and increase in the size of the hepatocytes
- Hepatic metabolism of drugs and steroid are reduced
- Decrease in albumin globulin ratio
- Colon motility decreased resulting in constipation

#### CHANGES IN RENAL SYSTEM

- Decrease in the weight of the kidneys
- Decrease in the number of the neurons
- Decrease in the GFR by 30 to 40% at the age of 80 years
- Impaired excretion of drugs may be seen

  
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decrease in tubular functions; renal functions become borderline

#### **CHANGES IN GENITOURINARY SYSTEM**

Prostatic enlargement may be seen

Urinary incontinence or urinary retention may be present

Menopause in women

Mucosal atrophy in urethra and vagina leading to dyspareunia and infections

#### **CHANGES IN ENDOCRINE SYSTEM**

Thyroid hormone secretion decreases

Impaired glucose homeostasis due to diminished sensitivity to insulin

Postprandial sugar increases

Plasma level of oestrogen and progesterone decreases

FSH and LH levels are increased due to negative feed back by the decreased levels of oestrogen and progesterone

ADH and Aldosterone levels decreases

Vit. D absorption and activation decreased

#### **CHANGES IN BLOOD AND IMMUNE SYSTEM**

Blood volume and blood cells are not changed significantly

Haemopoietic reserve is gradually decreased

Anaemia may occur in elderly

Senile purpura may be present

Raised ESR due to increased level of fibrinogen

Reduced immunosurveillance may lead to malignant conditions

#### **CHANGES IN MUSCULOSKELETAL SYSTEM**

Muscular power decreases

Loss of muscle fibres is present

Prolongation of latency of muscle contraction

Degenerative changes in bones

Osteoporosis may present

Changes in the vertebral column results in change stature and posture

#### **CHANGES IN SKIN AND HAIR**

Wrinkling of skin

Due to decreased elasticity and thinning of epidermis decreased subcutaneous fat

Greying of hair due to loss of melanin

Baldness in males; Loss of axillary and pubic hair

Increase in facial hair growth in females due to unopposed action of adrenal androgens

Sweating and sebaceous secretion decreased

#### **CHANGES IN CENTRAL NERVOUS SYSTEM**

At the age of 70 years, 45% cells lost in cerebrum and 25% loss in cerebellum

Degenerative changes in substantia nigra

Accumulation of lipofuscin granules in neurons and glial cells

Cerebral blood flow decreased by 40% at 70 years and oxygen utilization is reduced by 25%

Functions of neurotransmitters reduced

Dopaminergic deficit in parkinsonism

Cholinergic defect in senile dementia, alzheimer's disease

Catecholamine decrease → depression in old age

Hypokinesia due to dopaminergic defect

Reflexes are diminished

Decreased righting reflexes leads to swaying

  
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III and IV stage of NREM sleep reduced have early awakening

#### **CHANGES IN AUTONOMIC NERVOUS SYSTEM**

Impaired temperature regulation

Postural hypotension may be present

#### **CHANGES IN SPECIAL SENSES**

Presbyopia due to inefficiency of accommodation

Age related cataract

Age related corneal degeneration opacity near the limbus called arcus senilis

Age related macular degeneration

Dry eyes due to decreased tear secretion

Presbycusis due to degenerative changes in the organ of corti

Otosclerosis → decreased motility in ear ossicles → impaired hearing

Impairment of postural reflexes

Decrease in the number of taste buds → impairment in taste sensation

Impairment in smell sensation due to decrease in the smell receptors

#### **THEORIES OF AGEING**

Genetic theory of ageing

Random damage theory

#### **GENETIC THEORIES OF AGEING**

Programmed senescence theory

Mutation theory

Theory of pleiotropic antagonism

Theory of ecological niches

The error catastrophe theory

Intrinsic mutagenesis theory

Somatic mutation theory

#### **RANDOM THEORY OF AGEING**

Based on the balance between ongoing damage and repair is disrupted

Free radical theory

Cell replication theory

Cross linkage theory

#### **PREVENTION OF AGING**

Calorie restriction

Balanced diet

Regular physical exercise

Mental and physical relaxations

No smoking

Avoidance of excess of alcohol

Practice of asanas and breathing exercises

Spiritual life

9.202

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M.D., F.R.C.S. (Ed.)  
M.D., F.R.C.S. (Surg.)  
M.D., F.R.C.S. (Gen.)  
M.D., F.R.C.S. (Orth.)  
M.D., F.R.C.S. (Plast.)  
M.D., F.R.C.S. (Neuro.)  
M.D., F.R.C.S. (ENT)  
M.D., F.R.C.S. (Ophth.)  
M.D., F.R.C.S. (Paed.)  
M.D., F.R.C.S. (Geriatr.)  
M.D., F.R.C.S. (Radiol.)  
M.D., F.R.C.S. (Pathol.)  
M.D., F.R.C.S. (Microbiol.)  
M.D., F.R.C.S. (Pharmacol.)  
M.D., F.R.C.S. (Anesth.)  
M.D., F.R.C.S. (Dent.)  
M.D., F.R.C.S. (Histopathol.)  
M.D., F.R.C.S. (Cytopathol.)  
M.D., F.R.C.S. (Immunol.)  
M.D., F.R.C.S. (Infect.)  
M.D., F.R.C.S. (Nephrol.)  
M.D., F.R.C.S. (Oncol.)  
M.D., F.R.C.S. (Palliat.)  
M.D., F.R.C.S. (Rehabil.)  
M.D., F.R.C.S. (Sports Med.)  
M.D., F.R.C.S. (Travel Med.)  
M.D., F.R.C.S. (Vaccinology)

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Madurai, Madurai Ring Road  
Anuppana, Madurai, Tamil Nadu, India.

**Department of Physiology**  
**Velammal Medical College Hospital and Research Institute**  
**Madurai**  
**Report**

**Topic:** Certificate course on AGING  
**Date:** 8-12-2017  
**Venue:** Lecture Hall 1, Velammal Medical College  
**Target Audience:** First MBBS students 2017-18 batch  
**Number of participants:** 143


**Report:**

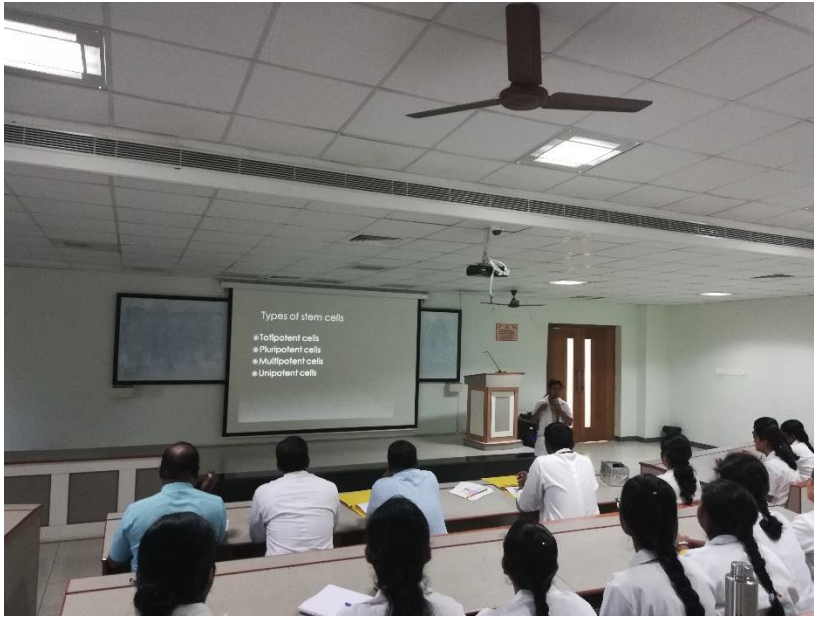
A certificate course on Aging was organised by Department of Physiology to first MBBS students on 8.12.2017. 143 first MBBS students participated in the course. The program began by 9am with pretest followed by scientific lectures on Introduction, Physiological changes in aging, Theories of aging, Pathophysiology of aging, Graceful aging, Stress and aging. The program concluded with vote of thanks. Pretest and posttest were conducted to sensitise the students with topic content and grade their knowledge gain of the course.

**Outcome:**

Students learnt about the concepts of aging, physiology and pathophysiology behind aging and learnt about the ways of delaying aging within physiological limits.

**Photos:**

  
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**Department of Physiology**  
**Velammal Medical College Hospital and Research Institute**  
**Madurai**

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VMCH/Physio/CC/2018/01

**CIRCULAR**

To First MBBS students of 2017-18 batch

30.3.2018

There will be a certificate course on **Learning and Memory** for first MBBS students from 9am to 4pm on Friday, 6-4-2018. All the first year students are expected to attend the course without fail.

Date: Friday, 6<sup>th</sup> April 2018

Time: 9am to 4pm

Venue: Lecture Hall 1

First floor

Velammal Medical College

Dr.S.Anu

Professor and Head  
Department of Physiology



Departments of Physiology  
Velammal Medical College Hospital and RI

Madurai

invite you for

*Certificate course in*

# **LEARNING AND MEMORY**

PATRON

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**Dr.R.M.Raja Muthaiah**  
Dean

**Dr.P.K.Mohanty**  
Vice Principal

Organising Secretary  
**Dr.S.Anu**, Professor and Head,  
Department of Physiology

6 April 2018 • 9:00 am onwards

**For First MBBS Students of 2017-18 batch**

Venue

Lecture Hall 1, Velammal Medical College

## Course Objective

To enlighten the first MBBS students about the physiological basis of human learning and memory & introduce them to applied aspects involved in learning and memory mechanism.

## AGENDA

9.00 am	Pretest
9.15 am	Introduction By <b>Dr.S.Anu</b> , Professor and Head, Physiology
9.45 am	Definition and types of Memory By <b>Dr.M.Shanthi</b> , Professor
10.45 am	Break
11.00 am	CNS circuits of learning and memory By <b>Dr.M.Saravanan</b> , Associate Professor
12 noon	Evidences of learning and memory By <b>Dr.K.Rekha</b> , Assistant Professor
1 pm	Lunch break
2 pm	Drugs acting on learning and memory By <b>Dr.Keba</b> , Assistant Professor
2.45 pm	Applied aspects By <b>Dr.A.S.Kanietha Priya</b> , Assistant Professor
3.30 pm	Vote of thanks By <b>Dr.John Rajpathy</b> , Professor
3.45 pm	Post test

## Learning and memory

### Definition of Memory

Learning: acquisition of the information that makes the ability to alter behavior on the basis of experience.

Memory is the retention and storage of that information.

### Types of Memory

2 ways of classification:

1. On the basis of Permanency of storage:
  - a. Working / Primary memory
  - b. Recent / Short term/ Secondary Memory
  - c. Remote / Long term /Tertiary Memory
2. On the basis of storage & recollection of Information:
  - a. Explicit / Declarative Memory
  - b. Implicit / Non-Declarative / Reflex memory

### Explicit / Declarative Memory

- Factual knowledge of People, Places & Things
- Recalled by deliberate conscious effort
- Highly flexible & involves the association of multiple bits & pieces of information.

### Types of Explicit Memory

Semantic Memory (Memory of Facts)

Episodic Memory (Memory for events & Personal experience)

1. Semantic Memory (Memory of Facts):
  - Naming of objects
  - Definition of spoken words
  - Verbal fluency

Semantic memory is stored in a distributed fashion in different association areas

Semantic Memory:

1. Visual Memory
  2. Auditory Memory
  3. Somatosensory Memory
2. Episodic (Autobiographic) memory:
    - Association area of Pre-Frontal Cortex
    - Defect ----- Source amnesia

  
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## **Mechanism of Declarative Memory:**

Result of 4 related but distinct types of Processing:

1. Encoding
2. Consolidation
3. Storage
4. Retrieval

Declarative Memory Involves:

Short term

Intermediate long term

Long term

### **1. Short term:**

The incoming information should be encoded thoroughly & deeply for memory.

Encoding occurs in Hippocampus.

Mechanism of Short term Declarative Memory:

Involves Hippocampus & neighbouring cortex like Entorhinal, Perirhinal & Para hippocampal areas

Input & Output of Hippocampus:

Unimodal & Polymodal association areas

Parahippocampal Cortex, Perirhinal Cortex, Entorhinal Cortex

Dentate gyrus, Hippocampus, Subiculum

### **2. Intermediate long term Memory:**

- Few mins to Weeks
- Temporary chemical or physical changes in the pre-synaptic or post synaptic membrane.

Physiological process studied in the aplysia as follows,

Facilitatory Terminal    Serotonin

Activates adenyl cyclase

Cyclic -AMP

Release of Protein Kinase

Phosphorylation of Protein


Blocks K<sup>+</sup> Channel

Neurotransmitter release

facilitates transmission at synapse

### **3. Long Term Memory:**

Needs Consolidation

  
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and Research Institute  
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(CONSOLIDATION – CONVERSION OF NEWLY STORED LABILE INFORMATION TO STABLE INFORMATION)

5 – 10 Mins - Minimal Consolidation

> 1 hr - Strong Consolidation

Absence of Consolidation – Retrograde Amnesia

Rehearsal Mechanism needed for Consolidation

**Process of Consolidation:**

Expression of Genes

Synthesis of new Proteins

Structural Changes

**Structural Changes:**

1. no. of Synaptic Terminals

2. no. of Synaptic Vesicles

3. Change in Shape & number of Post Synaptic Spines.

Neural Substrate for Long term Memory:

Various parts of neocortex.

Retrieval of Memory:

Recall & use of stored memory

Working Memory:

components:

1. Attention control system

2. Rehearsal System:

- Articulatory system

- Visuospatial Sketch Pad.

1. Attention control system:

Pre – frontal cortex

2. Articulatory system:

Memory for words & numbers

3. Visuospatial Sketch Pad:

Visual properties & Spatial location of the object.

**Evidence for Declarative Memory encoding**


1. Bilateral destruction of Ventral Hippocampus :

- Defects in recent memory with intact remote memory.

- Cannot form new long term memory.

2. Stimulation of Hippocampus with electrodes – Seizures with loss of recent memory

3. Chronic alcoholics with brain damage –

  
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Dean  
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Madurai-Tuticorin Ring Road  
Anuppr...

Impairment in the recent memory with changes in the mammillary bodies.

Implicit Memory:

- How to perform something
  - Builds up slowly
- ex : Driving car

Acquired through reflexive learning.

1. Non-associative learning:

- Habituation
- Sensitization

2. Associative Learning:

- Classical Conditioning
- Operant Conditioning

Mechanism of Implicit Memory:

Habituation:

Pre-Synaptic depression of Synaptic transmission.

Sensitization:

Pre-Synaptic facilitation of Synaptic transmission.

Classical Conditioning:

Pre- Synaptic facilitation that depends on activity in both pre & post synaptic cell.

### Physiology of Memory

Based on Clinical & Experimental observations:

1. Temporal lobe

Temporal lobe points are the key to unlock the memories stored in other parts of the brain.

Interhemispheric Transfer of Memory

Drugs facilitating Memory

Various CNS stimulants can improve learning when administered immediately before or after the learning session.

- By facilitating consolidation of memory.
  - Caffeine
  - Nicotine – stimulates Cholinergic receptor
  - Physostigmine
  - Amphetamine
  - Pemoline

### Applied Physiology

1. Amnesia
2. Alzheimer's Disease

  
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Anuppanadi, Madurai-625 009, T.N.

### 3. Senile Dementia

#### AMNESIA

1. Antegrade amnesia: inability to form new long term memories, occurs in lesions of hippocampus.
2. Retro grade amnesia: Inability to recall past memories, occurs in lesions involving temporal lobe

#### Alzheimer's Disease:

Progressive loss of memory and cognitive function in middle aged individual

  
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Anuppanadi, Madurai-625 009, T.N.

**Department of Physiology  
Velammal Medical College Hospital and Research Institute  
Madurai  
Report**

**Topic:** Certificate course on Learning and Memory  
**Date:** 6-4-2018  
**Venue:** Lecture Hall 1, Velammal Medical College  
**Target Audience:** First MBBS students 2017-18 batch  
**Number of participants:** 141


**Report:**

A certificate course on Learning and Memory was organised by Department of Physiology to first MBBS students on 6.4.2018. 141 first MBBS students participated in the course. The program began by 9am with pretest followed by scientific lectures on Introduction, Definition and types of Memory, CNS circuits of learning and memory, Evidences of learning and memory, Drugs acting on learning and memory and Applied aspects. The program concluded with vote of thanks. Pretest and posttest were conducted to sensitise the students with topic content and grade their knowledge gain of the course.

**Outcome:**

Students learnt about the concepts of learning and memory. The gained idea about physiology, pathophysiology of applied aspects related to human learning and memory.

**Photos:**

  
**Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
Dean  
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and Research Institute  
"Velammal Village"  
Madurai-Tuticorin Ring Road  
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**Velammal Medical College Hospital and Research Institute**

Ref. No: VMCHRI/BIOCHEM/CC-1

Date: 02.11.2017

**CIRCULAR**

To

All Doctors

**Certificate Course on POCT (Point of care testing)**

Department of Biochemistry is organizing a certificate course on POCT (Point of care testing) on 30.11.2017 (Thursday) between at 9.00 AM to 3.00 PM.

All Faculties are invited.

Copy submitted to:

The Hon. Chairman

Copy to:

The Dean

Medical Superintendent

Chief Administration Officer

HOD, Biochemistry

All Clinical and Non-Clinical HODs

**VICE PRINCIPAL**

**Dr. P.K. MOHANTY**

Vice Principal

Velammal Medical College Hospital  
and Research Institute

Madurai-625 009



**Velammal Medical College Hospital &  
Research Institute**  
Anuppanadi, Madurai - 625009

**Department of Biochemistry**

**Certificate course on**

**POCT (Point of care testing)**

**Venue: Biochemistry Demonstration Room**

**Date: 30.11.2017**

**For First year M.B.B.S., students**



**PATRON**

**Chairman:** Shri.M.V.Muthuramalingam

**Advisors:**

**Dean:** Dr.R.M. Raja Muthaiah

**MS:** Dr. Somasundarm

DR.A.Hariharan  
**Course coordinator**  
**Biochemistry**

DR.P.K.Mohanty  
**Vice Principal**  
**Prof. HOD Biochemistry**

For further detail contact:

DR.A.Hariharan

Mobile No. 97898784256

## Agenda

<b>Time</b>	<b>Topic</b>	<b>Speaker</b>
09.00 – 09.15 AM	Welcome address	DR.P.K.Mohanty
09.15 - 09.45 AM	Pre test	DR.A.Hariharan
09.45 – 11.00 AM	POCT – Basics	DR.A.Hariharan
11.00 AM – 1.00 PM	Demonstration: POCT	DR.A.Hariharan & Resource persons
1.00- 2.00 PM	LUNCH	
2.00- 2.30 PM	Post test	DR.A.Hariharan
2.30- 3.00 PM	Feedback and Valediction	DR.P.K.Mohanty

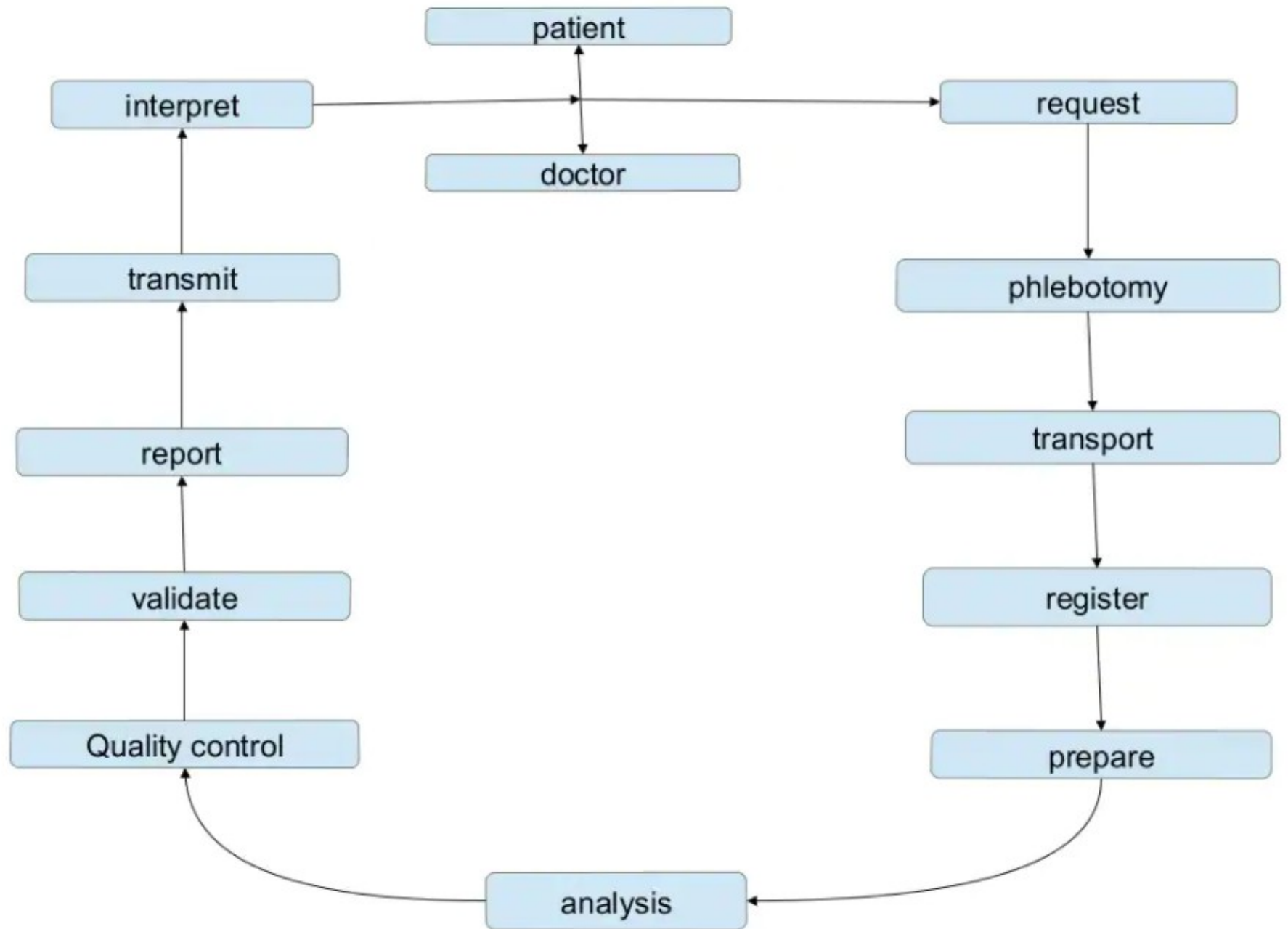
# POINT OF CARE TESTING (POCT)

-DR.A.Hariharan  
M.D, (Biochemistry)



# Definition

When a test is performed at a time at which test results enables a decision to be made and an action taken. That leads to an improved health outcome.



# Advantages

Reduced turn around time

Improved patient morbidity & mortality  
Reduction in hospital admission

Improved interaction between patient  
and carer

Reduction in clinical visits

Improved cost of care

Reduction in administrative work associated  
With test requesting & reporting



# Disadvantages

Increase in administrative work associated with training and Certification of operators

Caregivers required to perform test

Increased risk of errors

```
graph TD; A[Caregivers required to perform test] --> B[Increased risk of errors]
```

The diagram consists of three light blue rounded rectangular boxes. The top box contains the text 'Increase in administrative work associated with training and Certification of operators'. The middle box contains 'Caregivers required to perform test'. The bottom box contains 'Increased risk of errors'. A curved arrow points from the bottom of the middle box to the top of the bottom box, indicating a causal link.

Classification of types

```
graph TD; A[Classification of types] --> B[Single use]; A --> C["Multiple use quantitative Cartridge/ bench-top devices"]; B --> D["Qualitative cartridge/strip"]; B --> E["Quantitative cartridge/strip With reader device"];
```

Single use

Multiple use quantitative  
Cartridge/ bench-top devices

Qualitative  
cartridge/strip

Quantitative  
cartridge/strip  
With reader device

# Analytical Principle

- Reflectance
- Lateral flow or flow through immunoassays
- Electrochemistry
- Electrical impedance

# Reflectance

e.g.

Urine and blood  
dipsticks for glucose and  
various other analytes.



# Electrochemistry

**Strip device**

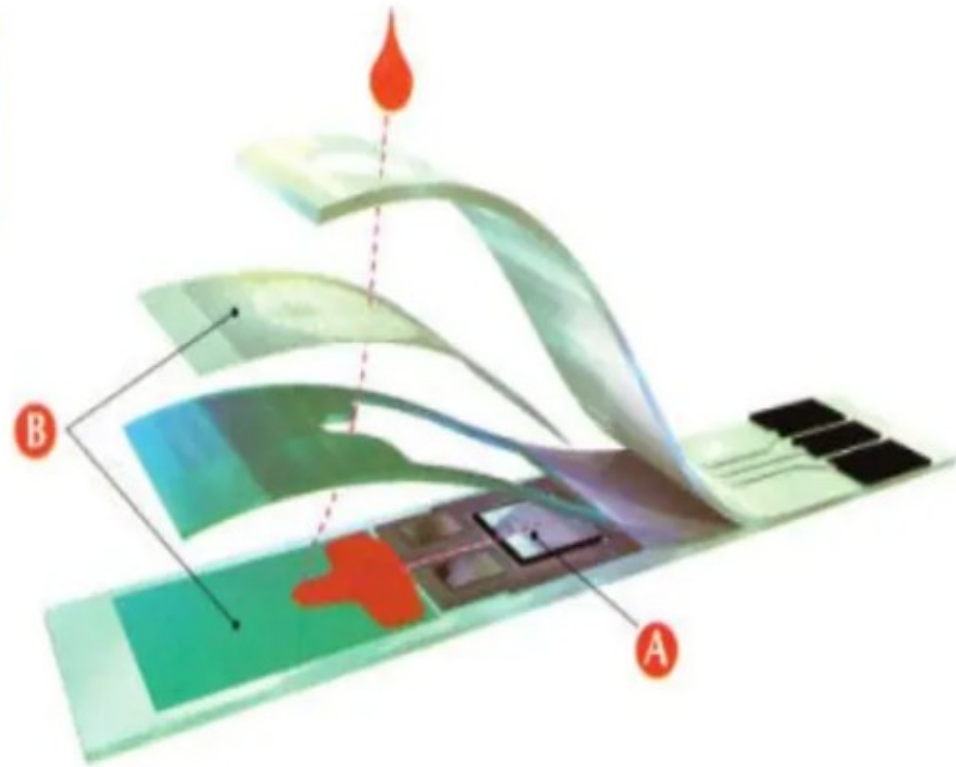


**Cassette and bench-top device**

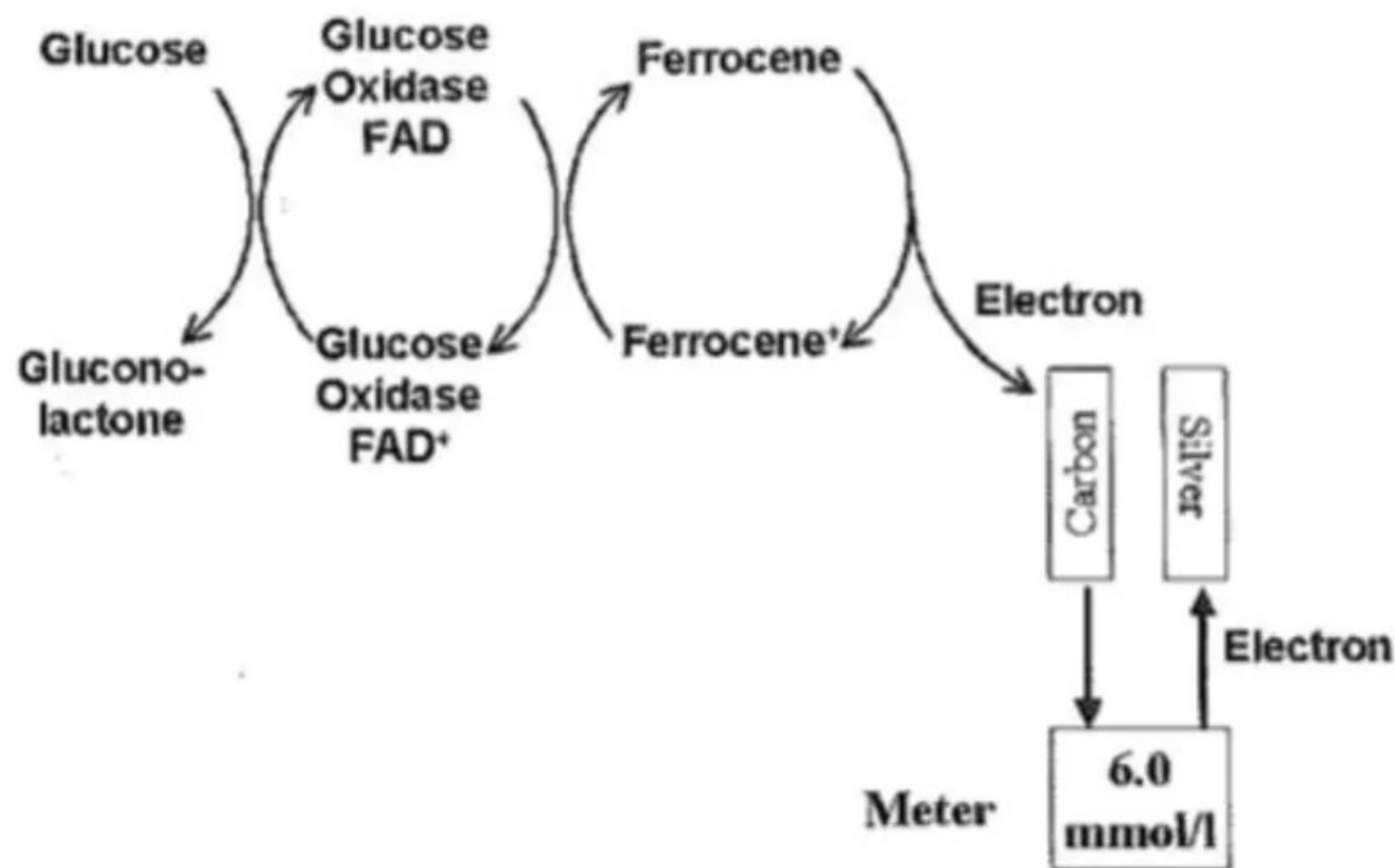


# Glucose strip

- A. Electrode sensors
- B. Separating layer

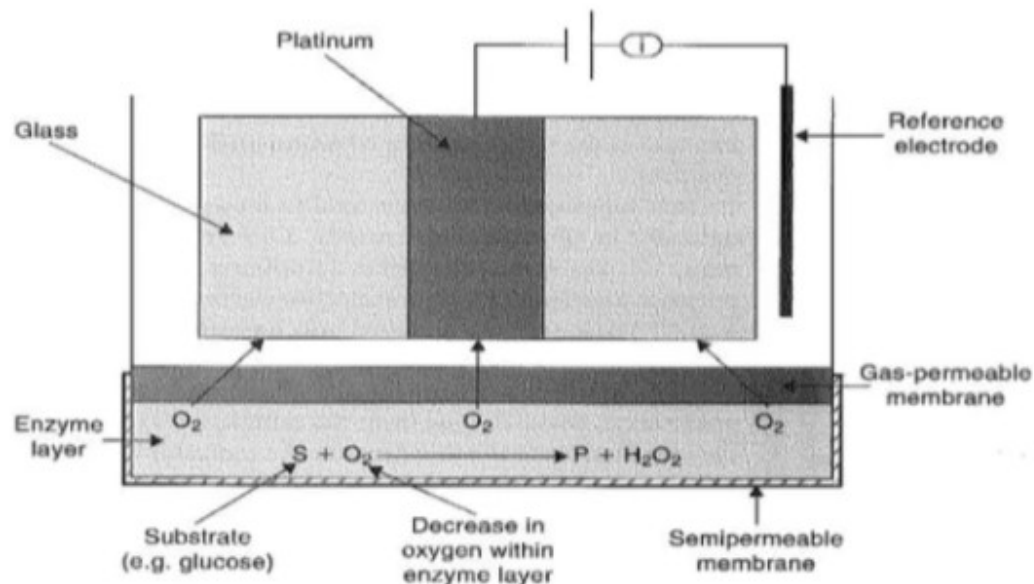


## Principle of Electrochemical Glucose strip



# Glucose meter

A negative voltage of  $-0.4\text{ V}$  is applied at the reference electrode. When blood or a glucose solution is placed in the strip, a chemical reaction occurs inside it, generating a small electrical current proportional to the glucose concentration



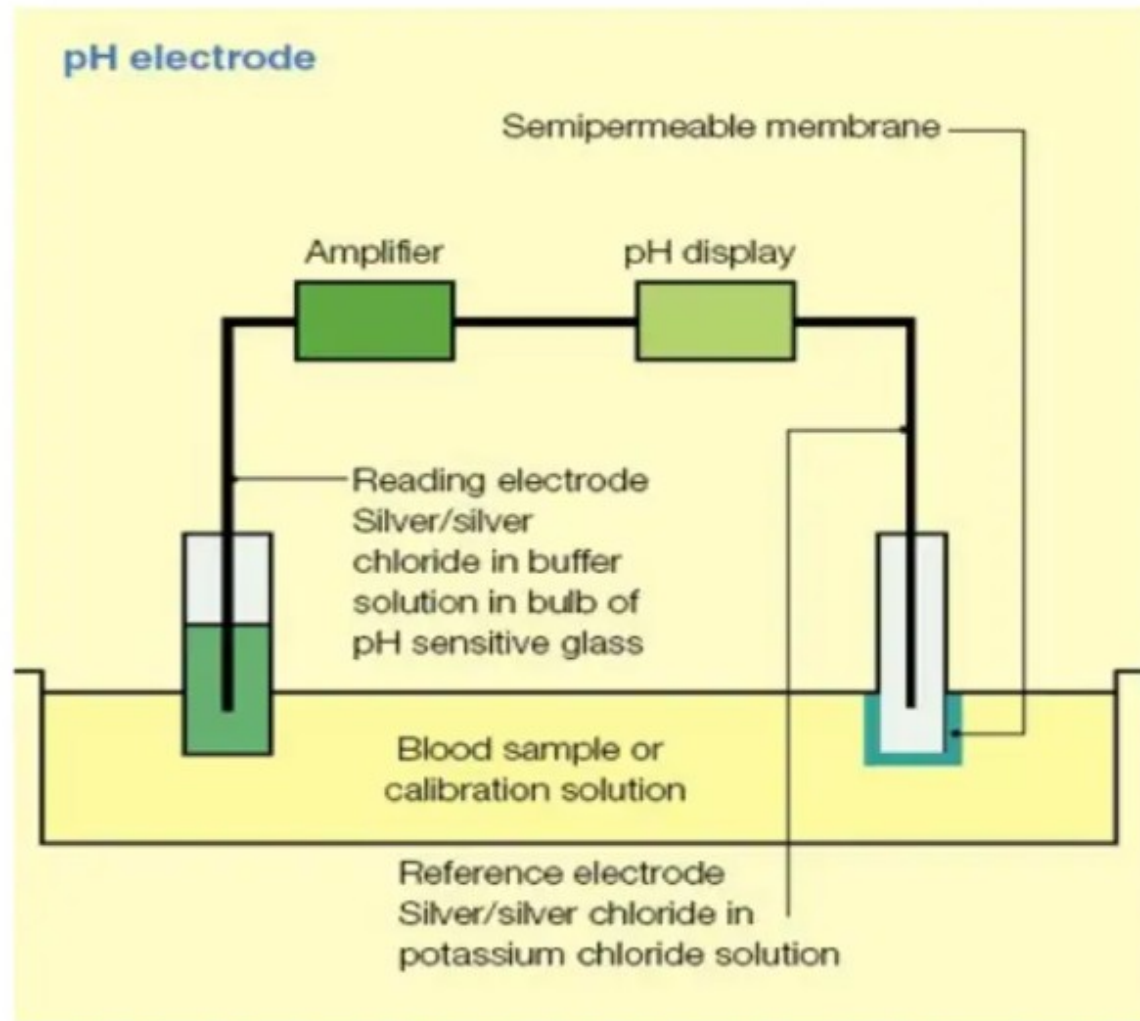
**Figure 4-11** Illustration of enzyme electrode prepared using oxidase enzyme immobilized at the surface of amperometric  $PO_2$  sensor. Increase in substrate concentration  $S$  reduces the amount of oxygen present at the surface of the sensor.

# Bench-top Devise



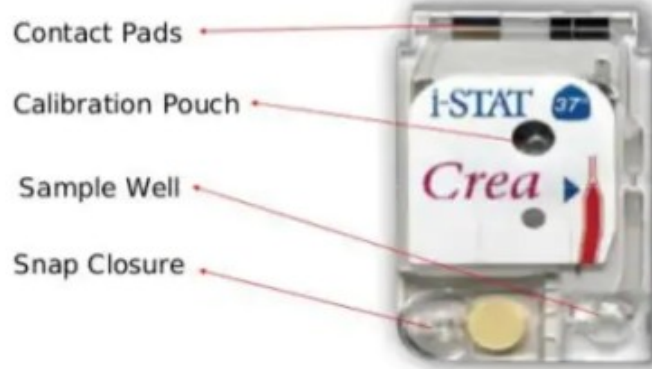
# Principle

Measurement of  
PH, PCO<sub>2</sub>, PO<sub>2</sub>,  
Electrolyte, Glucose,  
Lactate.



# Cassette device

i-STAT Cartridge



i-STAT Analyzer



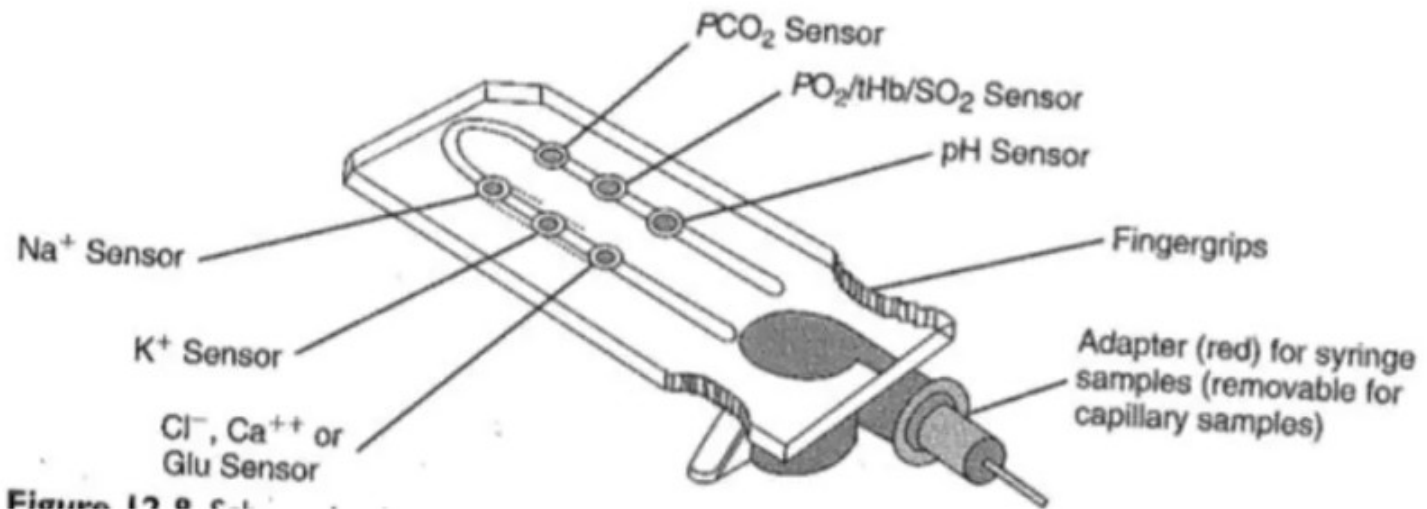
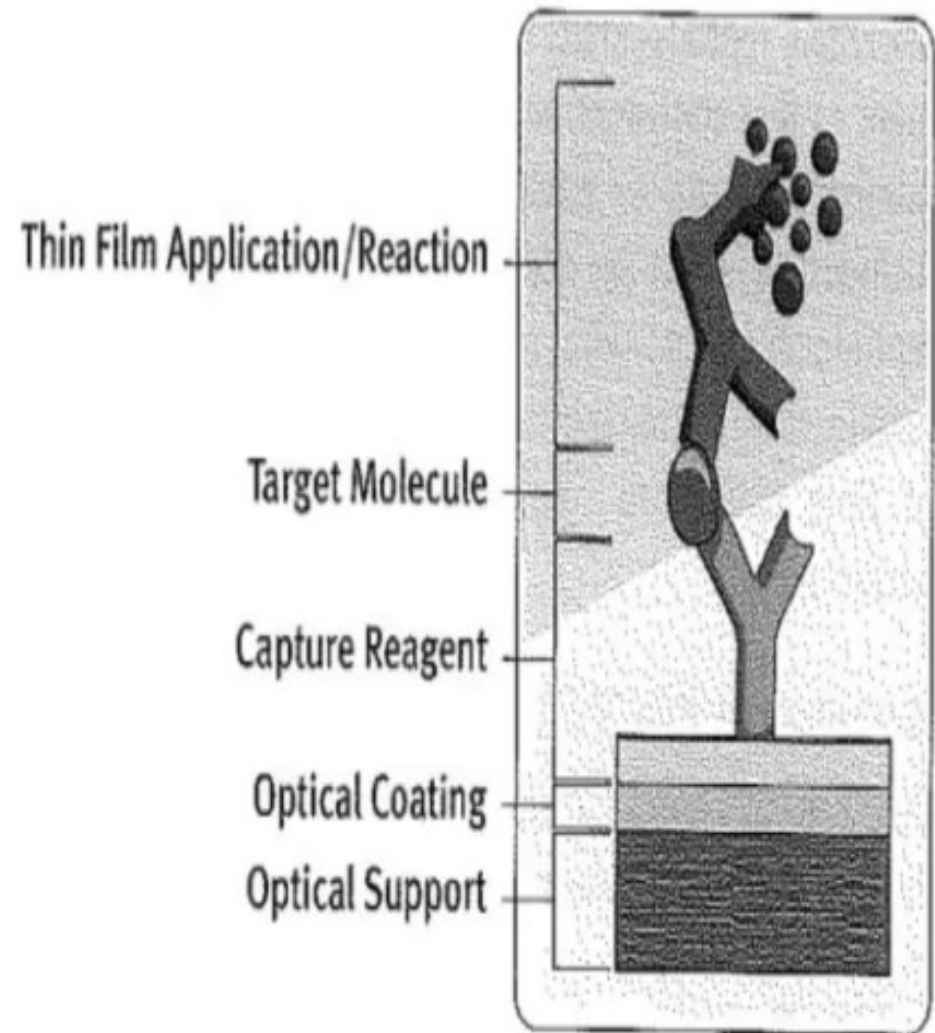


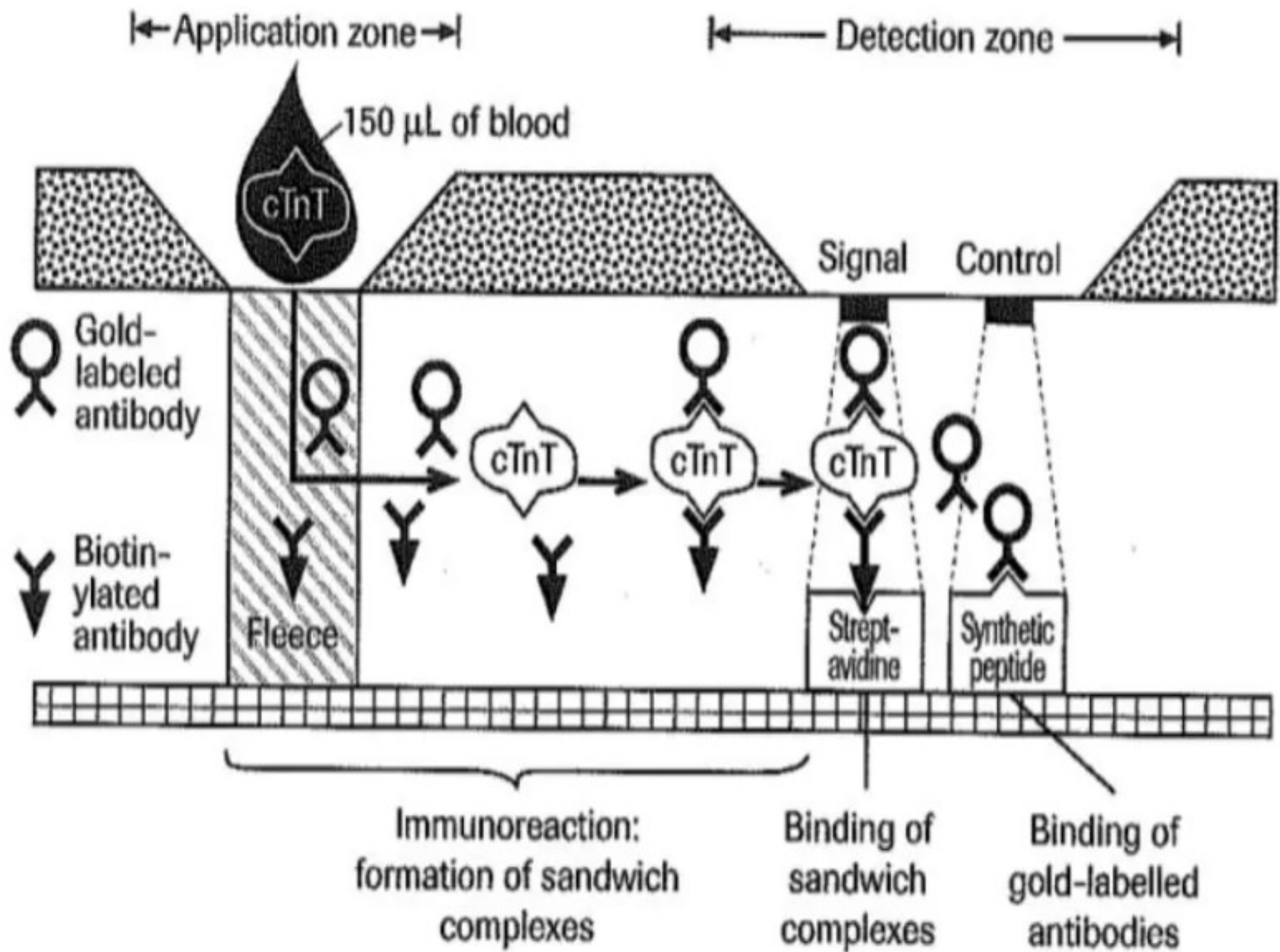
Figure 12.8.5.1

# Lateral flow immunoassays

It is a biological sensors in which the recognition agent is an antibody that binds to the analyte.

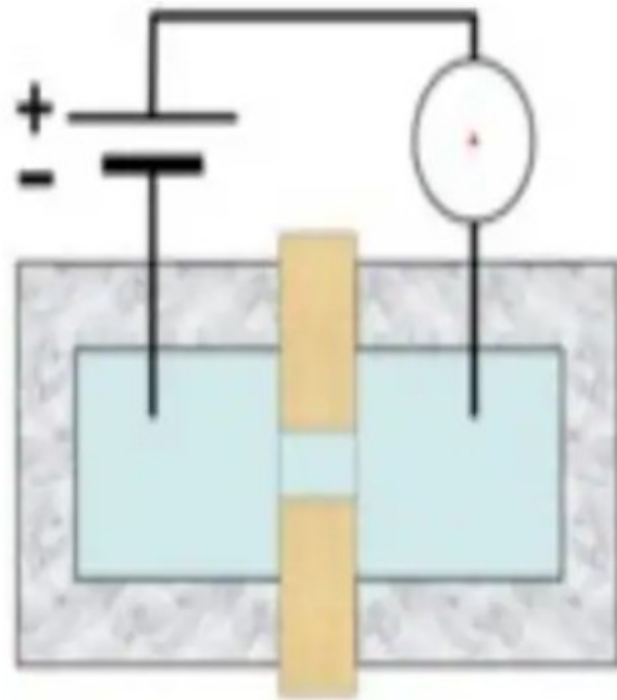
e.g. measurement of troponin T , myoglobin, and D-dimer.





# Electric Impedance

- Based on Coulter principle
- As fluid containing particles or cells is drawn through each micro-channel, each particle causes a brief change to the electrical resistance of the liquid.
- The counter detects these changes in electrical resistance.
- E.g. complete blood count





**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009

**Report on Certificate Course on Point of Care Testing (POCT)**

**Topic:** Point of care testing (POCT)


**Date:** 30.11.2017

**Venue:** Biochemistry Demonstration Room

**Target Audience:** Faculties and M.B.B.S., students

**Number of Participants:** 142

**Event Report:** The event started with the welcome address by Dr. P. K. Mohanty. Following that Dr. A. Hariharan started the lecture on POCT. He explained the basics concept in point of care testing and followed by this he showed a live demonstration of steps in estimation of capillary blood glucose, urine pregnancy testing and troponin I and its interpretation.

  
Prof. T. THIRUVAVUKKARASU, M.D.,D.A.,  
Dean  
Velammal Medical College Hospital  
and Research Institute  
"Velammal Village"  
Madurai-Tuticorin Ring Road  
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**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009





**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009



**Outcome:** Participants should be able to do the common point of care testing and should be able to interpretation the results.



**Velammal Medical College Hospital and Research Institute**

Ref. No: VMCHRI/BIOCHEM/CC-2

Date: 08.05.2018

**CIRCULAR**

To

All Doctors

**Certificate Course on Serum protein Electrophoresis**

Department of Biochemistry is organizing a certificate course on serum protein electrophoresis on 31.05.2018 (Thursday) between at 9.00 AM to 3.00 PM.

All Faculties are invited.

Copy submitted to:  
The Hon. Chairman

Copy to:  
The Dean  
Medical Superintendent  
Chief Administration Officer  
HOD, Biochemistry  
All Clinical and Non-Clinical HODs

**VICE PRINCIPAL**

**Dr. P.K. MOHANTY**

Vice Principal  
Velammal Medical College Hospital  
and Research Institute  
Madurai-625 009



**Velammal  
Medical College Hospital &  
Research Institute  
Anuppanadi, Madurai - 625009**

**Department of Biochemistry**

**Certificate course on**

**Serum protein  
electrophoresis**

**Venue: Biochemistry  
Demonstration  
Room**



**9.00 Am – 3.00 PM**

**For First year M.B.B.S., students**

**PATRON**

**Chairman:** Shri.M.V.Muthuramalingam

**Advisors:**

**Dean:** Dr.R.M. Raja Muthaiah

**MS:** Dr. Somasundaram

DR.P.K.Mohanty

**Course Coordinator**

**Vice Principle**

**Prof. HOD Biochemistry**

**Teaching Method:** Lecture  
or demonstration

**Objective:** To understand  
procedure and interpret the  
serum protein electrophoresis

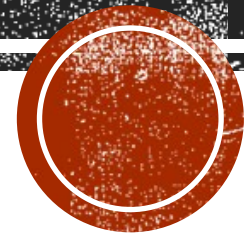
**31**

**MAY 2018**

# Agenda

<b>Time</b>	<b>Topic</b>	<b>Speaker</b>
9.00 – 9.15 AM	Welcome address	DR.K.Suganthy
9.15 – 9.45 AM	Pre test	DR.P.K.Mohanty
9.45 – 11.00 AM	Electrophoresis – Basics	DR.P.K.Mohanty
11.00 AM – 1.00 PM	Demonstration: Serum protein electrophoresis	DR.P.K.Mohanty & Resource persons
1.00-2.00 PM	LUNCH	
2.00 – 2.30 PM	Post test	DR.P.K.Mohanty
2.30 – 3.00 PM	Feed back and Valediction	DR.K.Suganthy

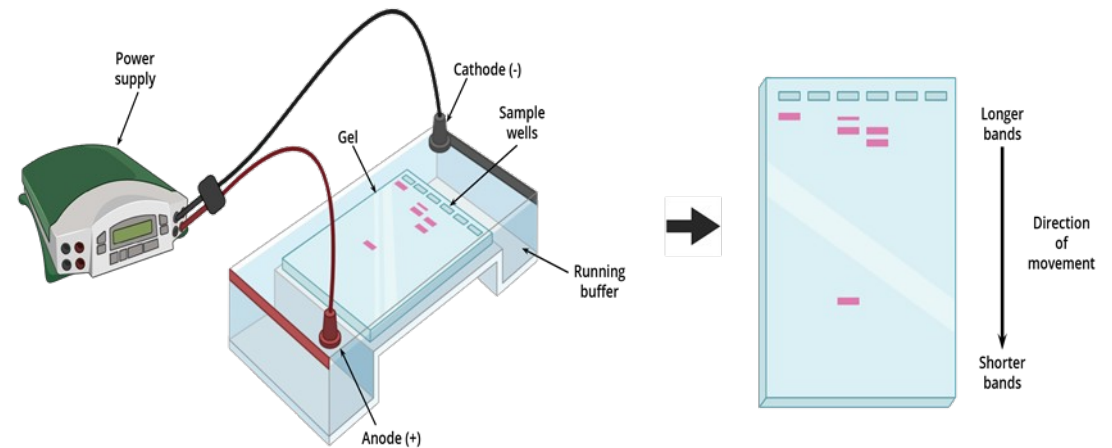
# Serum protein electrophoresis



Dr.P.K.Mohanty M.D., (Biochemistry)

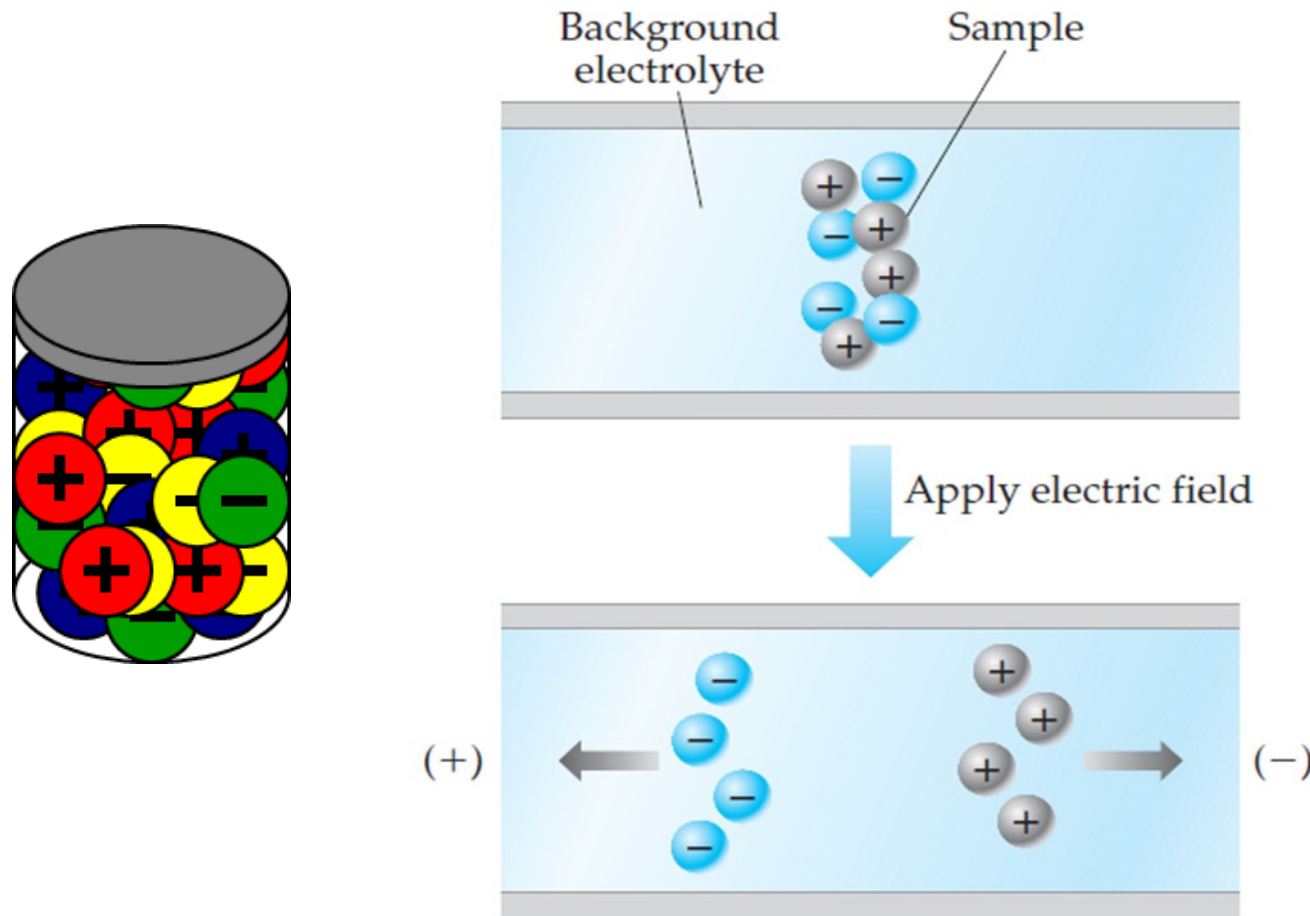
# Contents

- Definition
- Principle of electrophoresis
- Requirements of agarose gel electrophoresis
- Procedure
- Electrophoretic pattern in various diseases



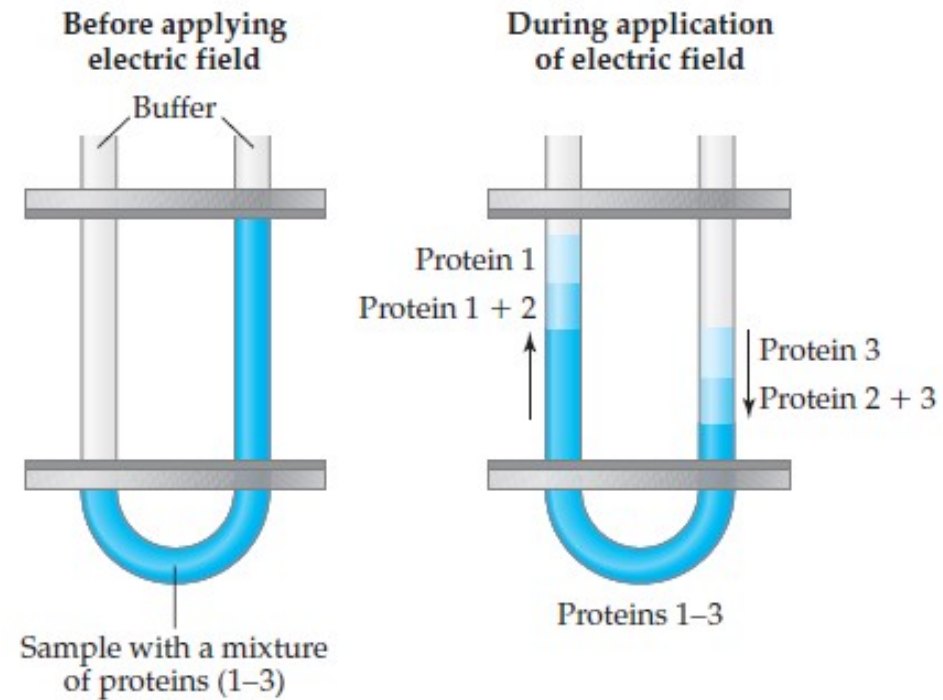
# What is electrophoresis?

- Movement of charged particles through an electrolyte when subjected to an electrical field.



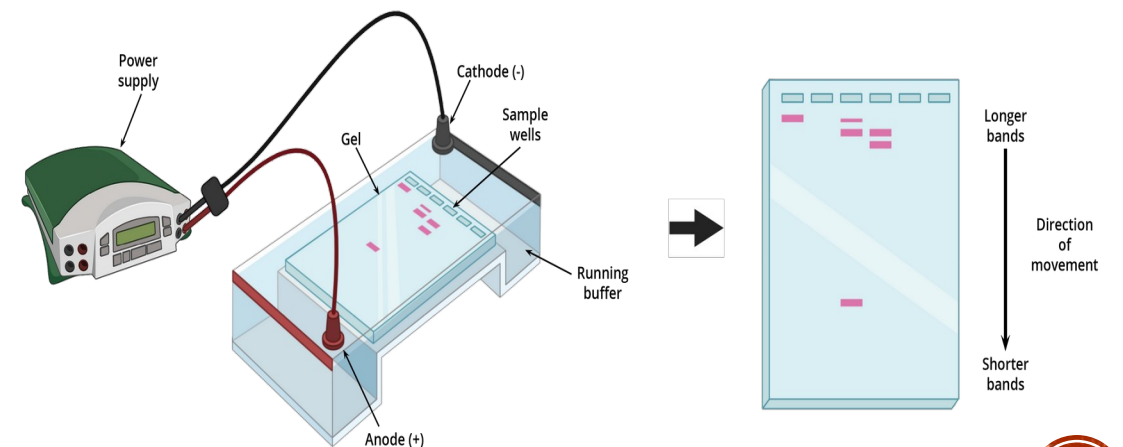
# History

- Invented by Tiselius



# Clinical applications of electrophoresis

- a separation technique
- Simple, rapid and highly sensitive
- used in clinical laboratories to separate charged molecules from each other in presence of electric field
  - Proteins in body fluids: serum, urine, CSF
  - Proteins in erythrocytes: haemoglobin
  - Nucleic acids: DNA, RNA
  - Lipoprotein analysis
  - Isoenzyme separation



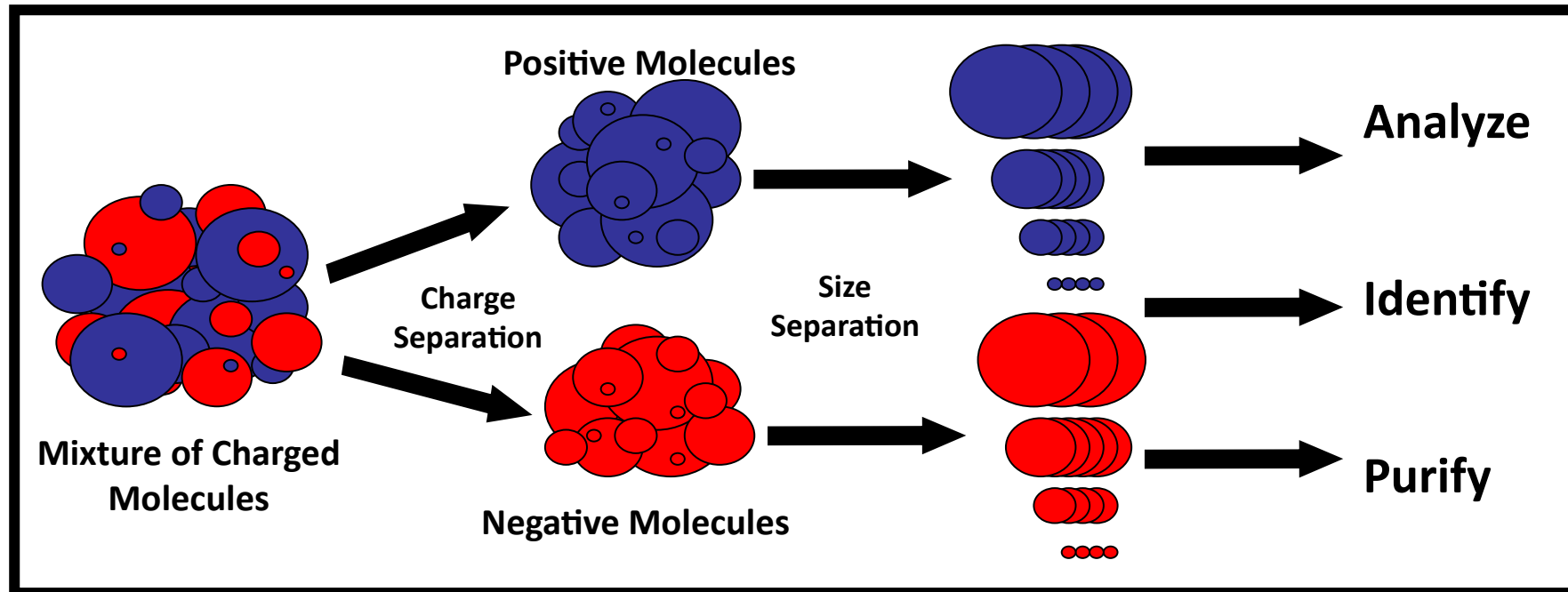
# Principle

- Comprehensive term that refers to the migration of charged particle of any size in liquid medium under the influence of an electric field.
- Depending on kind of charge the molecule carry, they move towards either
  - To cathode
  - To Anode
- An ampholyte become positively charged in acidic condition and migrate to cathode, in alkaline condition they become negatively charge and migrate to anode.



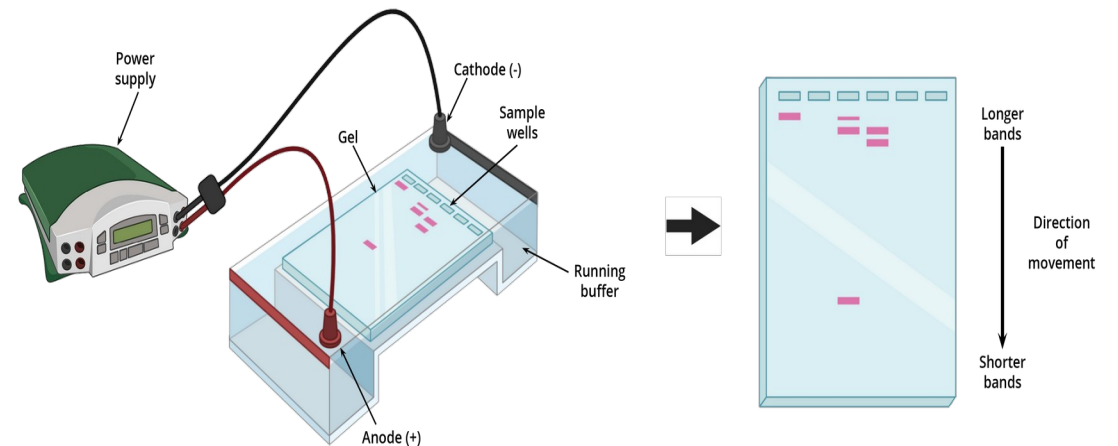
# Types of separation

- Native : Charge/ Mass
- Denaturing : Mass



# Factors affecting Electrophoresis

- Net charge
- Mass
- Shape
- pH of medium
- Strength of electrical field
- Viscosity of medium
- Temperature



# *Factors affecting Electrophoresis*

- The Electrophoretic mobility is directly proportional to net charge and inversely proportional to molecular size/mass and viscosity of the electrophoresis medium
- The pH of solution affects the mobility of the ion by determining the amount and nature of charge



# *Electrophoresis of Biomolecules*

- Proteins, nucleic acids, nucleotides and amino acids bear charged polar groups making them suitable groups for electrophoresis
- Carbohydrates carrying no charged groups are first bound to charged groups like Borate or Sulfite ions and then electrophoresis is carried out
- Lipids are not electrophoresed because electrophoretic current requires polar solvents in which most lipids are insoluble

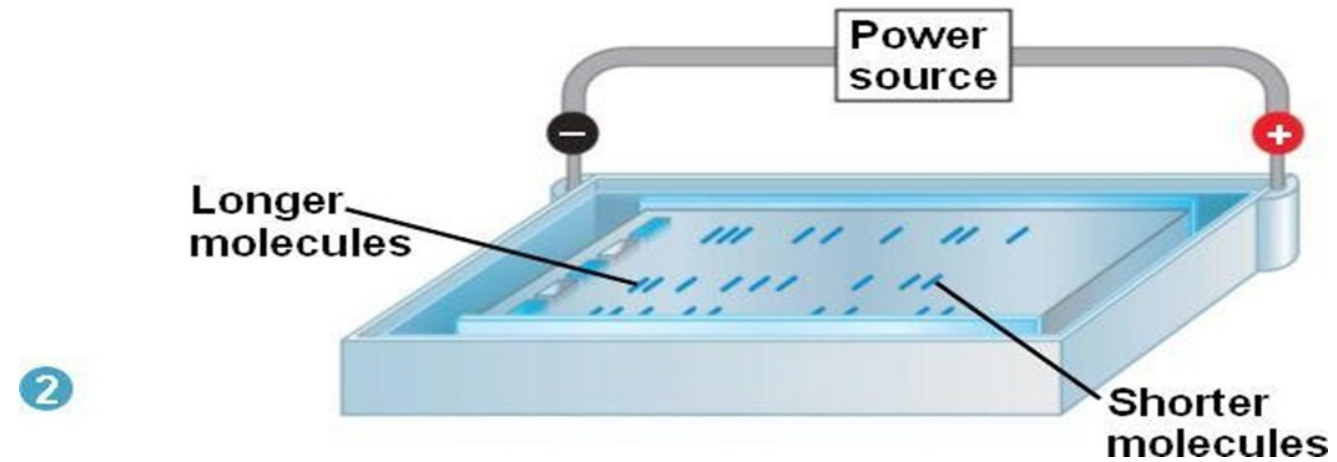
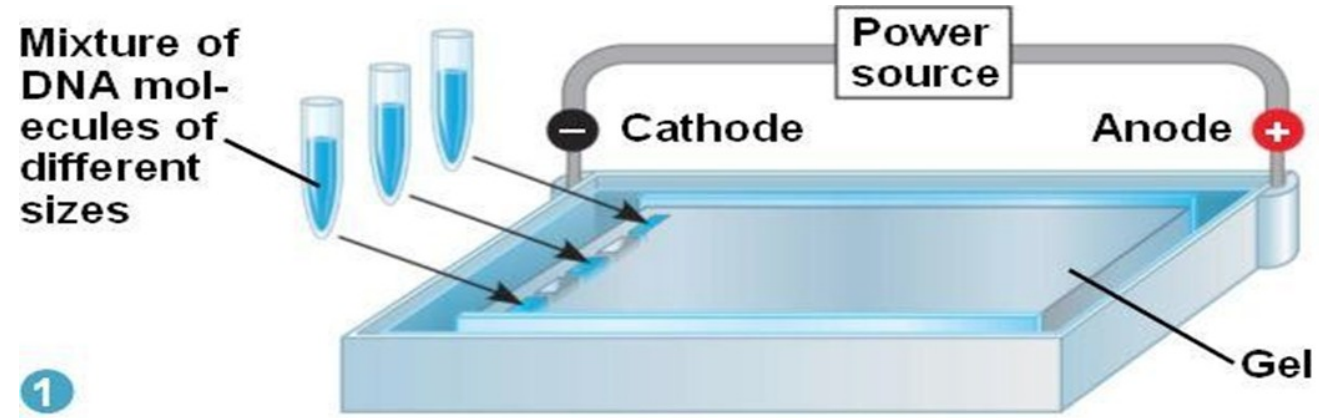
# *Factors affecting Electrophoresis*

- The rate of migration depends on size, shape, net charge & the applied current

$$v = EqF$$

- $v$  = velocity
  - $E$  = electric field (v/cm)
  - $q$  = net charge
  - $F$  = Frictional coefficient
- Charge – Higher the charge, greater the mobility
  - Shape – rounded contour elicit less frictional and electrostatic retardation compared to sharp contour. Therefore globular proteins moves faster than fibrous proteins.



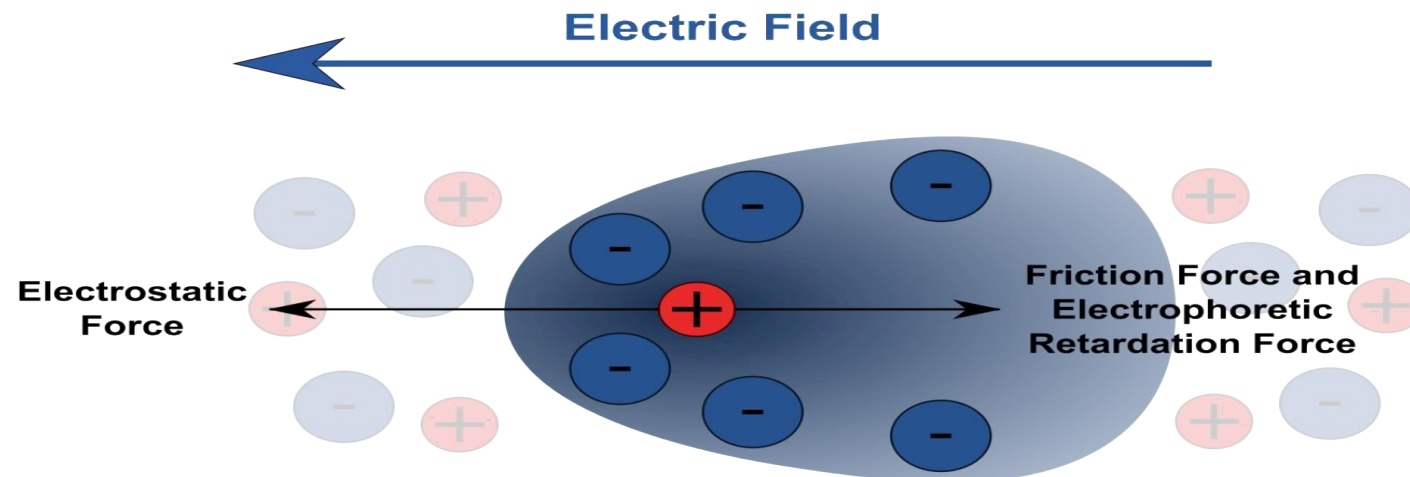


# *Strength of electrical field*

- It determined by the force exerted on the particle, and the charge the particle carrying.

$$F=QV$$

when force is exerted on the particle it start moving, however the moment is restricted by the experience of the frictional force because of the viscosity.

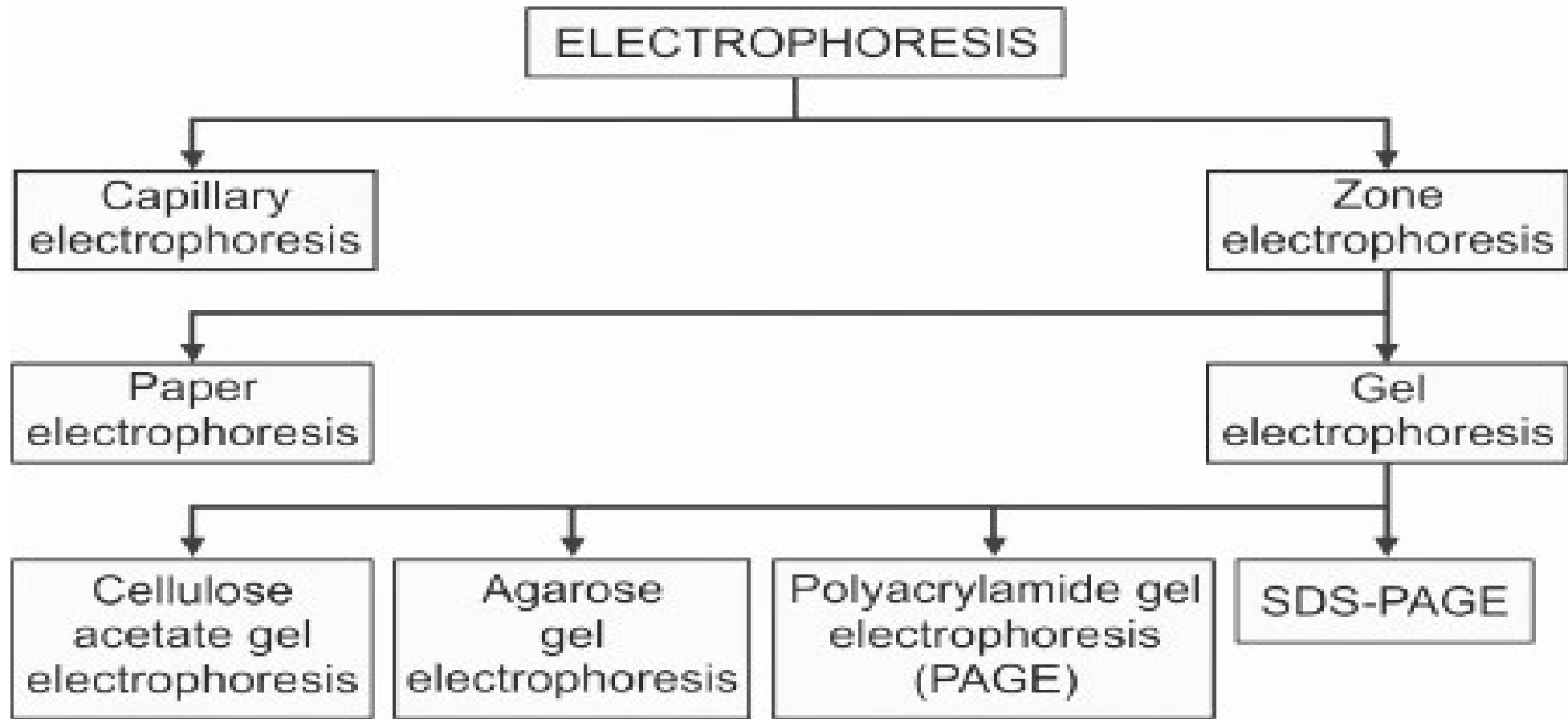


## *Effect of pH on Mobility*

- As the molecule exist as amphoteric , they will carry the charges based on the solvent pH.
- Mobility is directly proportional to the magnitude of the charge, which is functional of the pH of solvent.
- The pH is maintained by the use of buffers of different pH.



# *Types of electrophoresis*

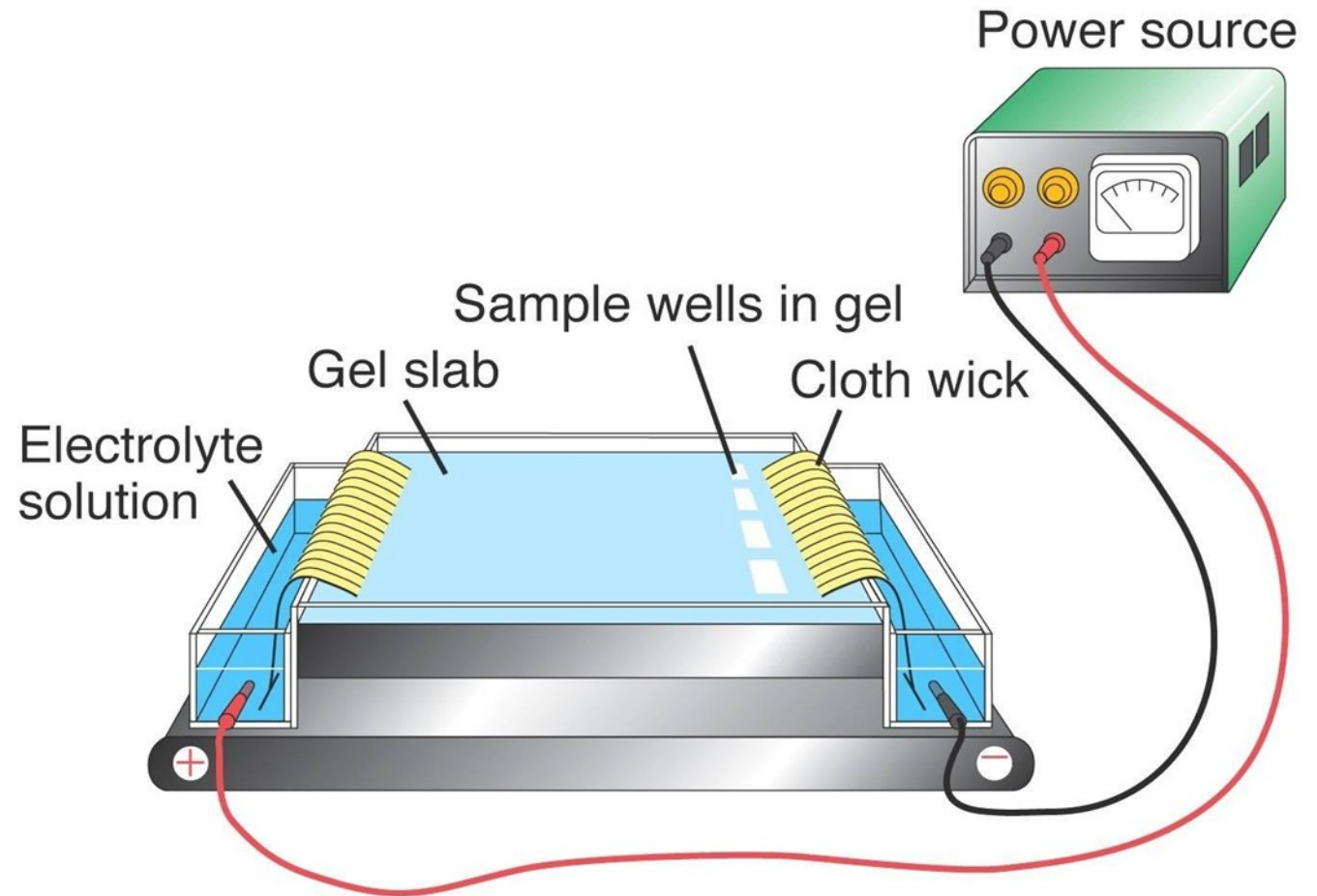


# *Gel Electrophoresis*

- “Gel” is the matrix used to contain, and then separate the target molecules
- The gel is a cross linked polymer whose composition and porosity is chosen based on the specific weight and composition of material to be analyzed
- A gel block made of Polyacrylamide, Agarose or substituted starch gel is used in this method as the solid support
- *Agarose gel - separation of different types of protein mixtures*
- *Polyacrylamide is best suitable for separation of nucleic acids.*

# Instrumentation

- Two reservoir for the buffer
- Power supply and Electrodes
- Supporting medium

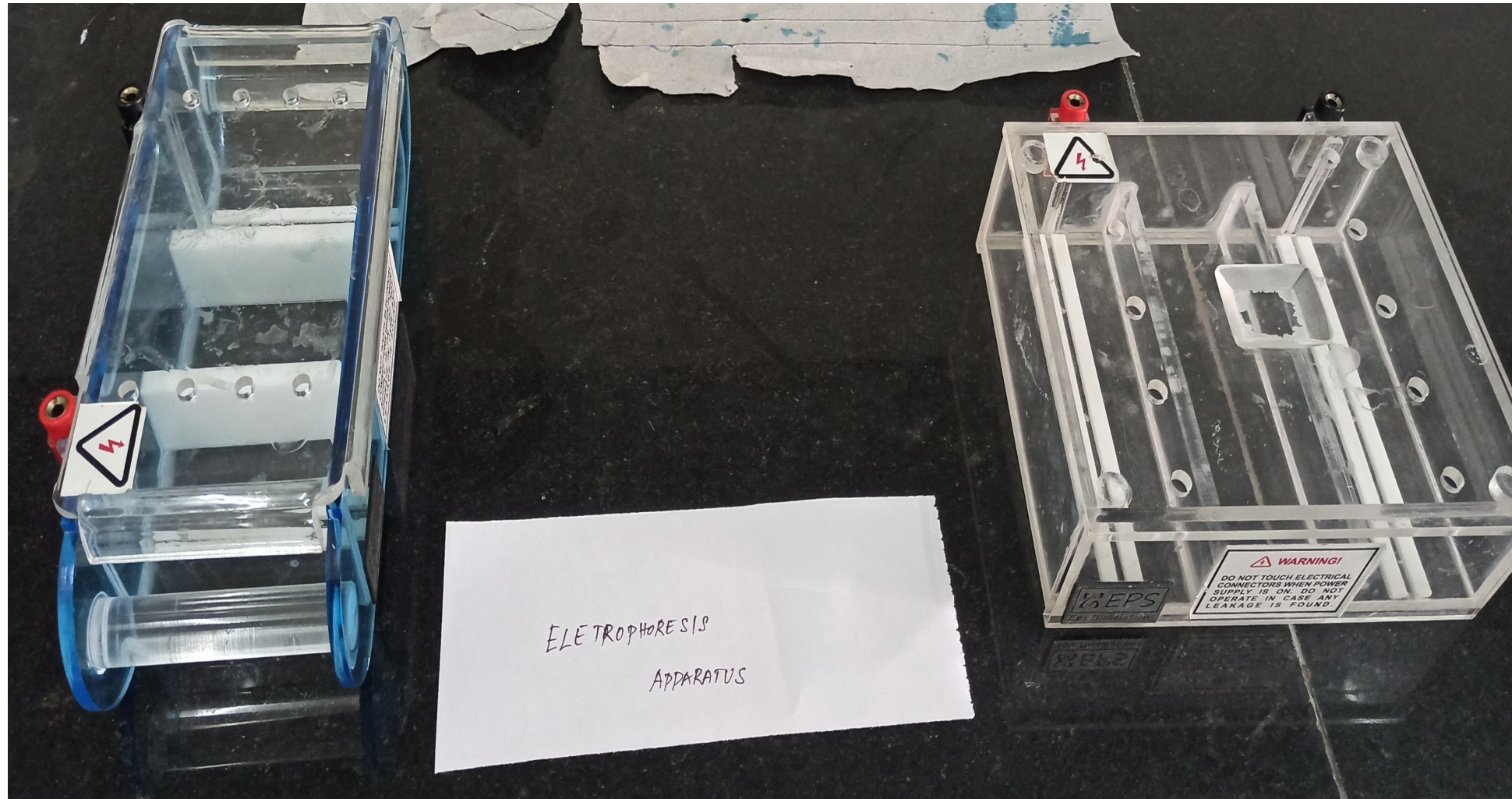


# **Requisites for Agarose gel Electrophoresis**

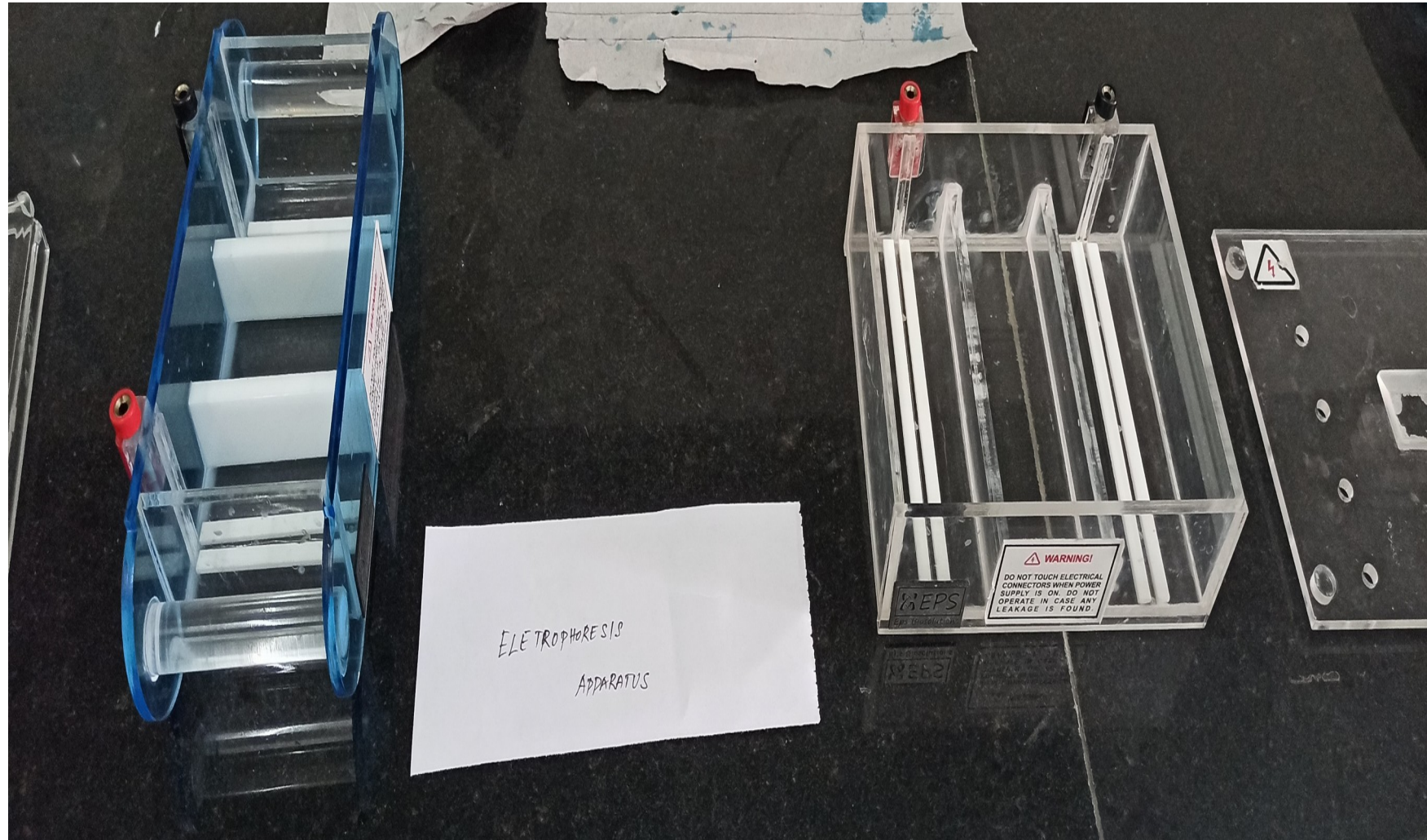
- Electrophoresis apparatus
- Supporting medium - Agarose
- Buffer
- Power pack to supply constant current and voltage
- Tracking dye
- Fixing solution
- Staining solution
- Destaining solution



# Electrophoresis apparatus



# *Electrophoresis apparatus*



# *Supporting medium*

- Supporting medium is an matrix in which the protein separation takes place.
- Separation is based on to the charge to mass ratio of protein depending on the pore size of the medium, possibly the molecular size.
- Starch gel
- Cellulose acetate
- Agarose
- Polyacrylamide gel

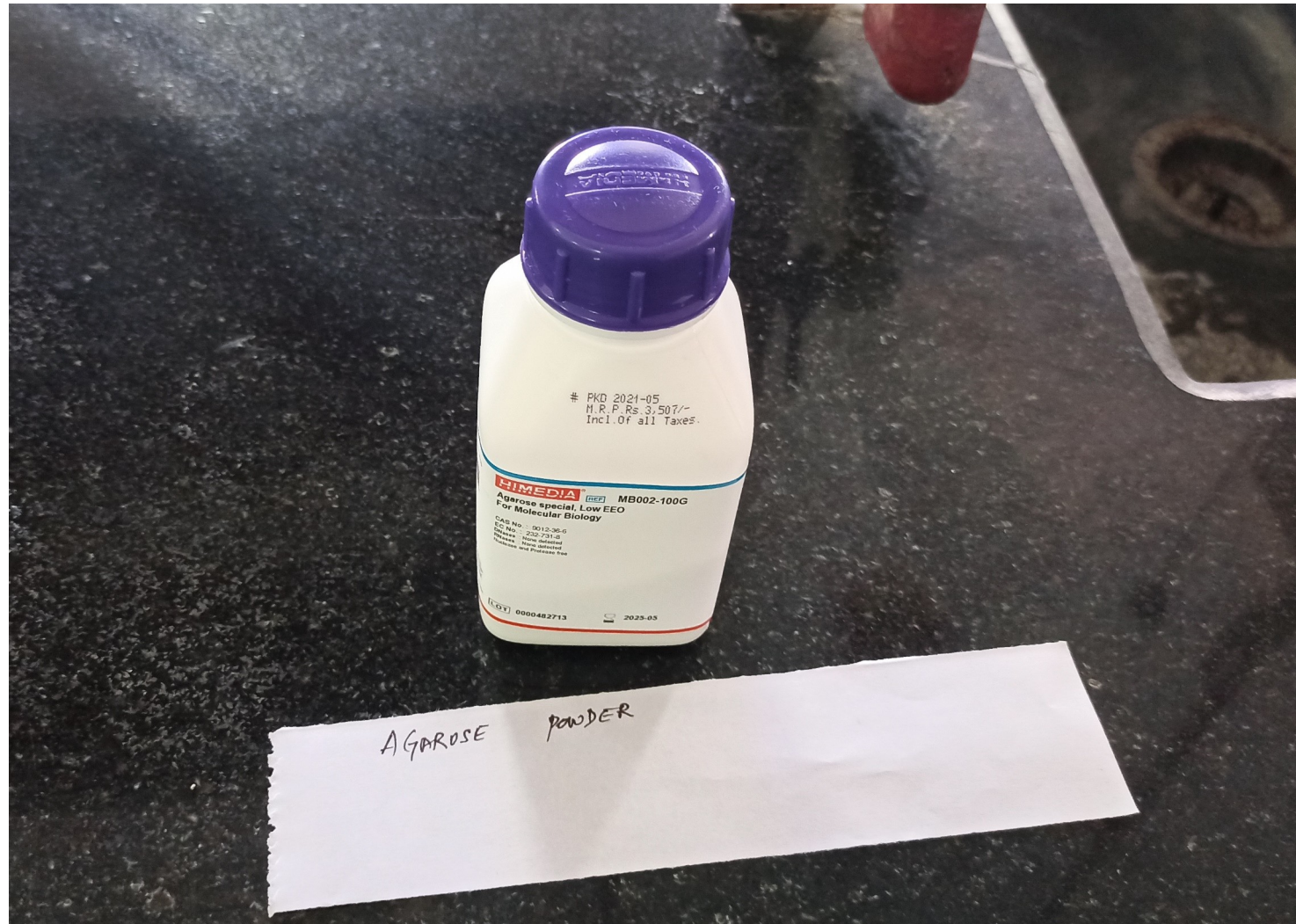


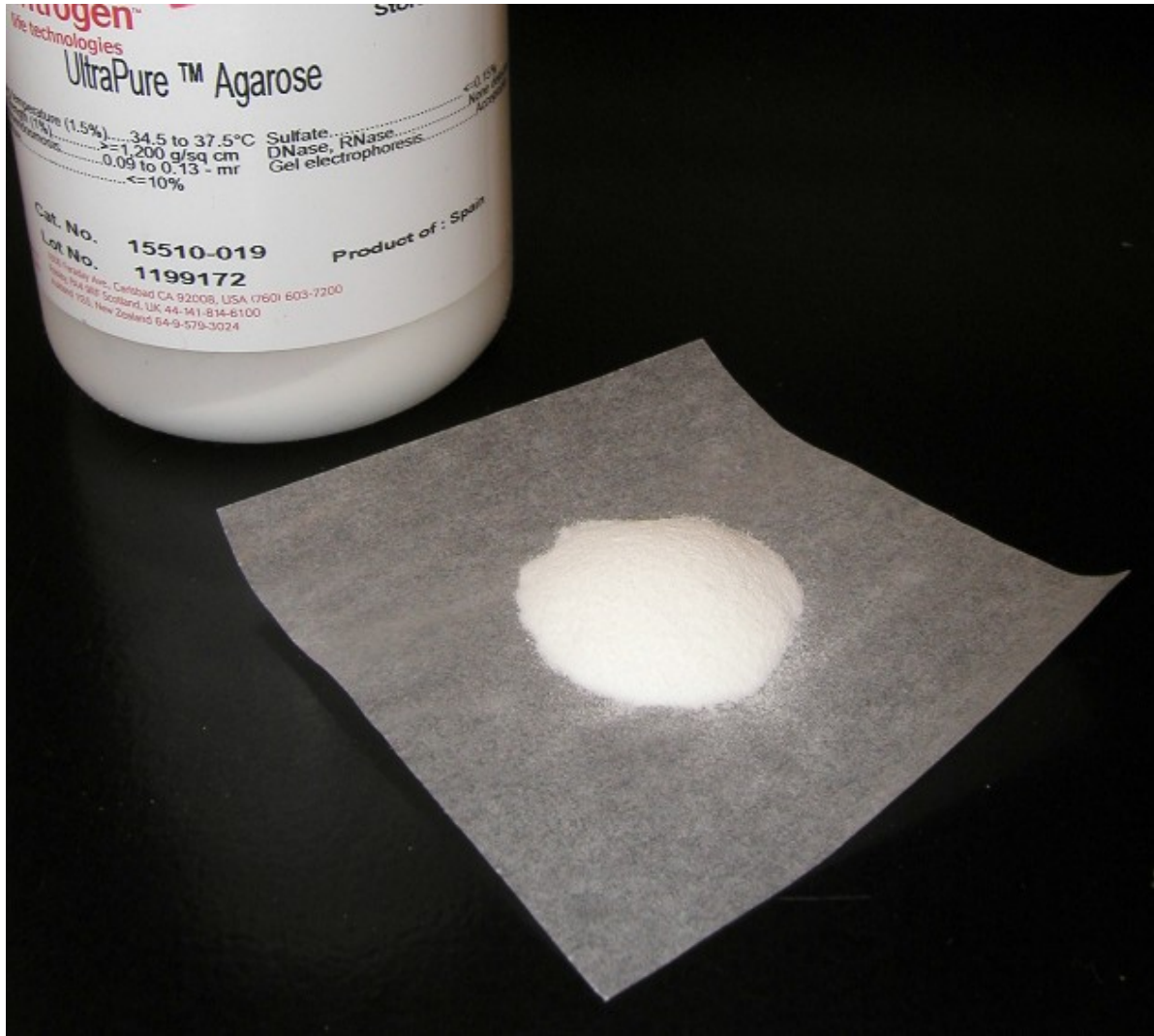
# Properties of supporting medium

Chemical nature	inert
Availability	easy
Electrical conductivity	high
Adsorptivity	low
Sieving effect	controlled
Porosity	high
Electro-endosmosis (EEO)	low
Preservation	feasible
Toxicity	low
Preparation	easy

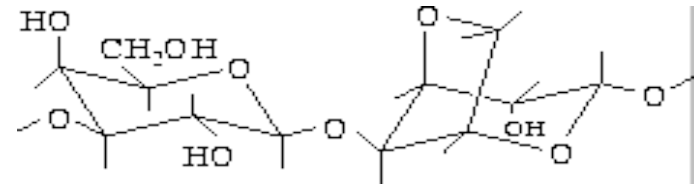


# Agarose





## Agarose



D-galactose    3,6-anhydro L-galactose

Agarose was first used in biology when Robert Koch\* used it as a culture medium for Tuberculosis bacteria in 1882

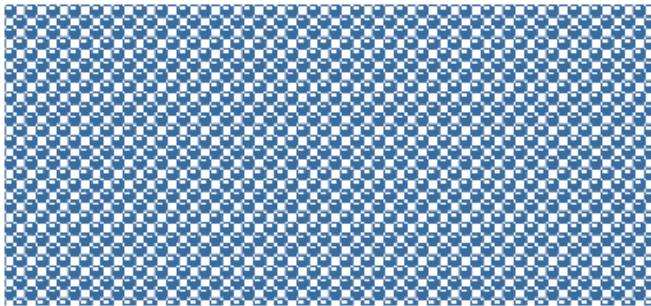
\***Lina Hesse**, technician and illustrator for a colleague of Koch was the first to suggest agar for use in culturing bacteria

Agarose is a linear polysaccharide extracted from seaweed.

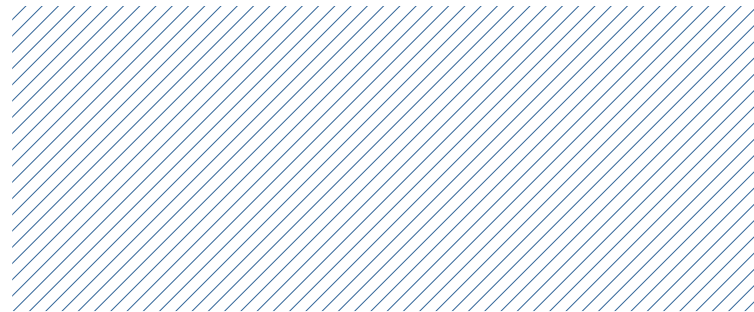


# Agarose

- Used in conc as 1% and 3%.
- The gelling property are attributed to both inter-and intramolecular hydrogen bonding
- Pore size is controlled by the % of agarose used.
- Large pore size are formed with lower conc and vice versa.



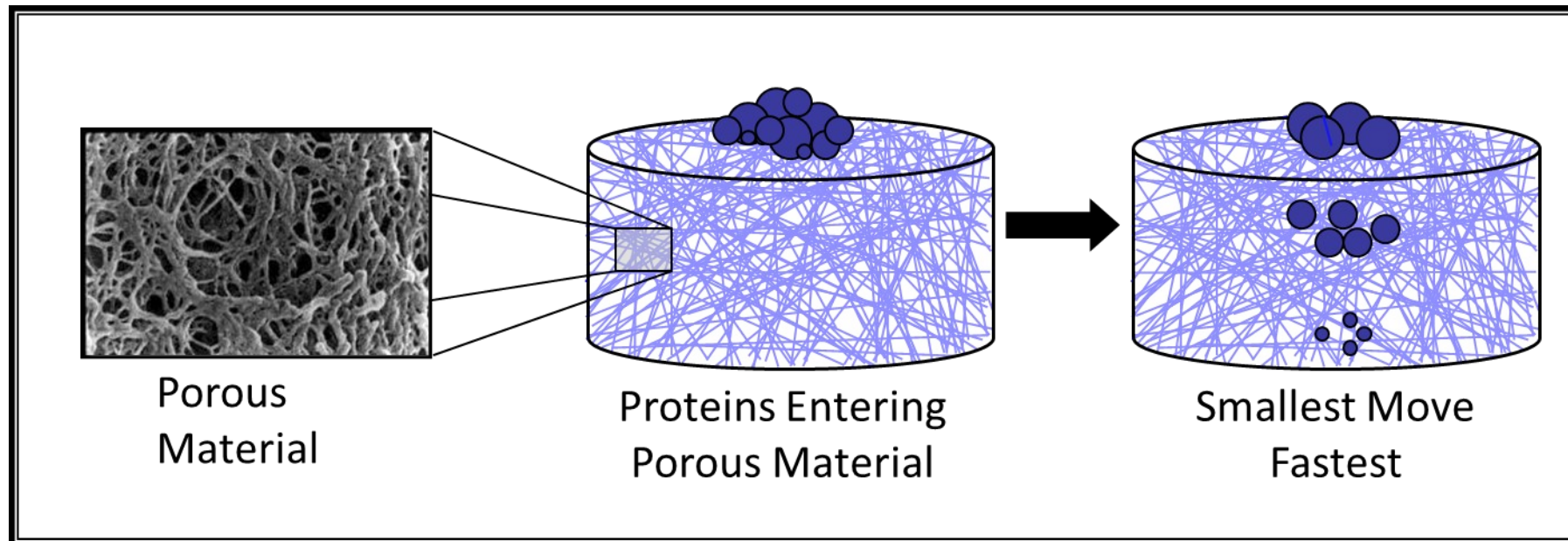
1% agarose



2% agarose



# Acts as molecular sieve

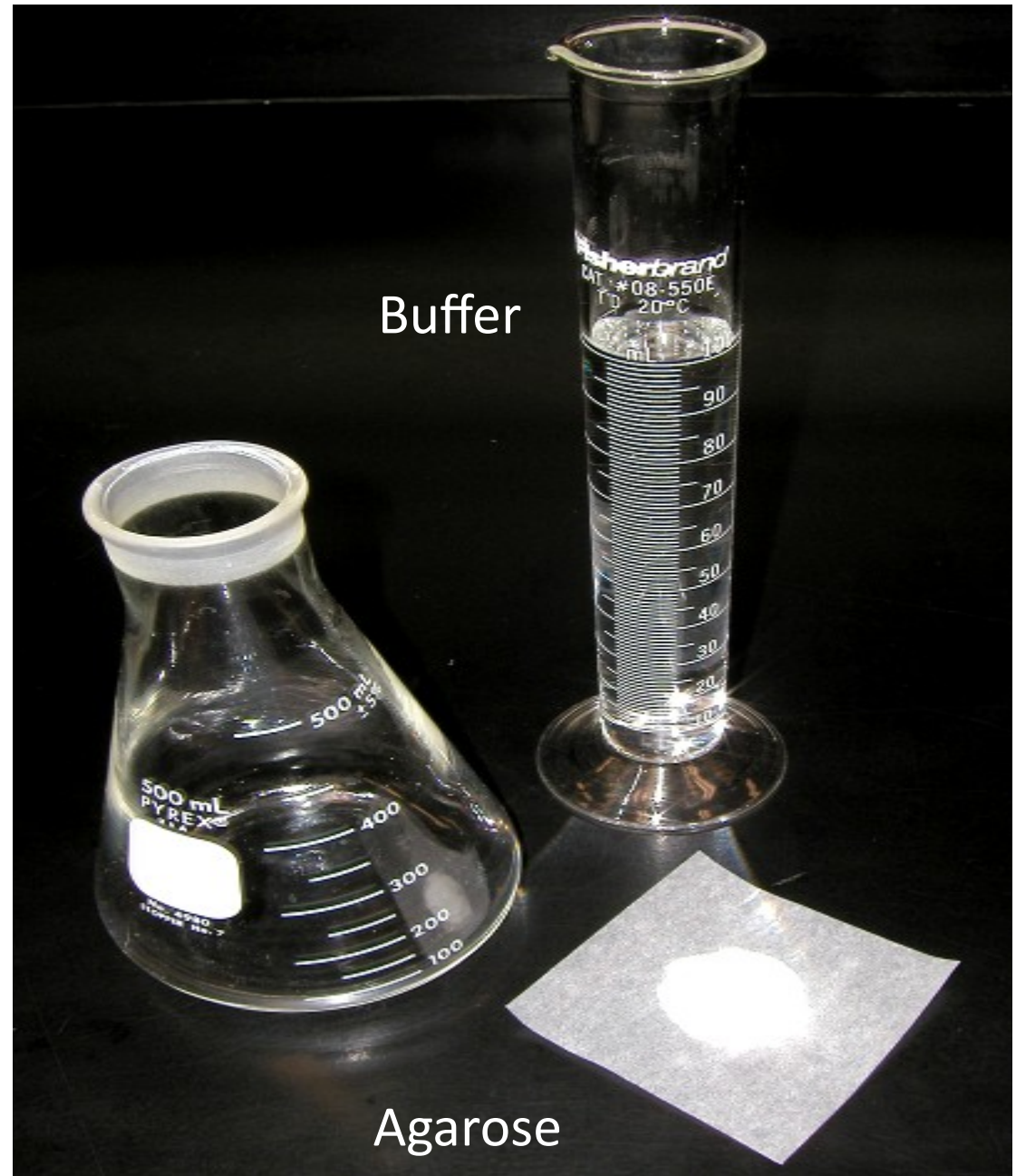


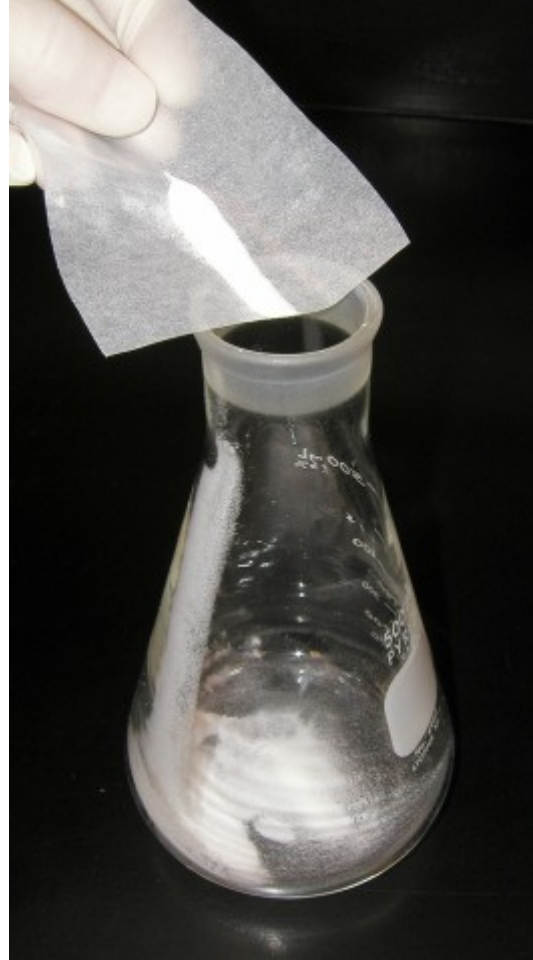
An agarose gel is prepared by combining agarose powder and a buffer solution.

Flask for boiling

Buffer

Agarose





Agarose

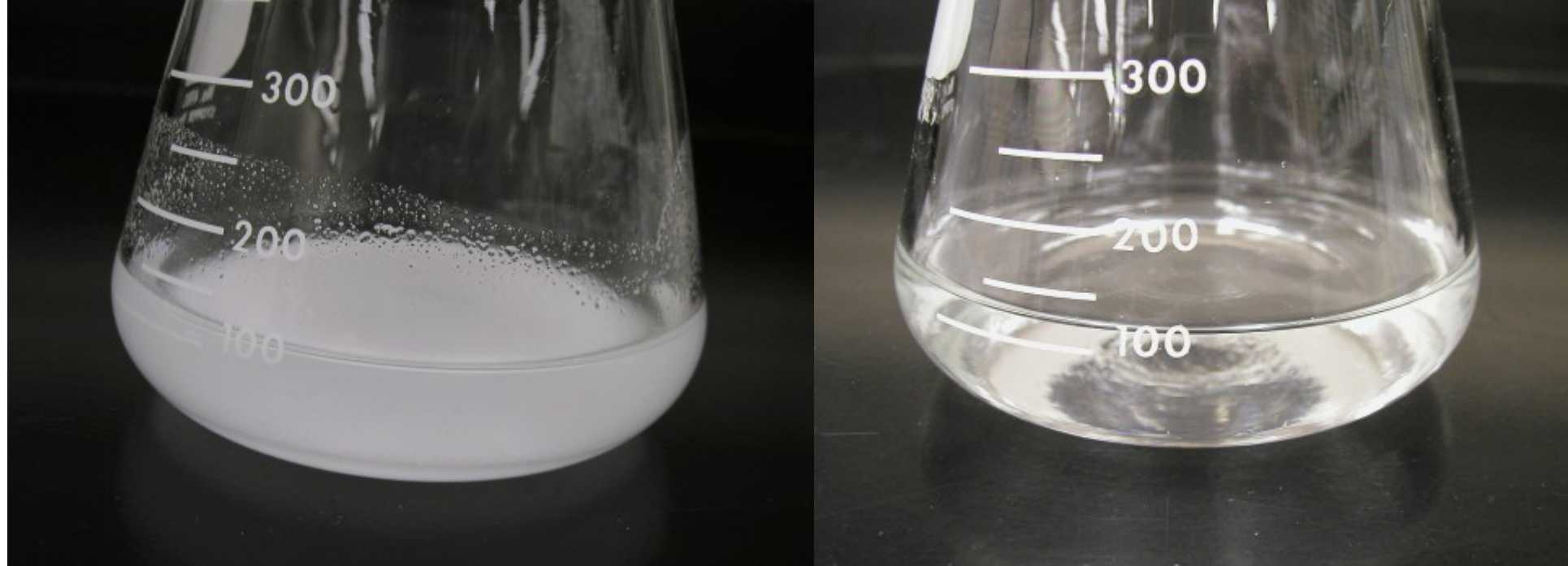


Buffer Solution

Combine the agarose powder and buffer solution. Use a flask that is several times larger than the volume of buffer.



## Melting the Agarose



Agarose is insoluble at room temperature (left).  
The agarose solution is boiled until clear (right).

Gently swirl the solution periodically when heating to allow all the grains of agarose to dissolve.  
\*\*\*Be careful when boiling - the agarose solution may become superheated and may boil violently if it has been heated too long in a microwave oven.



# Buffer

The buffer in electrophoresis has two purpose:

- Carry applied electrical current
- They set the pH as which electrophoresis is carried out.

Thus they determine;

- Type of charge on solute.
- Extent of ionization of solute
- Electrode towards which the solute will migrate.

The buffer ionic strength will determine the thickness of the ionic cloud.



# Commonly used buffers

Commonly used buffers are,

- Phosphate buffer
- Tris-Borate-EDTA buffer (TBE)
- Tris-Acetate EDTA buffer (TAE)
- Tris -EDTA buffer (TE)
- Lithium Borate - buffer (LB)
- Tris -Citrate-EDTA buffer (TCE)

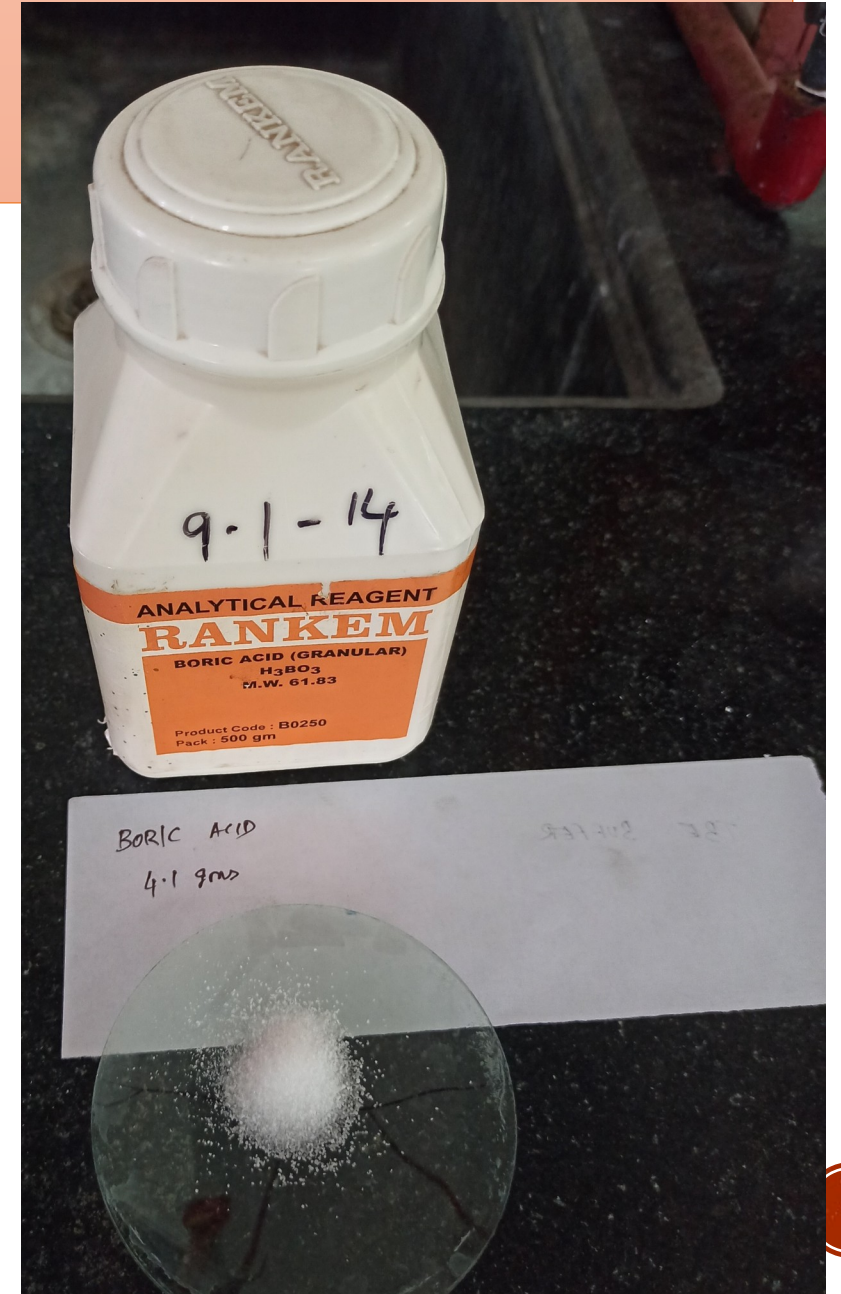
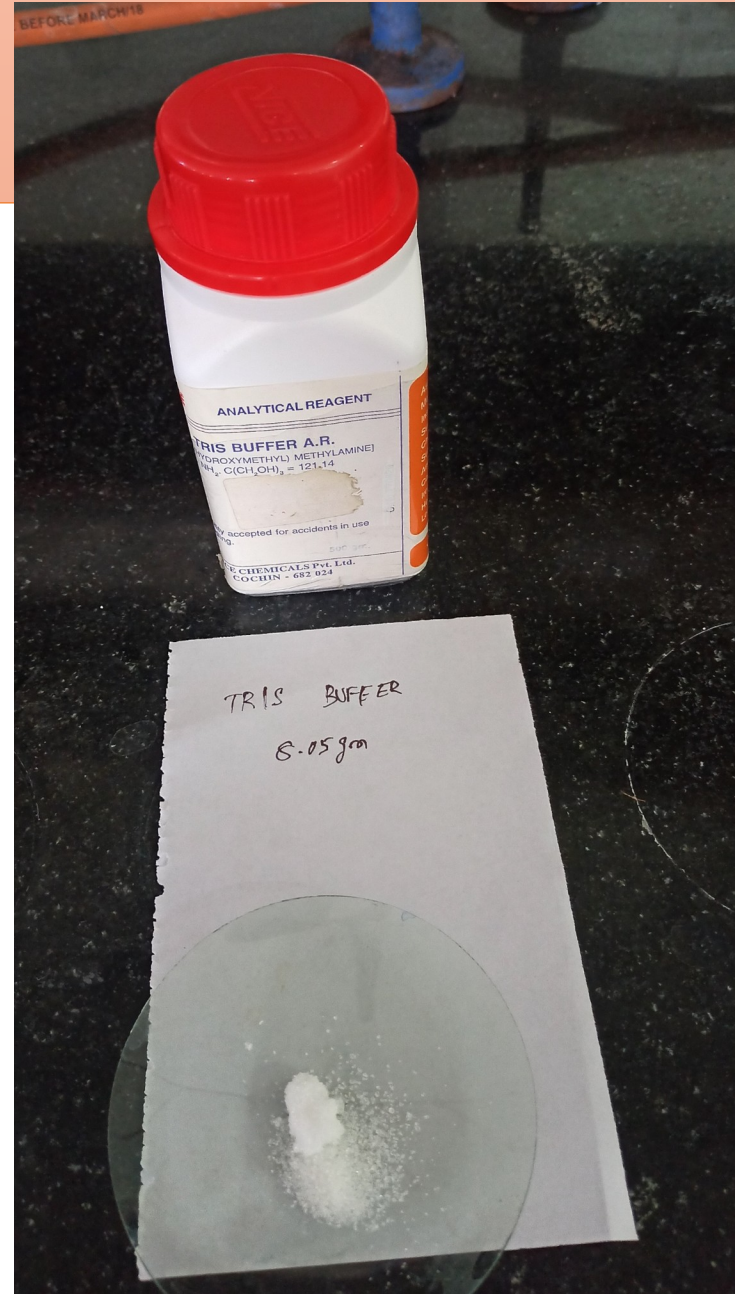
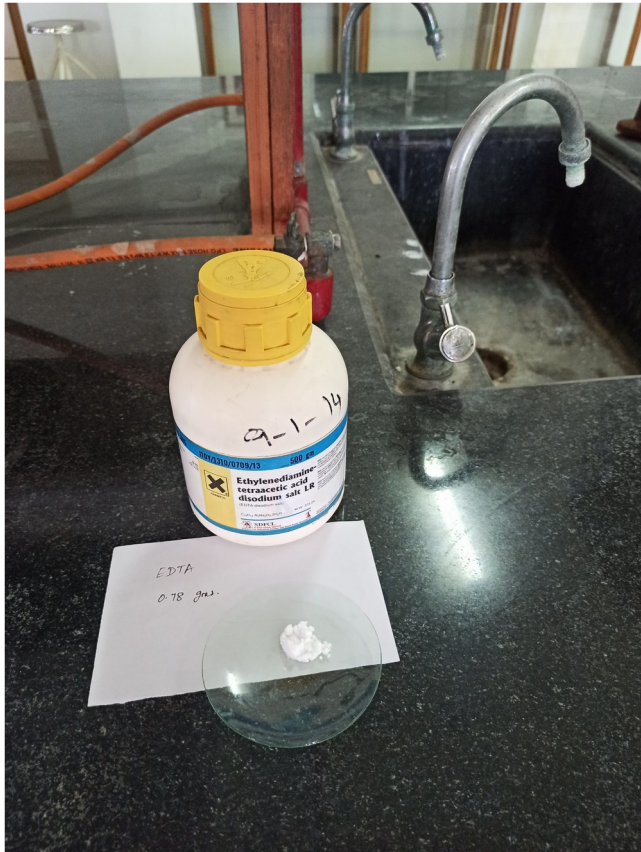


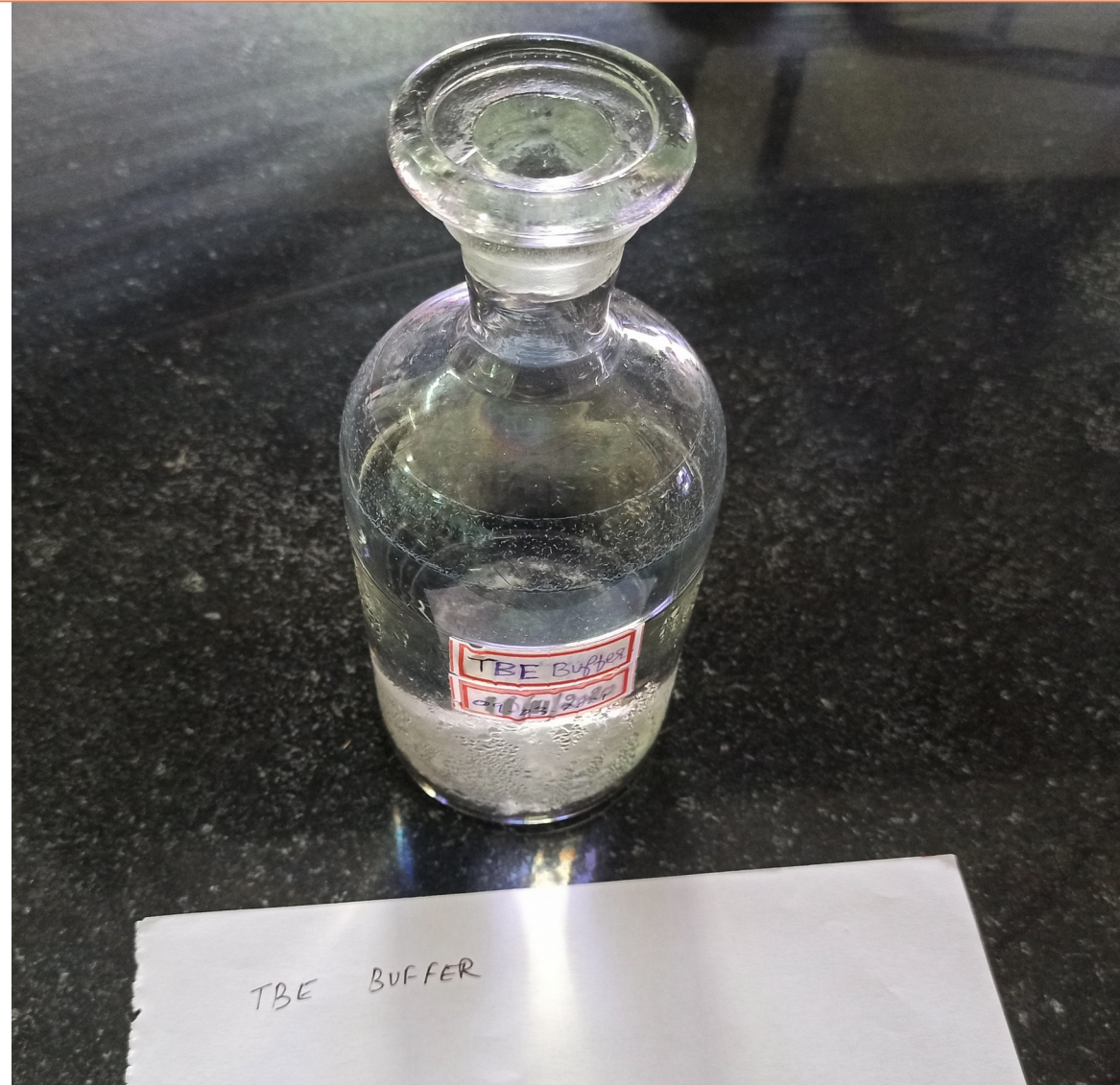
# *TBE Buffer*

TAE (Tris -acetate-EDTA) and TBE (Tris-borate-EDTA) – pH buffer of 8.6

- Tris a pH buffer.
- Acetic acid provide ions to support conductivity and maintain pH.
- EDTA, prevent brake down of molecules.
  - “all dissolved in water”.
- The important feature of any buffer is that it contains an electrolyte so that it can conduct electricity.

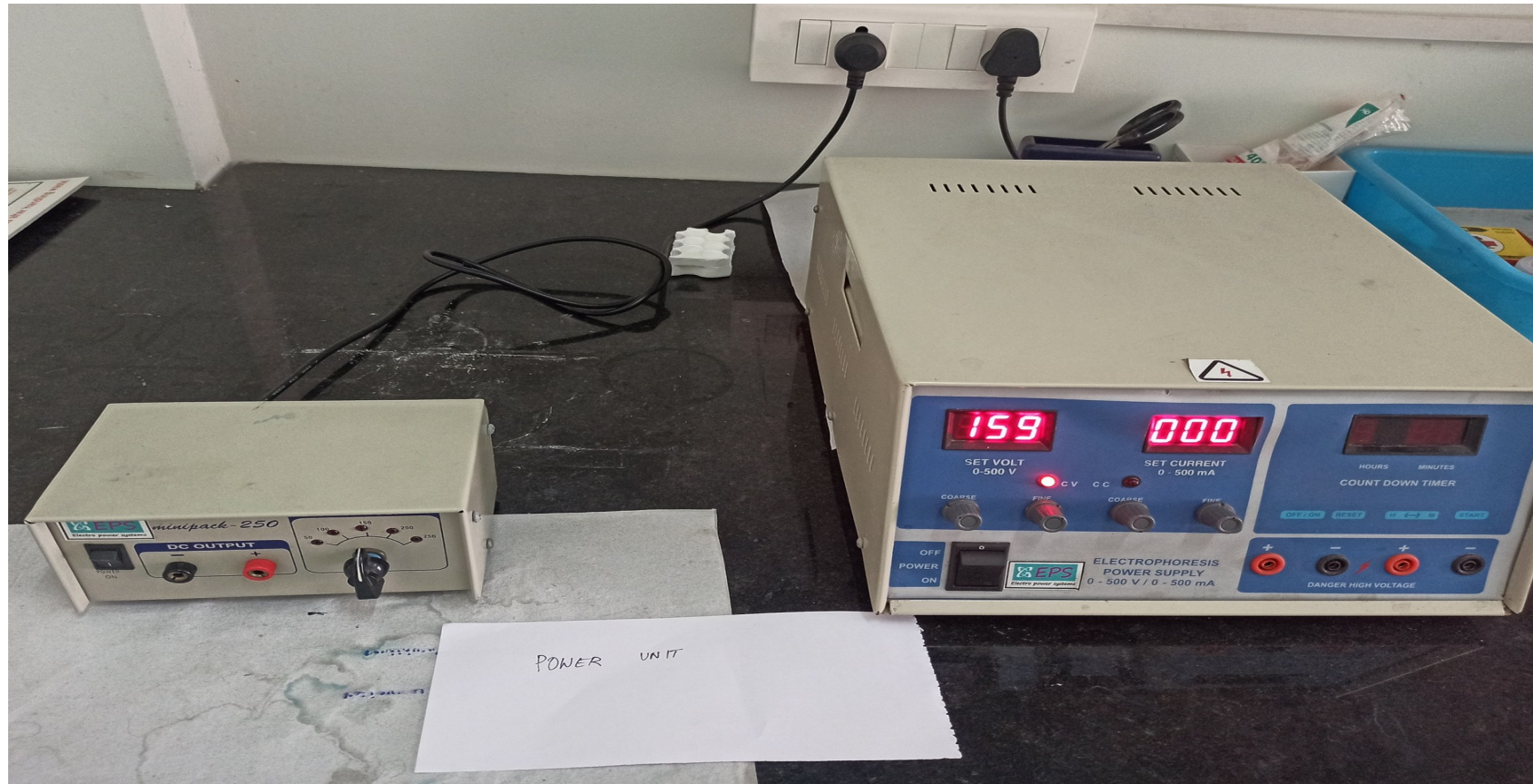






# Power supply

- Drives the movement of ionic species in the medium and allow the adjustment and control of the current or voltage.
- Constant delivery is required.

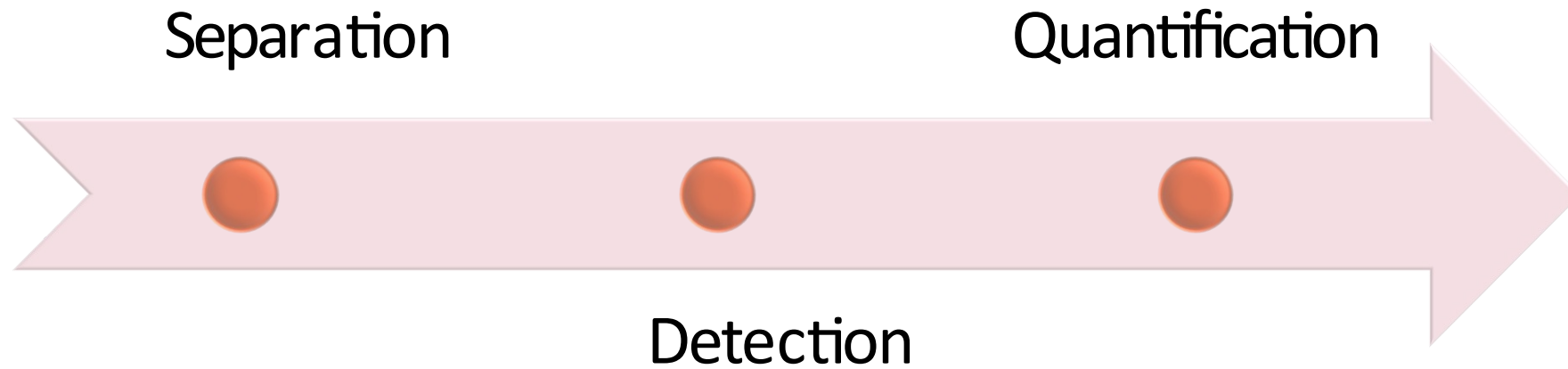


# Power supply

- Flow of current -> Heat produced
  - increase in migration rate -> broadening of separated samples
  - formation of convection currents -> mixing of separated samples
  - thermal instability of heat sensitive samples
  - water loss -> concn of ions -> decrease of buffer viscosity -> decrease in resistance
- To minimize problems: use constant-current power supply



# *General operation*



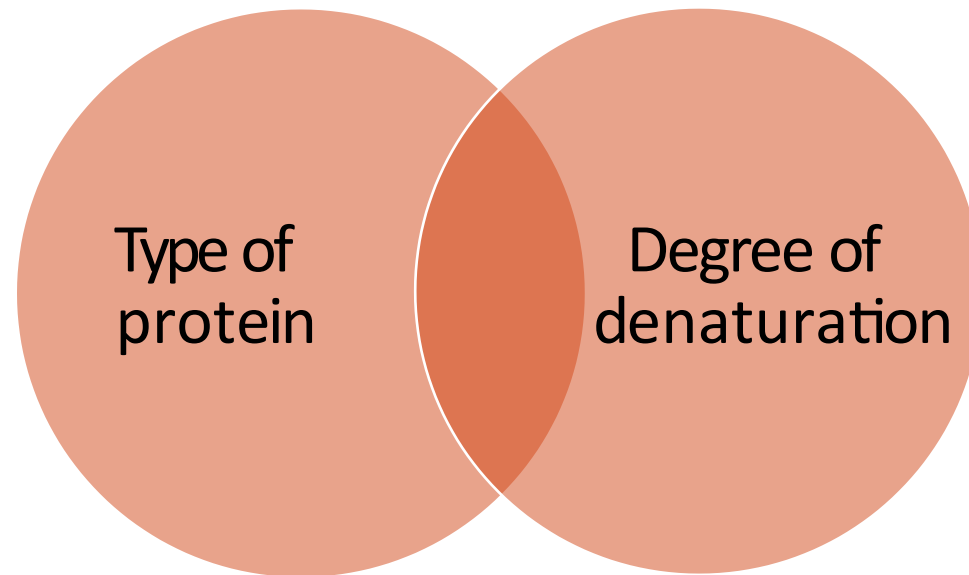
## *Steps performed:*

- Buffer in buffer tank after pH check
- 5-7  $\mu\text{L}$  sample with tracking dye
- Placed in electrode chamber
- Whatmann paper wick is applied
- Current application
- Gel is fixed and dried
- Stained and destained
- Scanned under densitometry



# Staining

- Fixation of Protein - by using acetic acid or methanol (this will prevent diffusion of protein out of the gel when submerged in stain solution)
- Amount of dye taken by sample is affected by many factors,



# *Different stains of Electrophoresis*

## **Plasma Proteins**

- Amido black
- Coomassie Brilliant Blue
- Bromophenol Blue

## **Hemoglobin**

- Amido black
- Coomassie Brilliant Blue
- Ponceau Red

## **Lipoproteins**

- Sudan Black
- Oil red O

## **DNA ( Fluorescent dyes)**

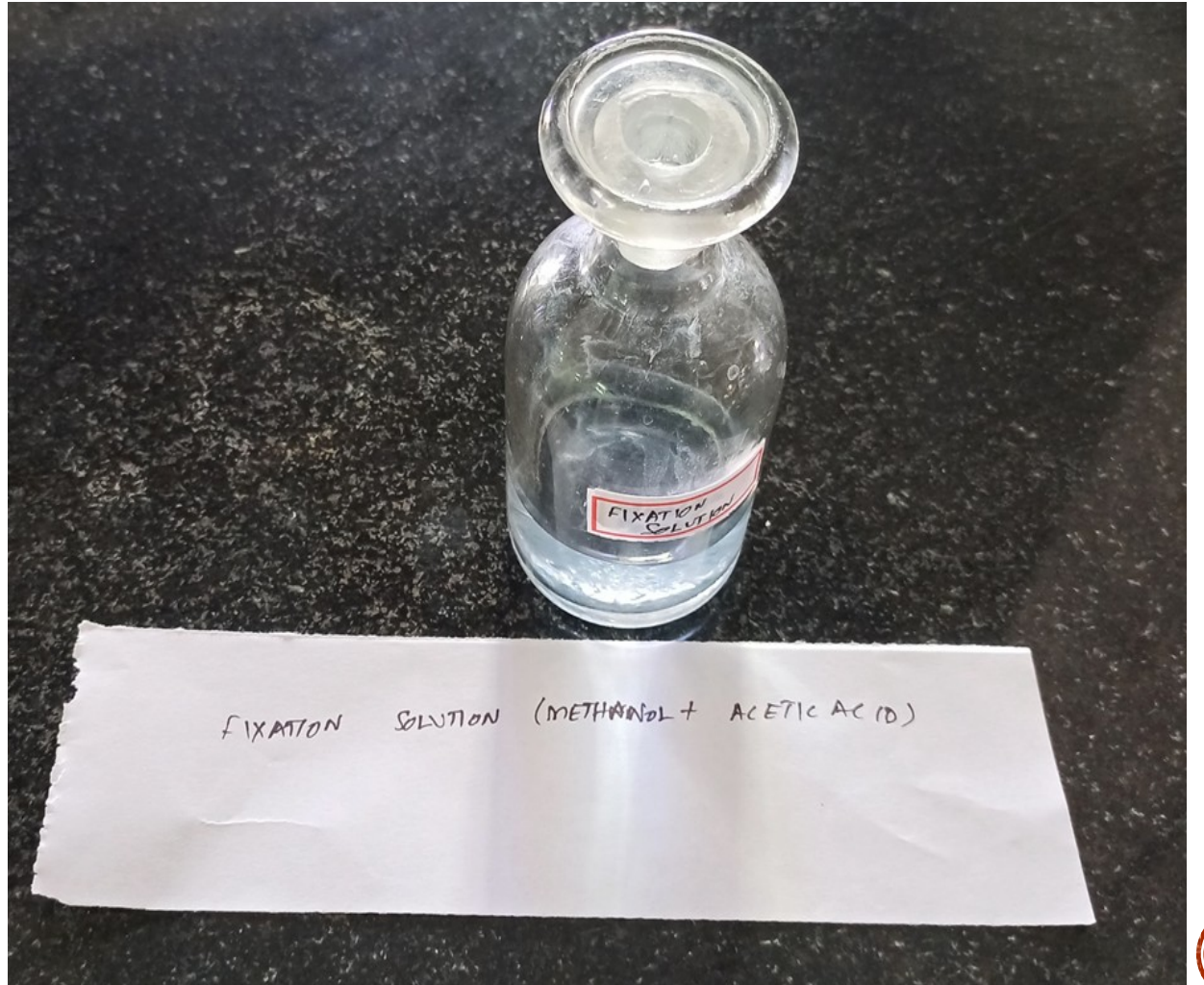
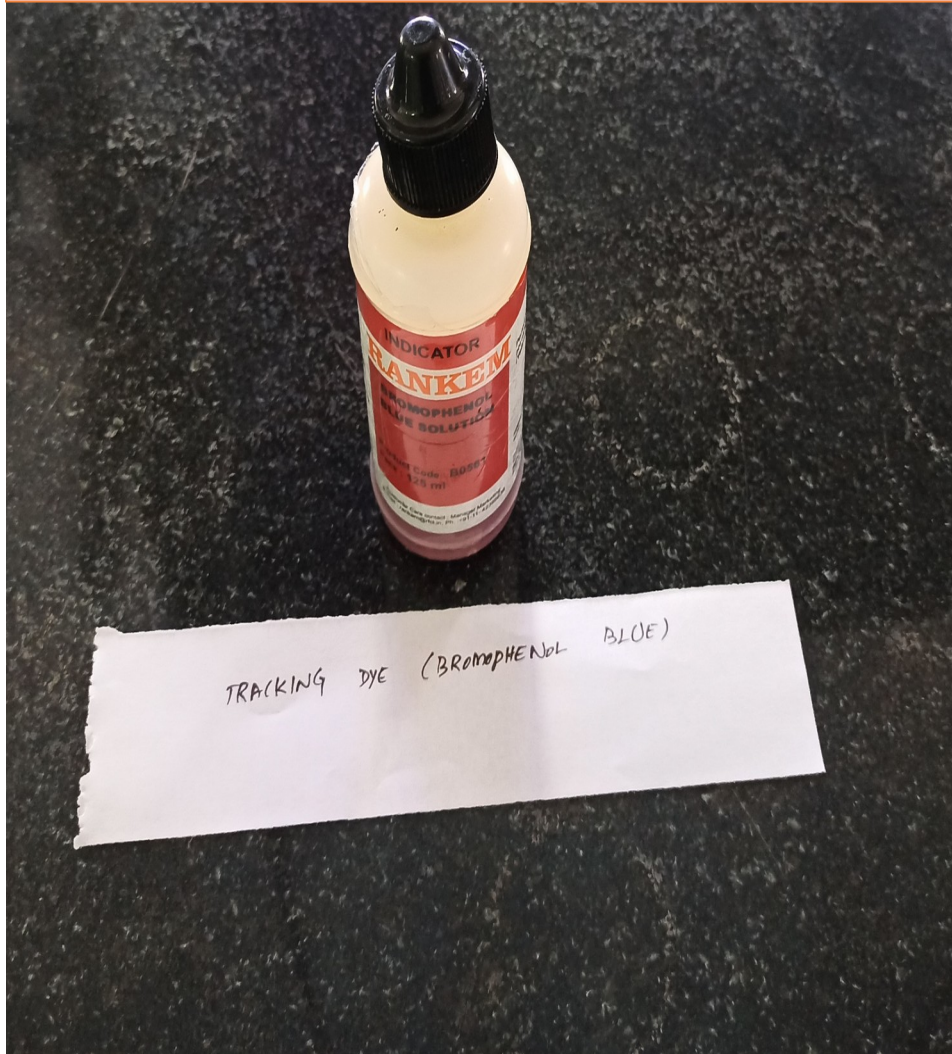
- Ethidium Bromide
- Sybr Green, Sybr Gold

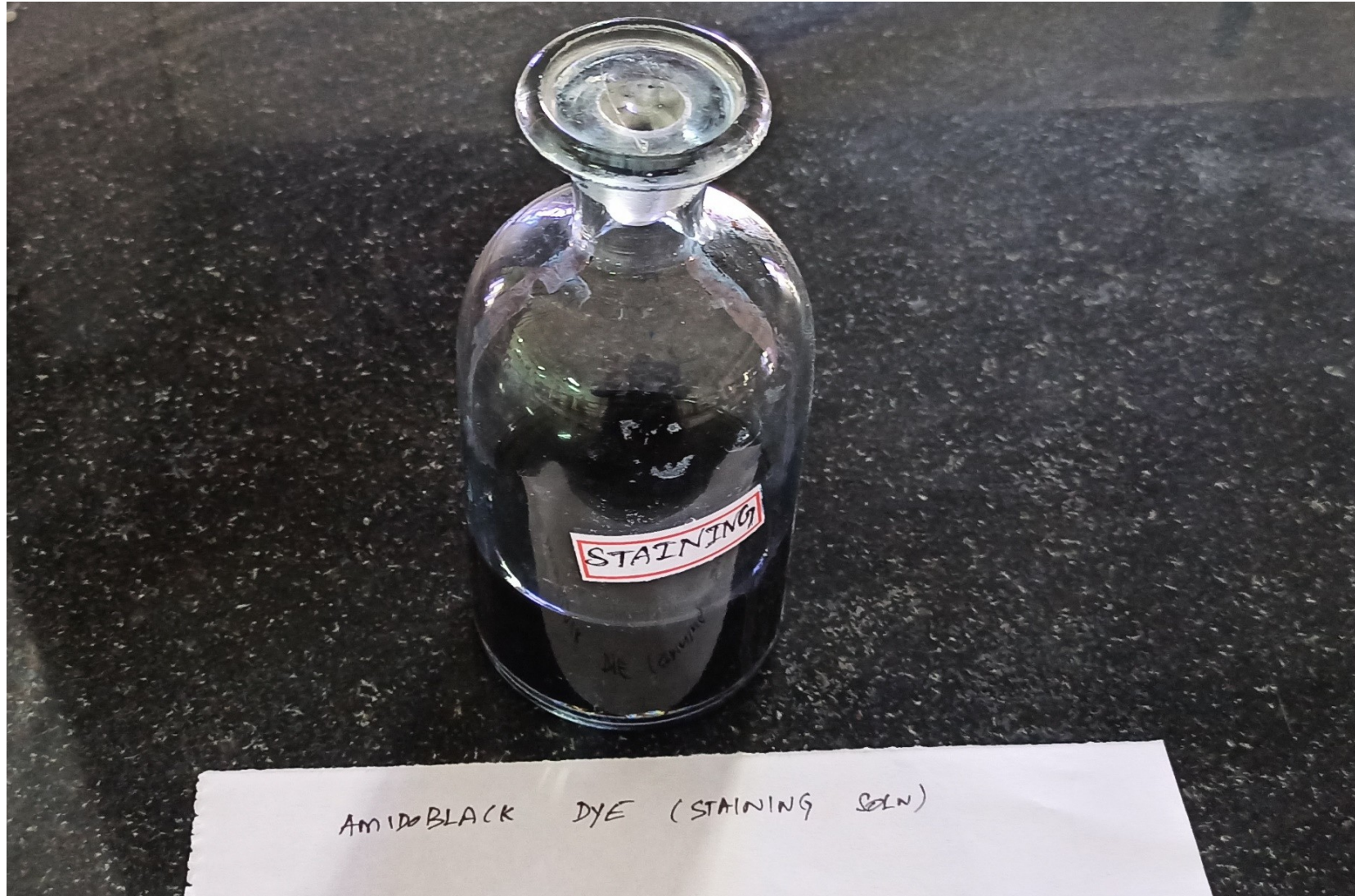


# *Steps in running a sample*

- Tracking the sample with bromophenol blue
- Fixing with methanol & acetic acid
- Staining with amido black
- Destaining with 5 % acetic acid



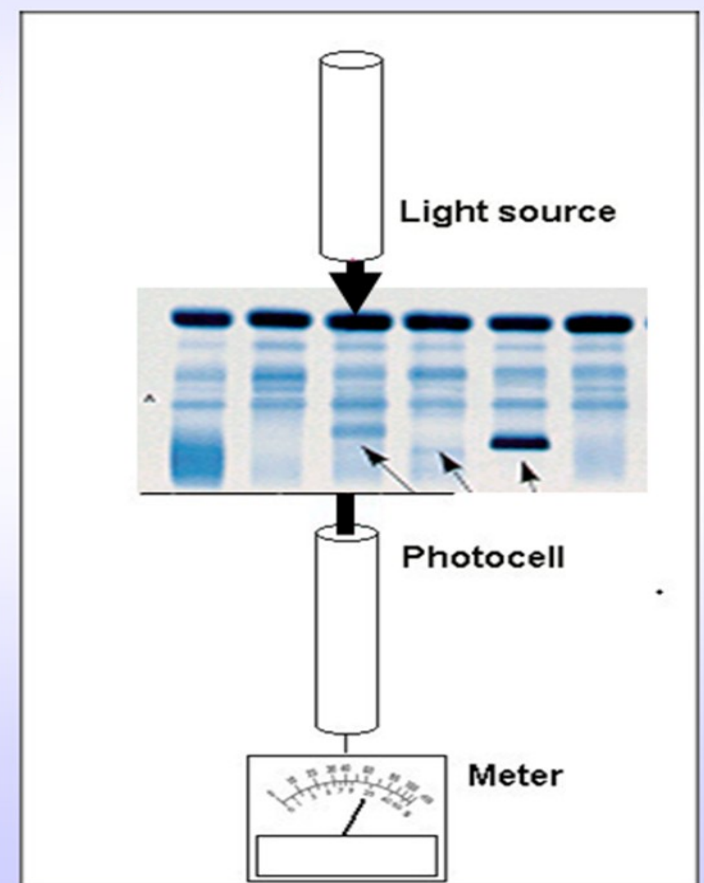




# Detection and Quantification

- Once separated, protein may be detected by staining followed by the quantification using the densitometer.

⌘ Densitometer is a device that measures the degree of darkness in photographic or semi-transparent material.

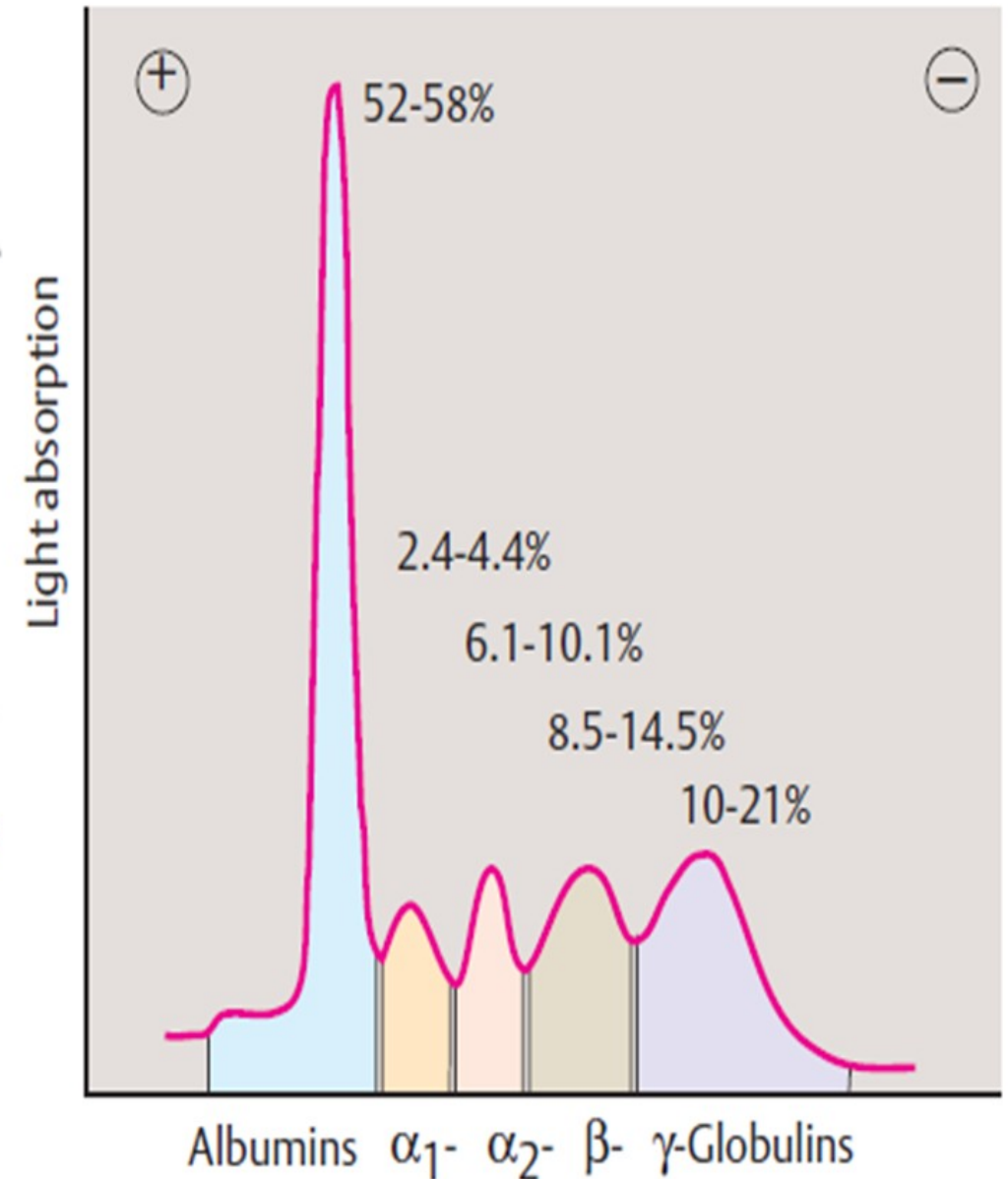
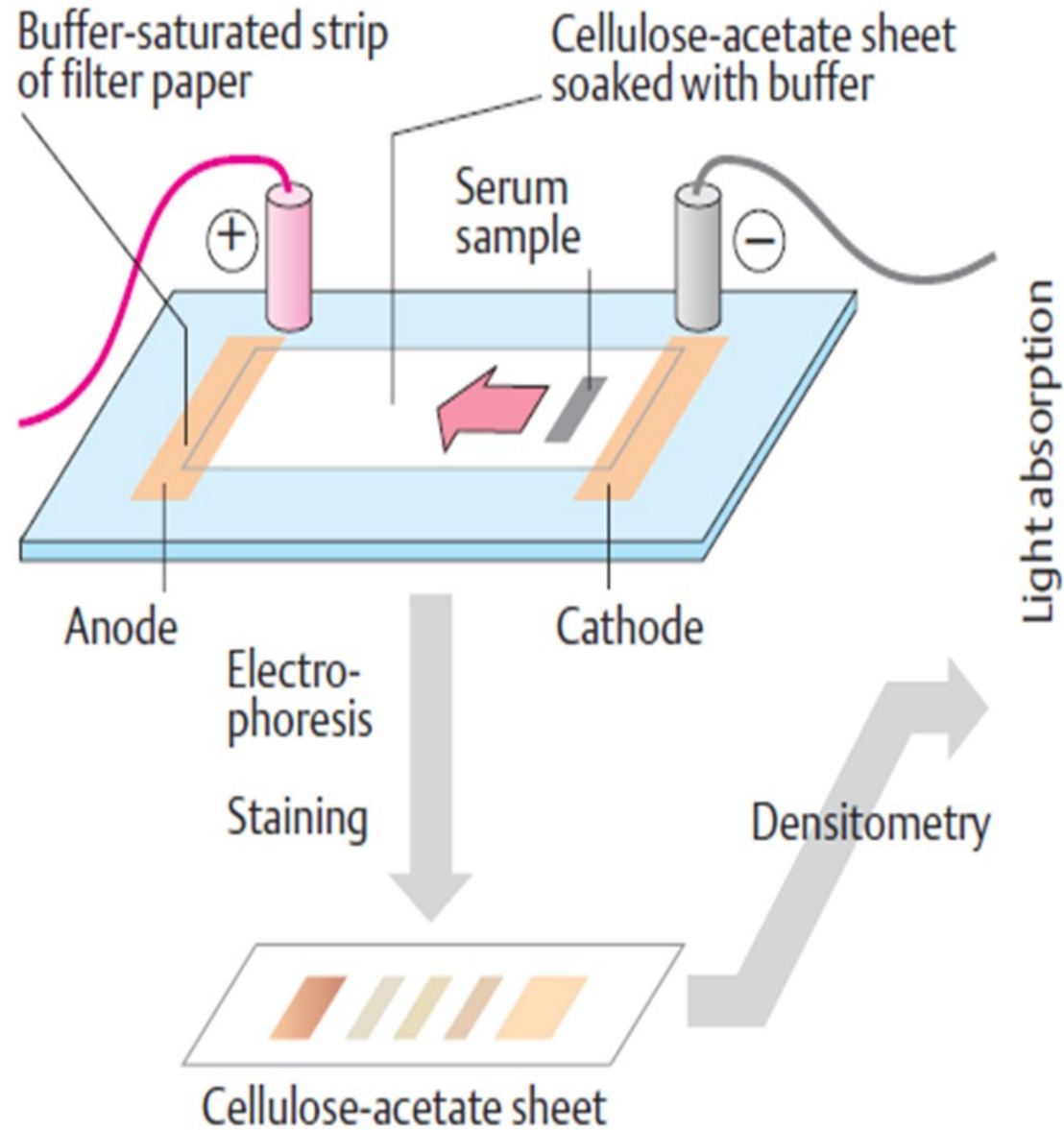


# Common effect of variables on separation

pH	Changes charge of analyte, effective mobility; structure of analyte-denaturing or dissociating a protein.
Ionic strength	Changes in voltage; increased ionic strength reduces migration velocity and increase heating.
Ions present	Change migration speed; cause tailing of bands.
Current	Too high current cause overheating.
Temperature	Overheating cause denature protein; lower temp reduce diffusion but also migration; there is no effect on resolution.
Time	Separation of bands increases linearly with time, but dilution of bands increase with square root of time.
Medium	Major factors are endosmosis and pore size effect, which effect migration velocities.



## B. Electrophoresis



# *Paper Electrophoresis*

- The support medium is a filter paper
- 10 $\mu$ l serum is applied in the form of thin line on hydrated Whatman no1 filter paper
- Chamber are filled with buffer (pH 8.6) and constant current of 1-2 mA
- Time duration to run the test is 10-16 hrs
- Paper is then stained with Bromophenol blue to visualize individual bands
- Drawback- long time interval and blurring of margins



# *Cellulose Acetate Membrane Electrophoresis*

- Preferred solid support media
- Less time consuming
- Excellent separation
- No blurring of margins
- Membranes can be stored for a longer time
- **Widely used for separation of Proteins, lipoproteins and hemoglobin variants**

# *Poly Acrylamide Gel Electrophoresis (PAGE)*

- Most popular type
- Polyacrylamide is a polymer formed when acrylamide is heated with a variety of catalysts with or without cross linking agents
- It is thermostable, transparent, strong and relatively chemically inert
- Gels are uncharged and are prepared in a variety of pore sizes
- Proteins are separated on the basis of charge to mass ratio and molecular size, a phenomenon called Molecular sieving.
- PAGE is the backbone of blotting techniques



# *Types of PAGE*

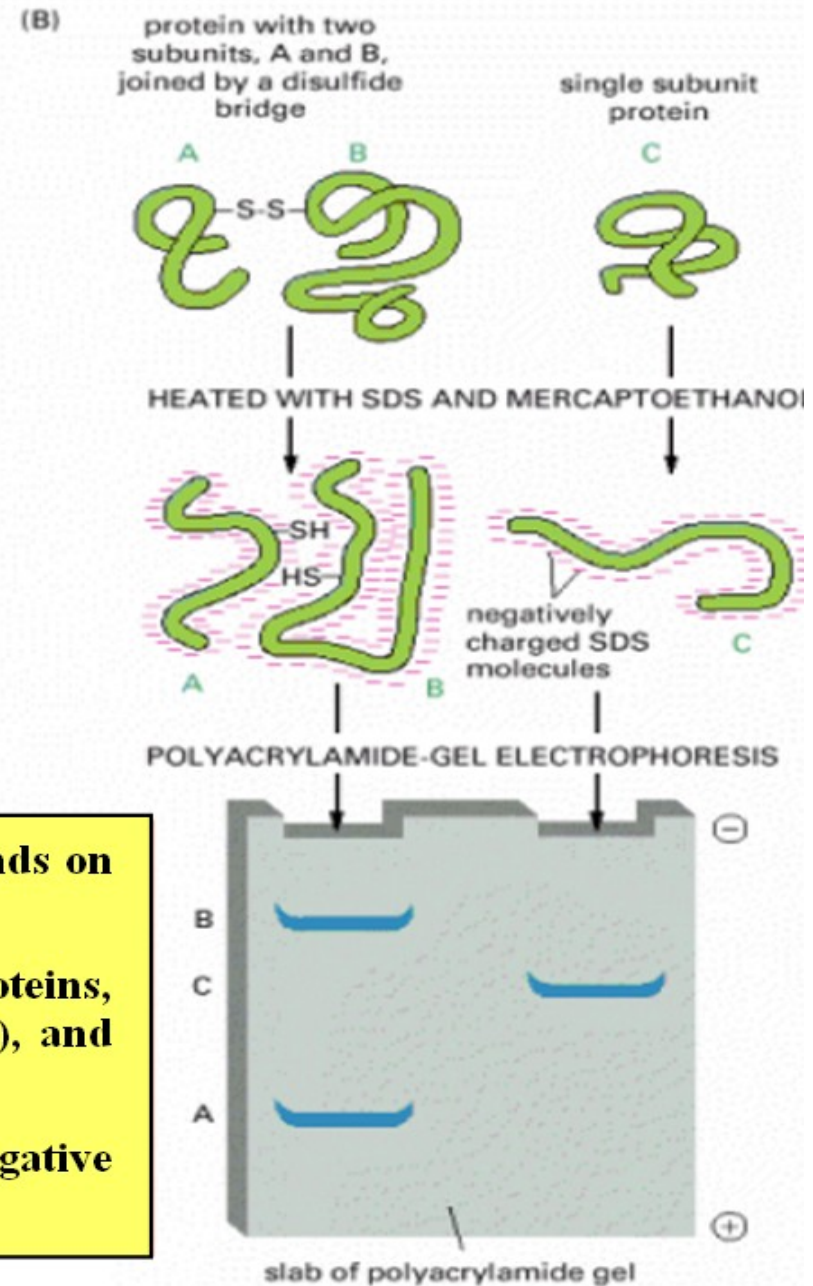
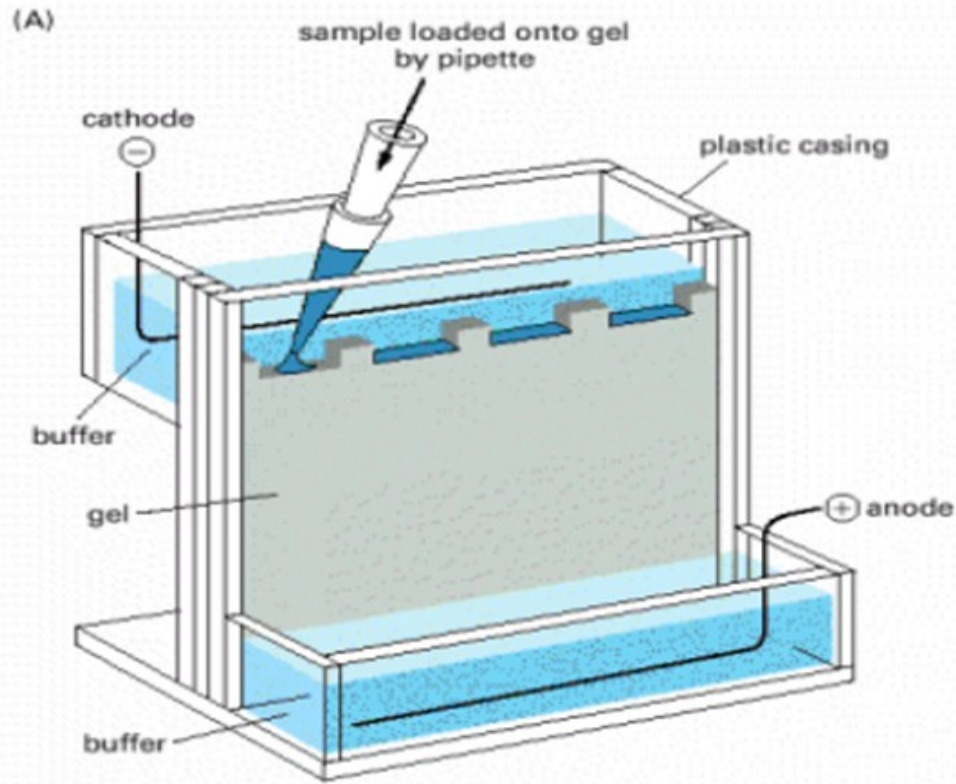
PAGE can be classified according the separation conditions into:

- ***Native-PAGE:*** – Separation is based upon charge, size, and shape of macromolecules. Useful for separation and/or purification of mixture of proteins – This was the original mode of electrophoresis.
- ***Denatured-PAGE or SDS-PAGE*** – Separation is based upon the molecular weight of proteins. The most common method for determining MW of proteins – Very useful for checking purity of protein samples.



# PAGE-Procedure

- The gel of different pore sizes is cast in to a column inside a vertical tube, often with large pore gel at the top and small pore gel at the bottom.
- Microgram quantity of the sample is placed over the top of the gel column and covered by a buffer solution having such a p H so as to change sample components in to anions
- The foot of the gel column is made to dip in the same buffer in the bottom reservoir.
- Cathode and anode are kept above and below the column to impose an electric field through the column
- Rate of migration depends on the charge to mass ratio



The speed of migration in an electrical field depends on the dimension, form and charge of the molecules.

For deaggregation and denaturation of the proteins, SDS,  $\beta$ -mercaptoethanol or DTT (reducing agents), and heat is used.

SDS (strongly anionic detergent) provides negative charge to the proteins.



# SDS-PAGE

**1** Denature sample with sodium dodecylsulfate

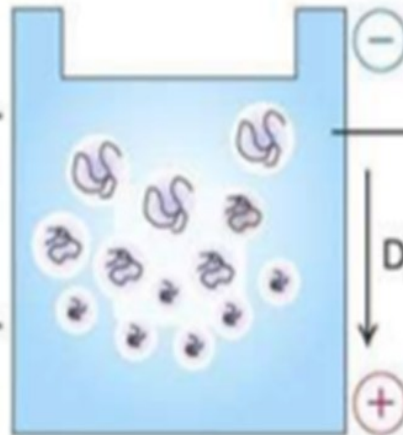


SDS-coated proteins

SDS is an ionic detergent that binds stoichiometrically to number of peptide bonds, thus separates on basis of size (MW).

**2** Place mixture of proteins on gel, apply electric field

Partially separated proteins



Cross-linked polyacrylamide gel

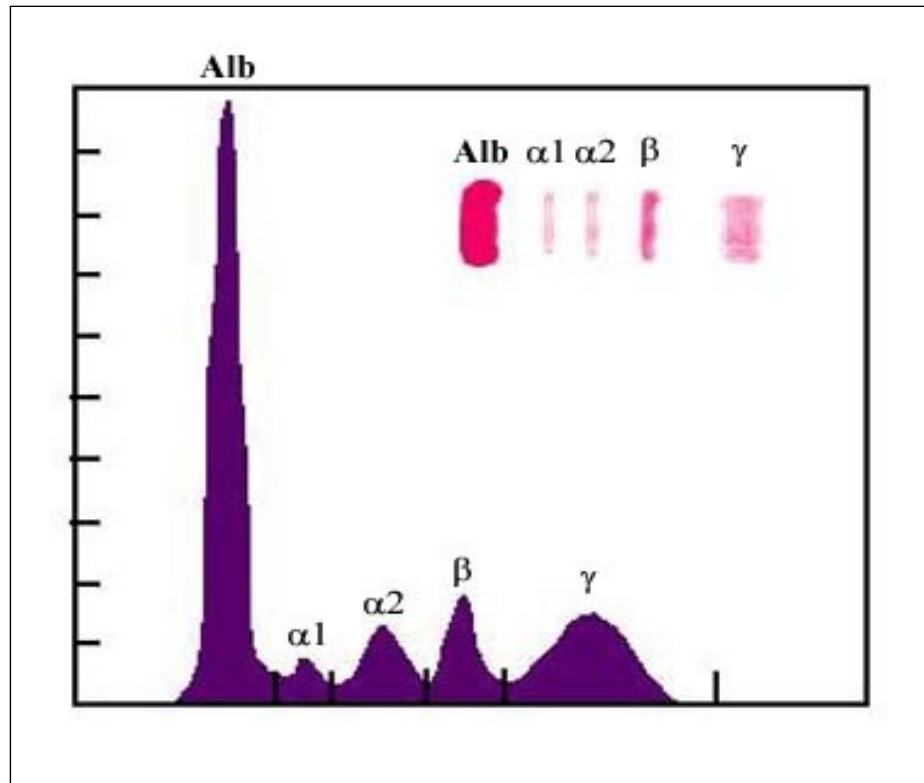
Direction of migration

**3** Stain to visualize separated bands



# *Serum proteins electrophoresis in diagnostics of diseases*

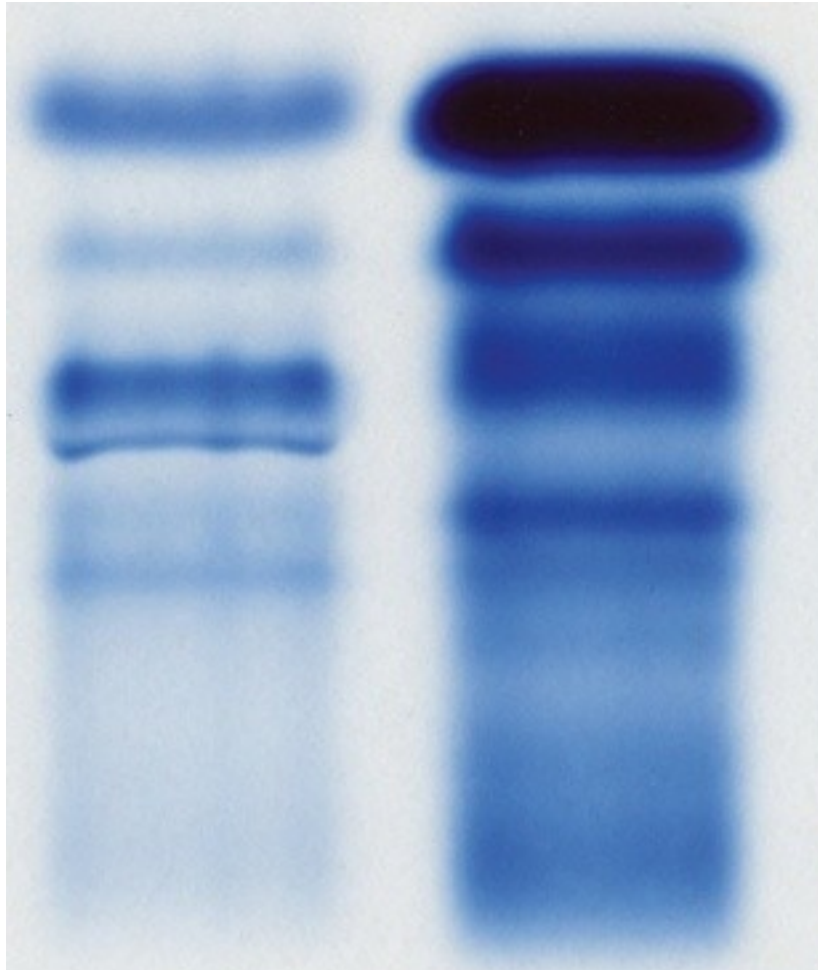
## *Normal pattern*



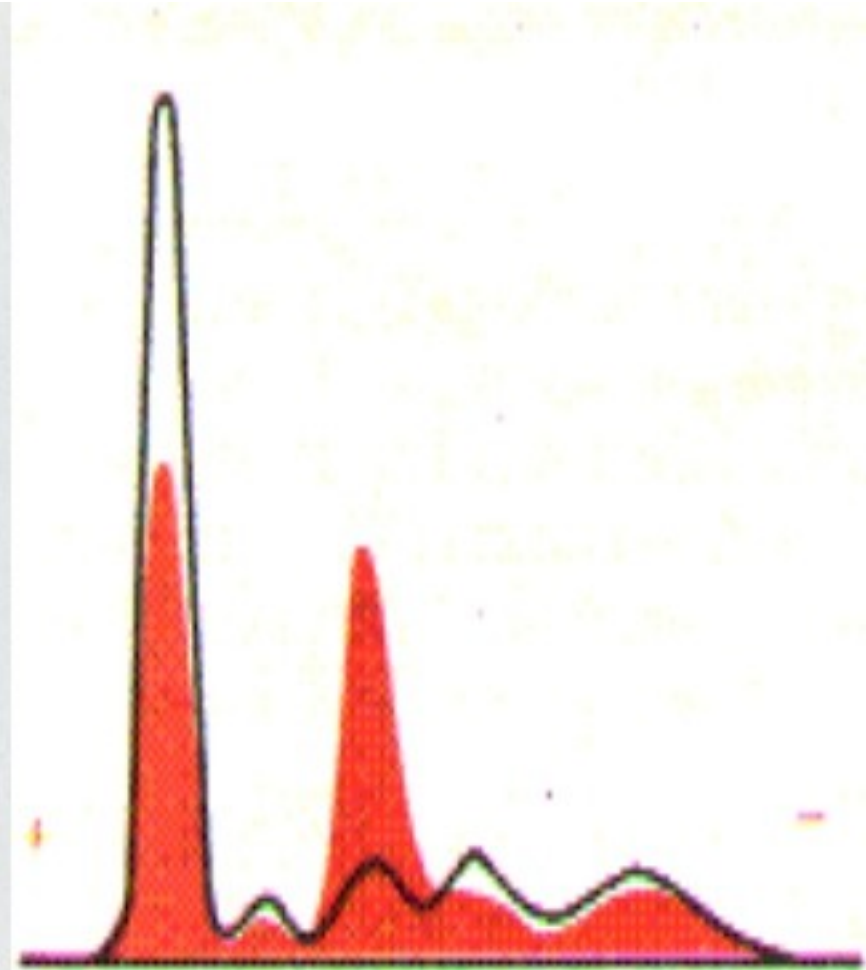
## *Reference ranges:*

Total protein	6.0 – 8.0 g/dL
Albumin	3.5 – 5.0 g/dL
$\alpha 1$ -globulins	0.1 – 0.4 g/dL
$\alpha 2$ -globulins	0.4 – 1.3 g/dL
$\beta$ -globulins	0.6 – 1.3 g/dL
$\gamma$ -globulins	0.6 – 1.5 g/dL



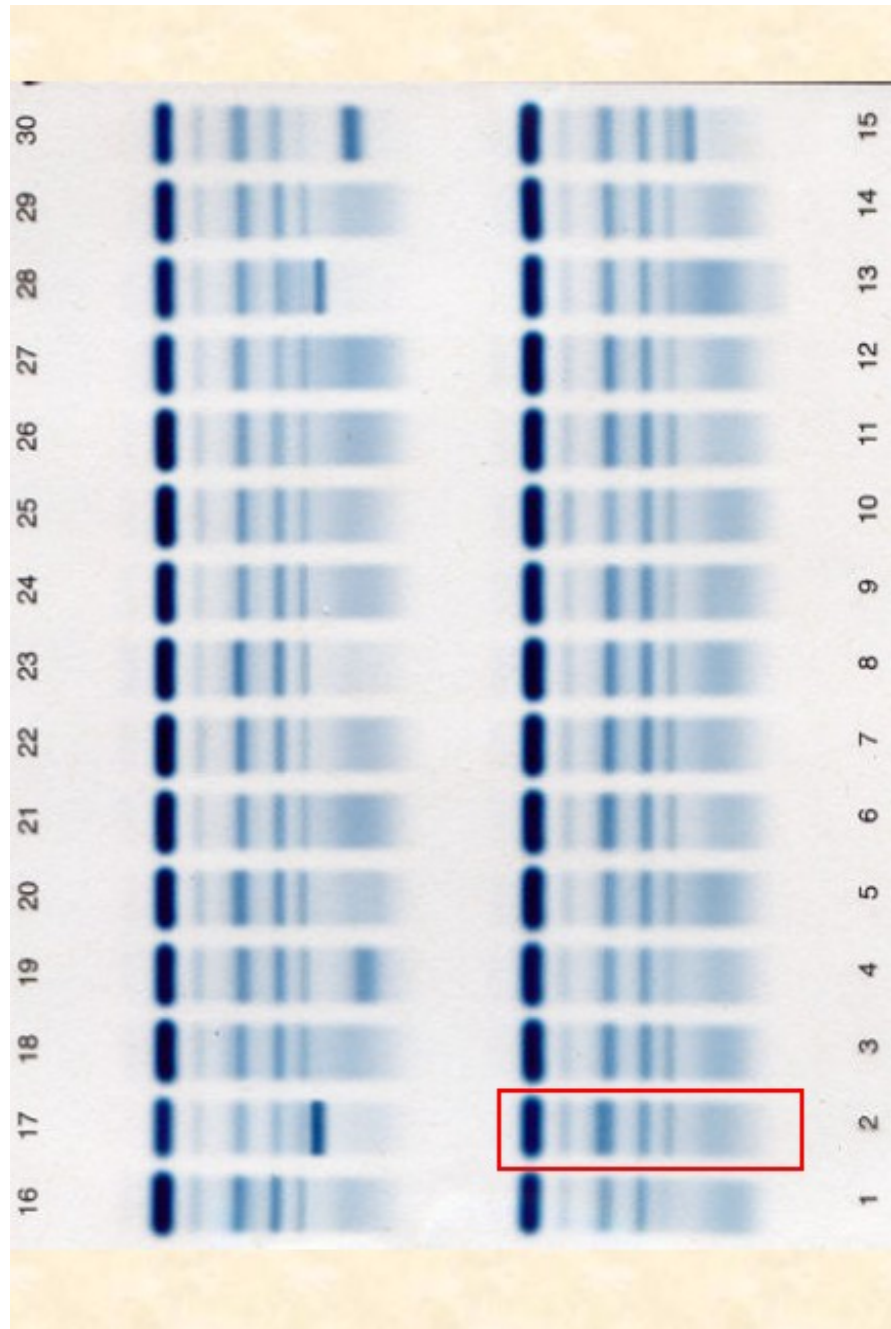


Protein electrophoretic patterns of serum (Ser) and concentrated urine (Ur) in a patient with nephrotic syndrome.



NEPHROTIC SYNDROME





## Selective protein loss



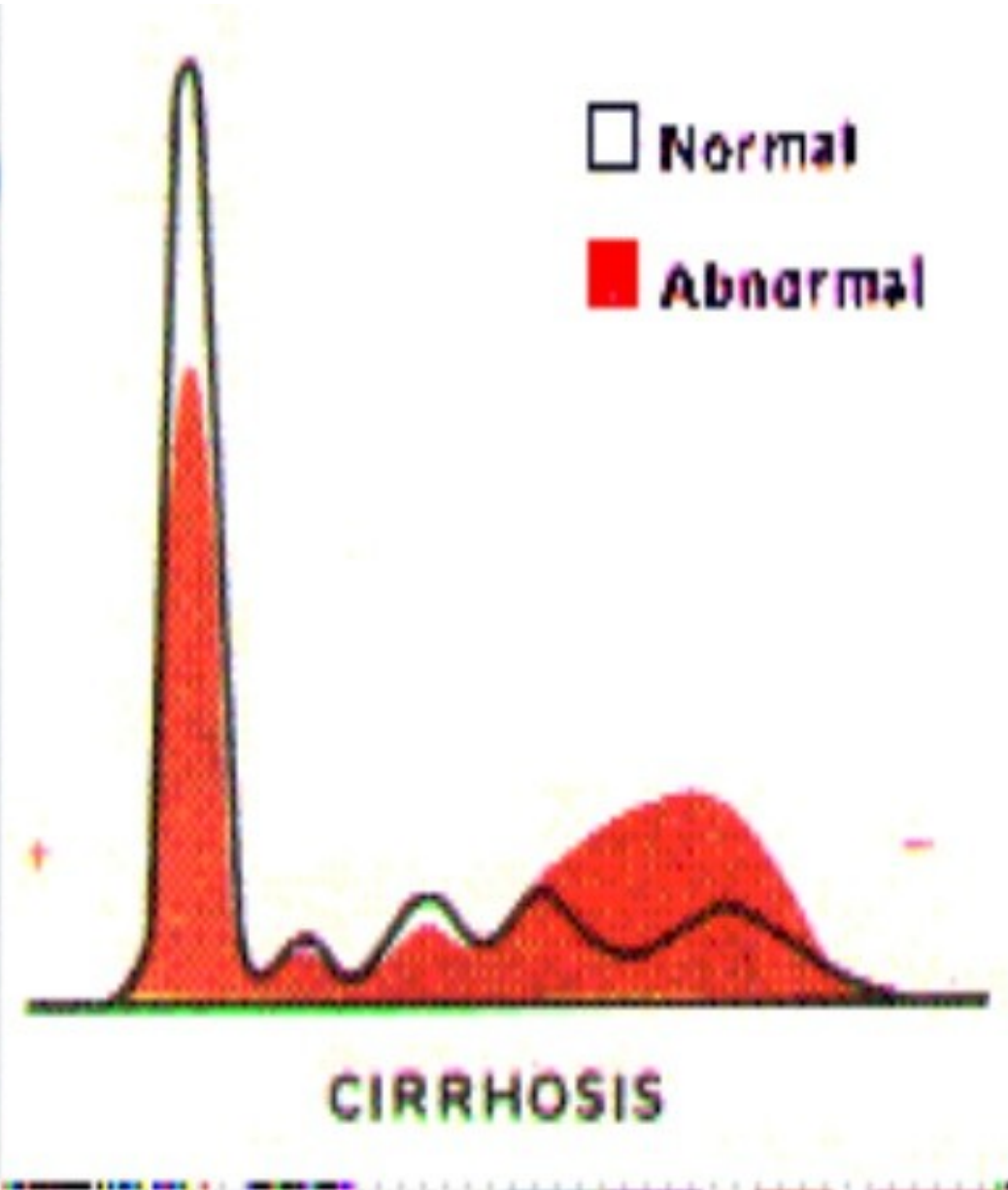
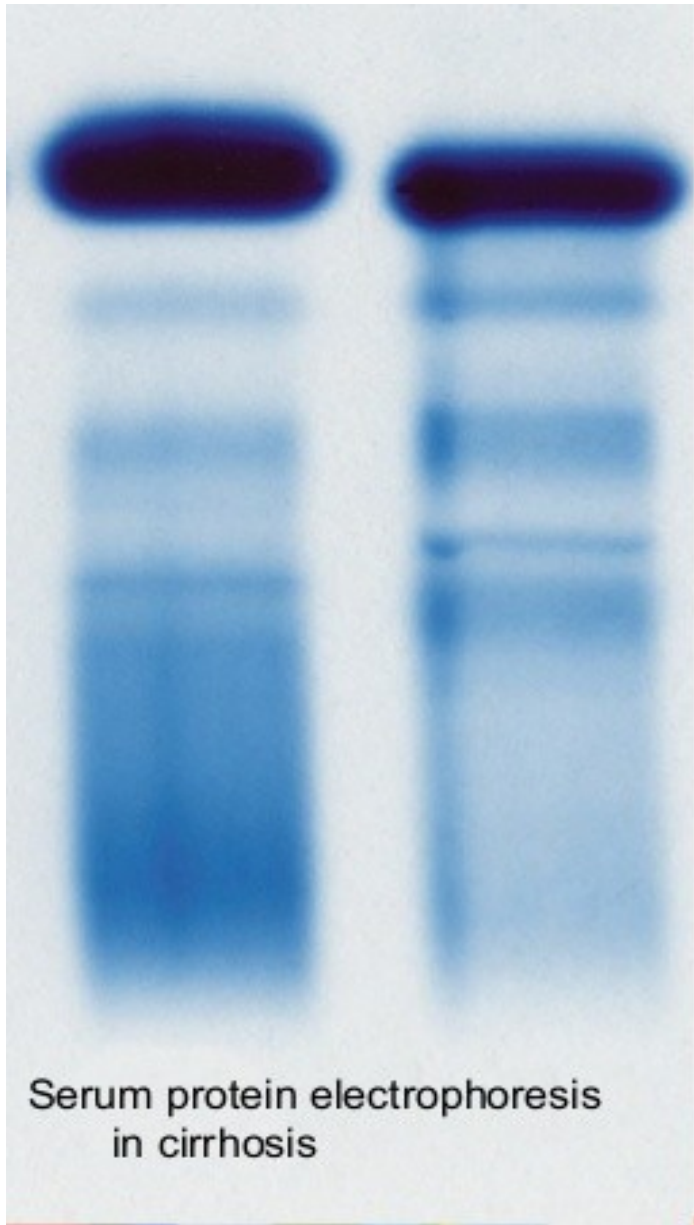
Long-term loss of albumin  
and IgG in kidney

↓ albumin

↑↑  $\alpha_2$  & ↑  $\beta$  globulins

- nephrotic syndrome

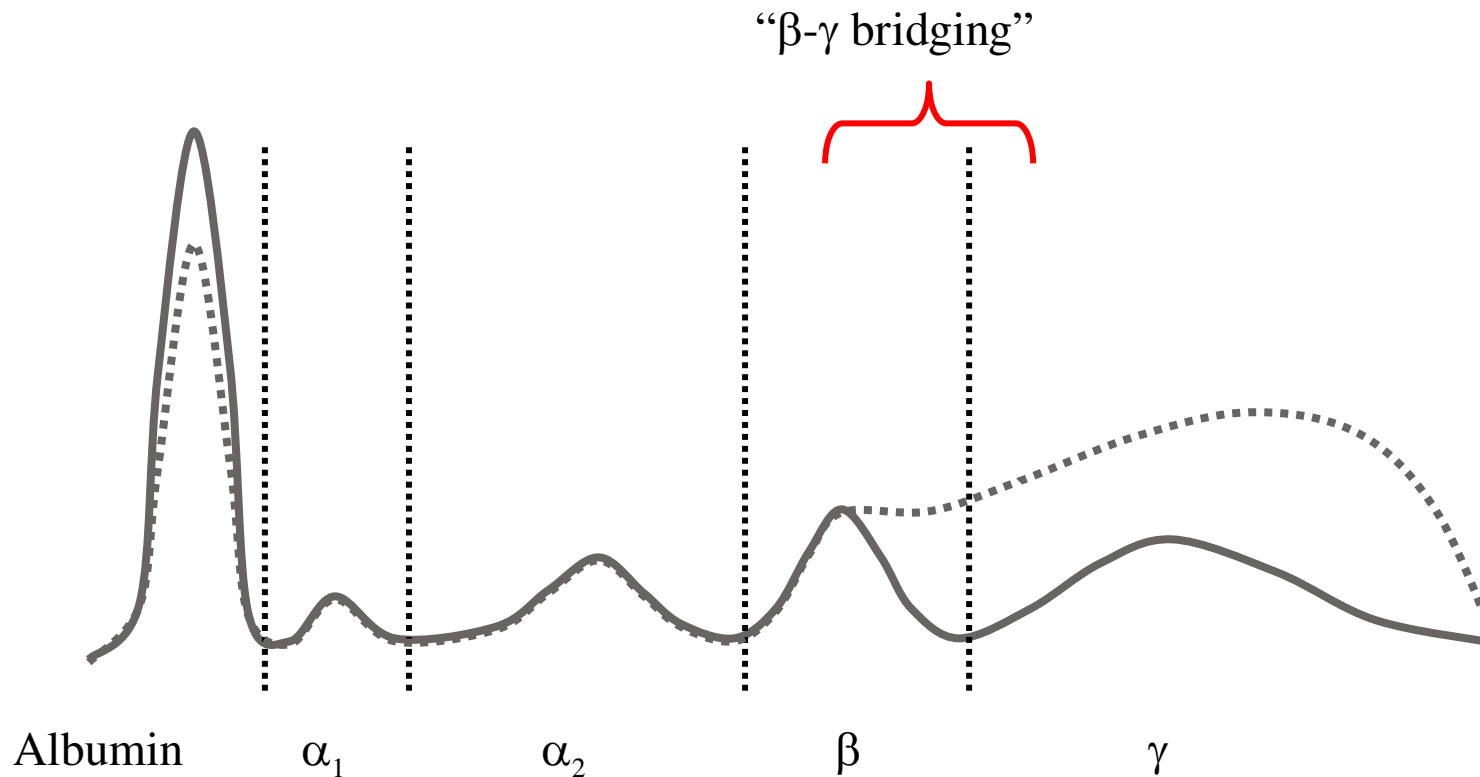


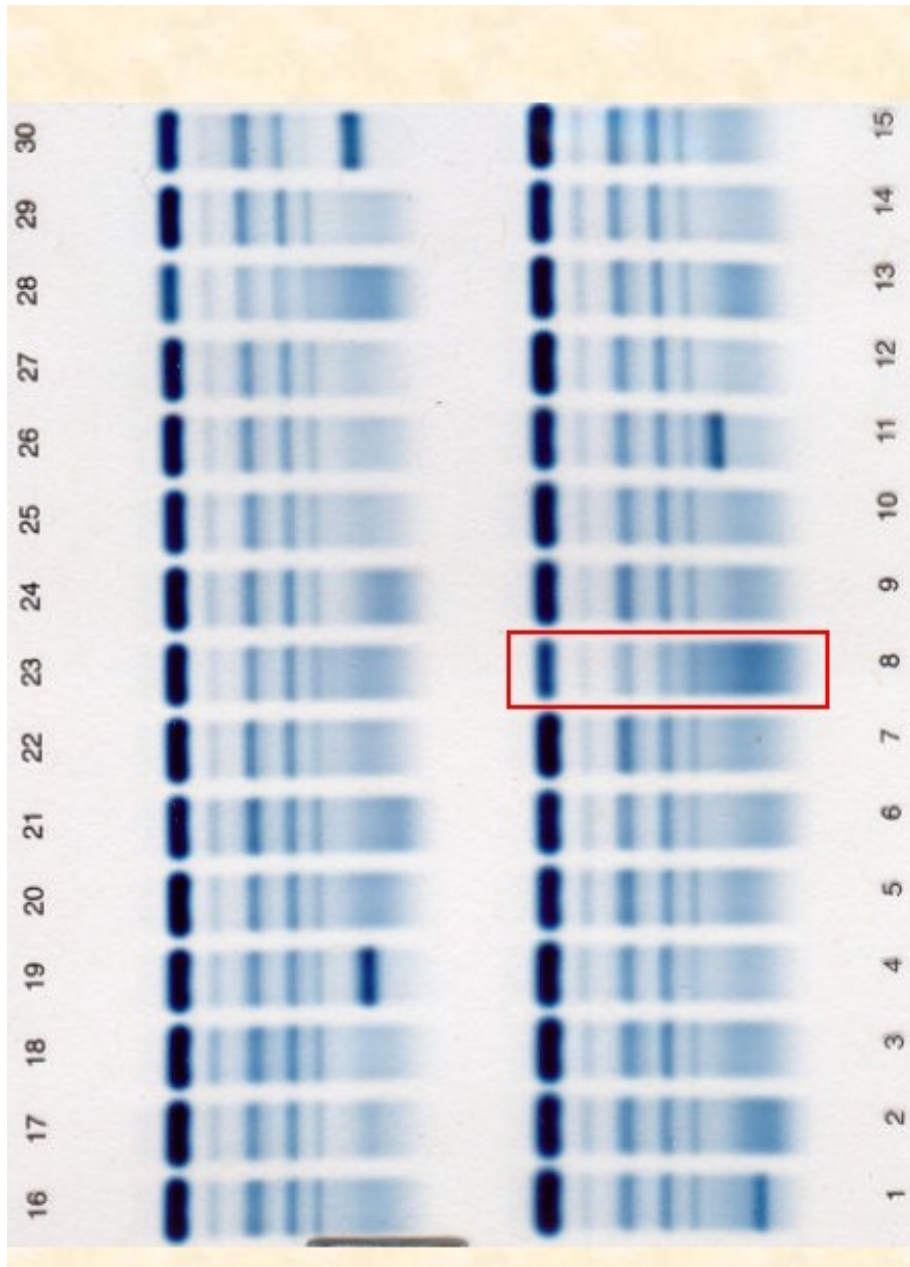


# Hepatic cirrhosis

- Decreased albumin
- Increased  $\gamma$  globulins

Condition	Albumin	Globulins			
		$\alpha$ 1	$\alpha$ 2	$\beta$	$\gamma$
Cirrhosis	↓↓	N	N	↑	↑↑





## $\beta$ - $\gamma$ bridging

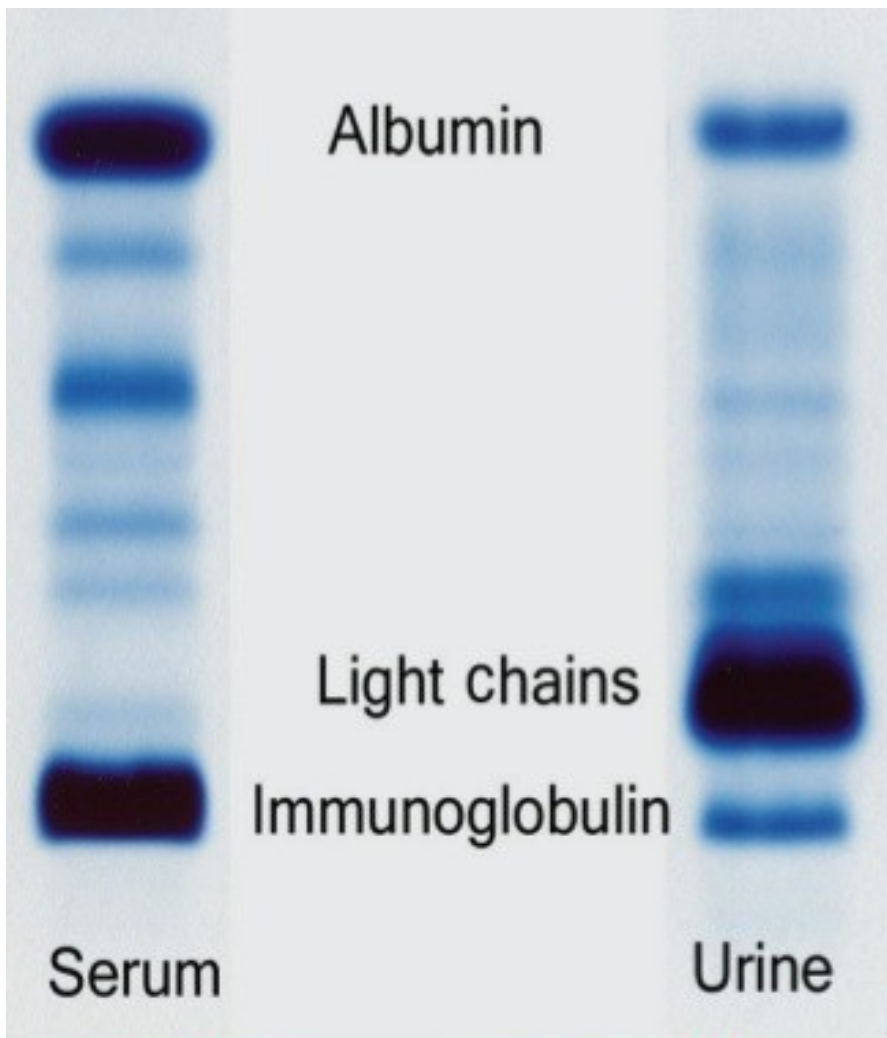


Polyclonal increase in IgA  
extending into beta region

This patient also shows  
decreased albumin

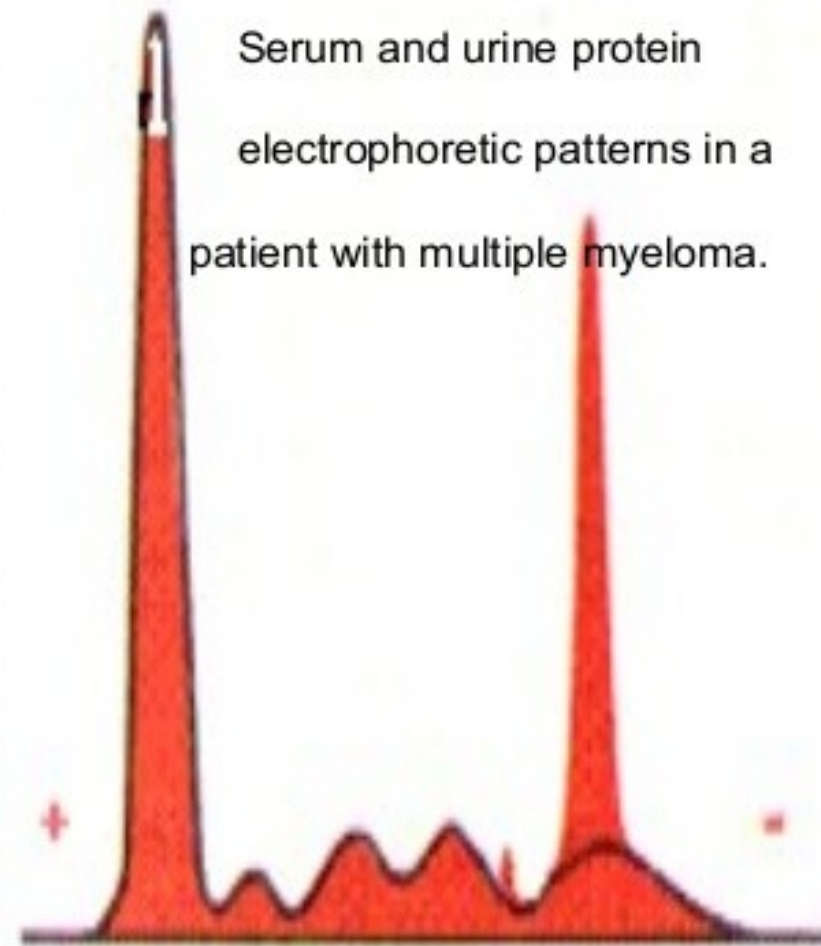
- cirrhotic liver disease
- malignancy
- inflammatory disease





A predominance of the larger complete immunoglobulin.

A large amount of the smaller-sized light chains with only a small amount of the whole



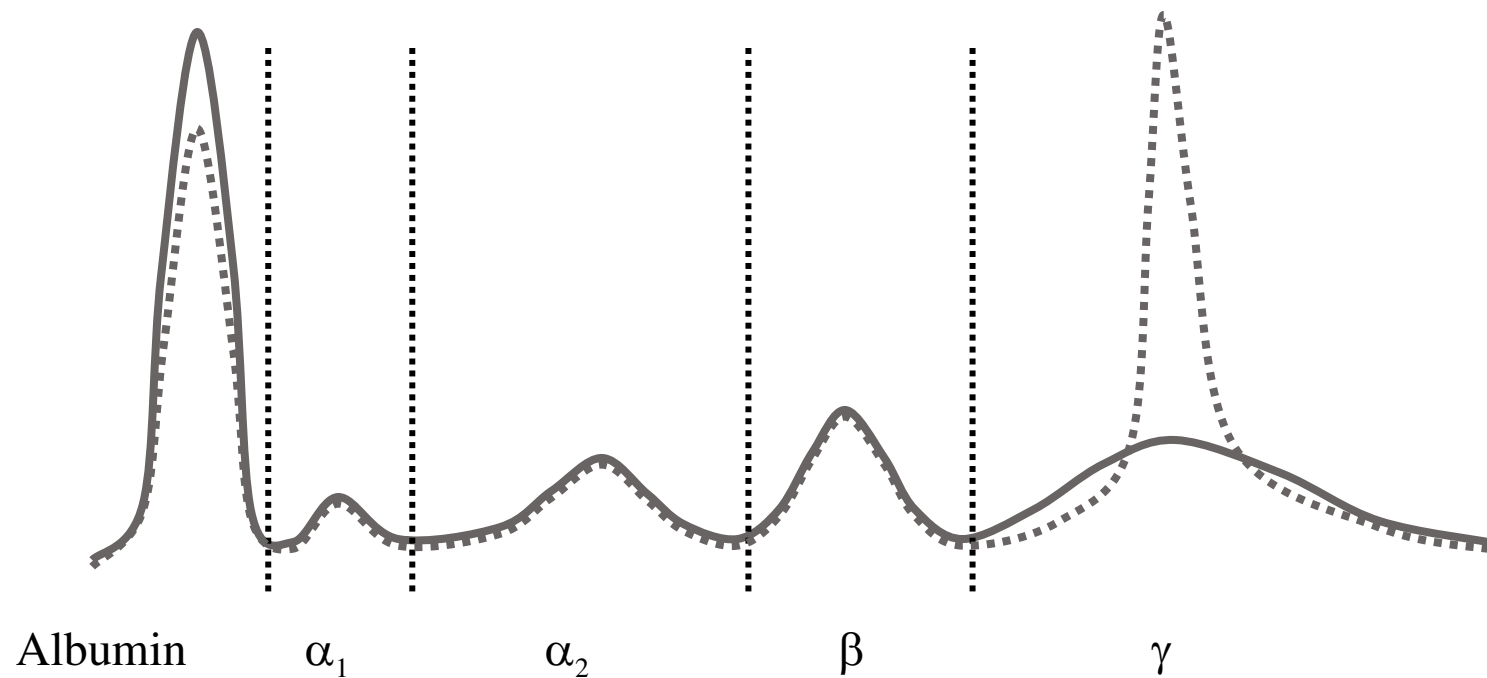
MONOCLONAL GAMMOPATHY

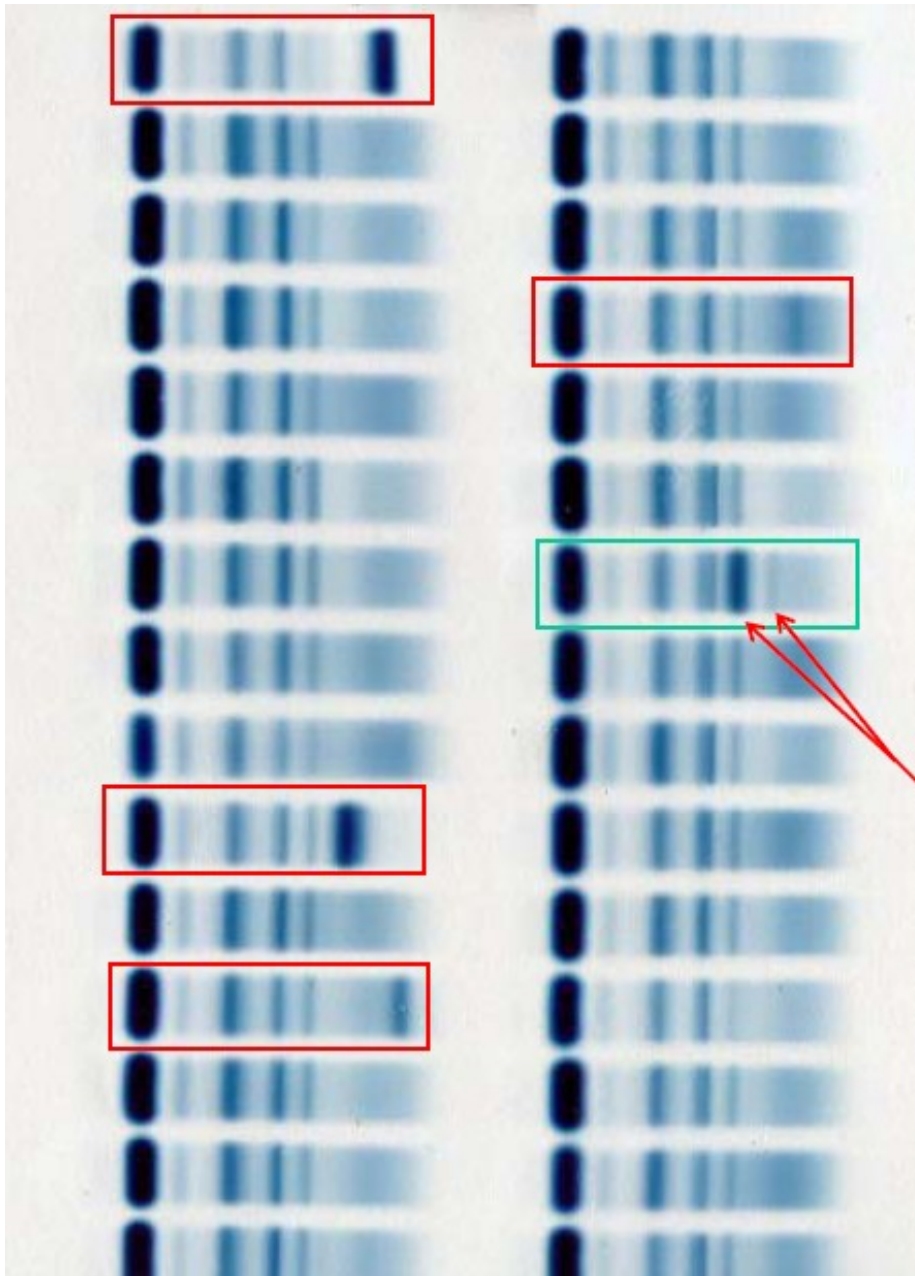


# Monoclonal gammopathy

- Albumin decreased
- Sharp peak in  $\gamma$  region

Condition	Albumin	Globulins			
		$\alpha$ 1	$\alpha$ 2	$\beta$	$\gamma$
M. Myeloma	↓	N	N	N	↑↑





## Monoclonal Gammopathy

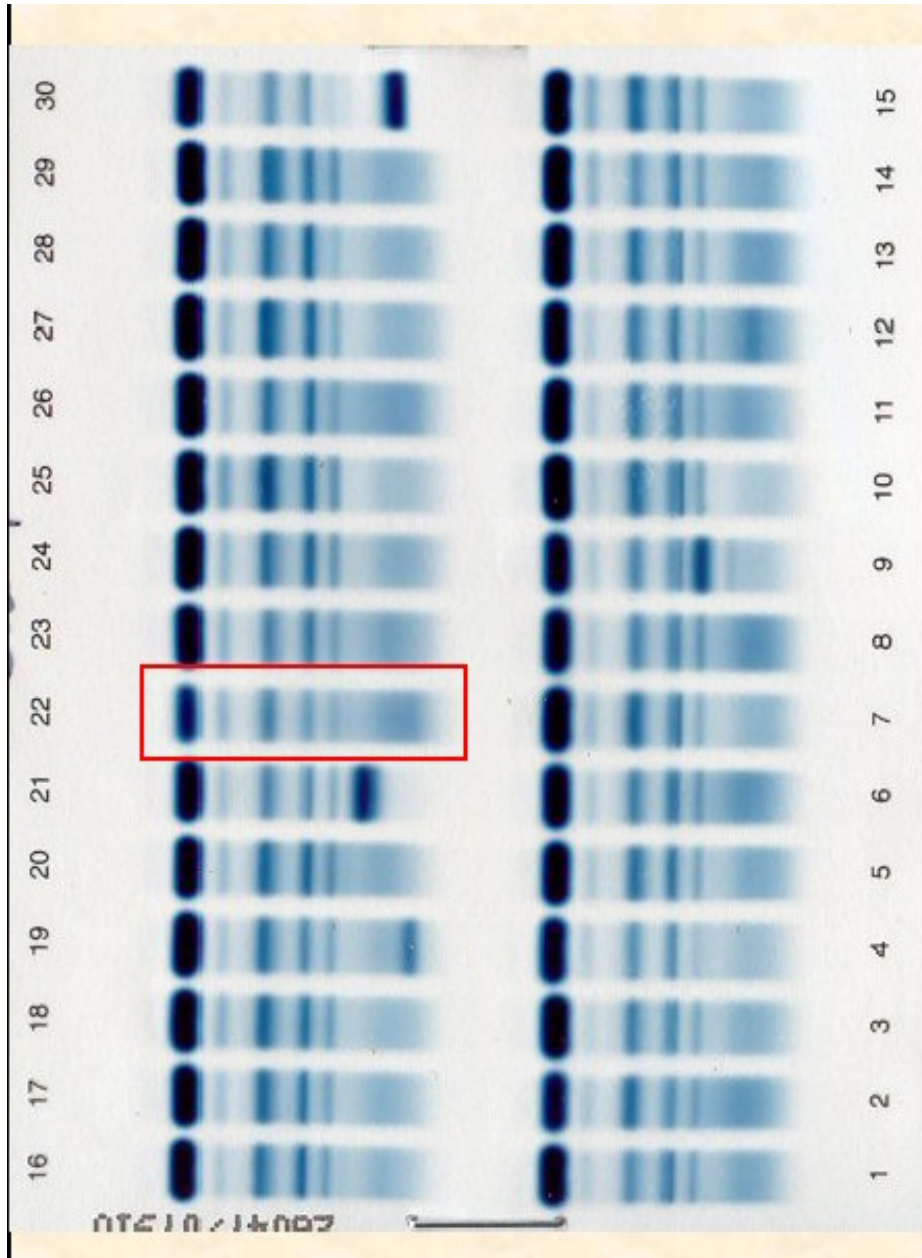
Monoclonal proliferation of  $\beta$ -lymphocytes, producing an abnormal immunoglobulin paraprotein

Discrete band, typically within  $\beta - \gamma$  region

Monoclonal IgA and free light chains may migrate as far as  $\alpha_2$  region

*example of a biconal gammopathy*



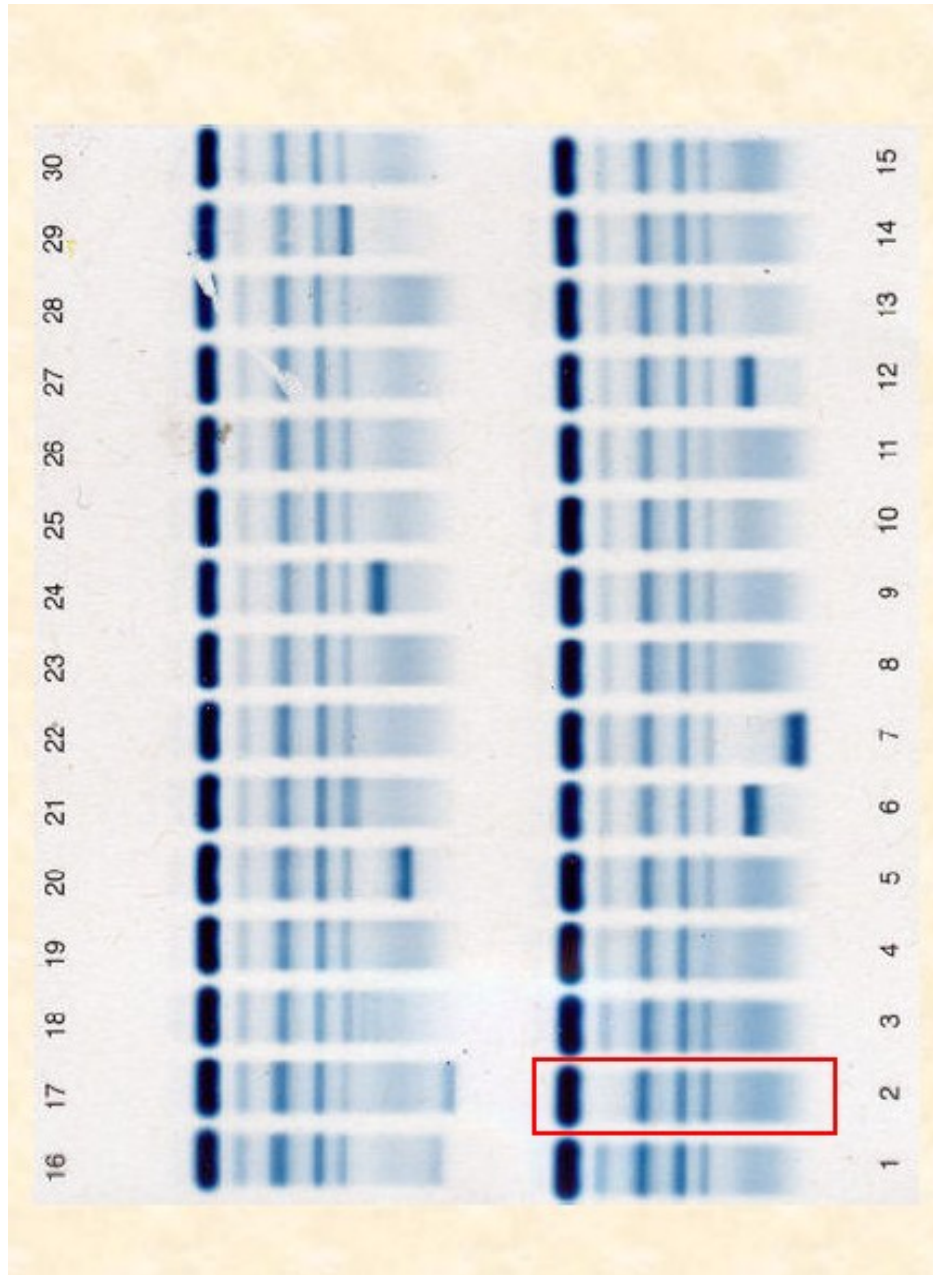


## Hypoalbuminemia



- organ losses
  - renal
  - GI
- liver disease
- malnutrition





Markedly decreased  
alpha-1 globulins



*Isolated* ↓  $\alpha$ 1-AT

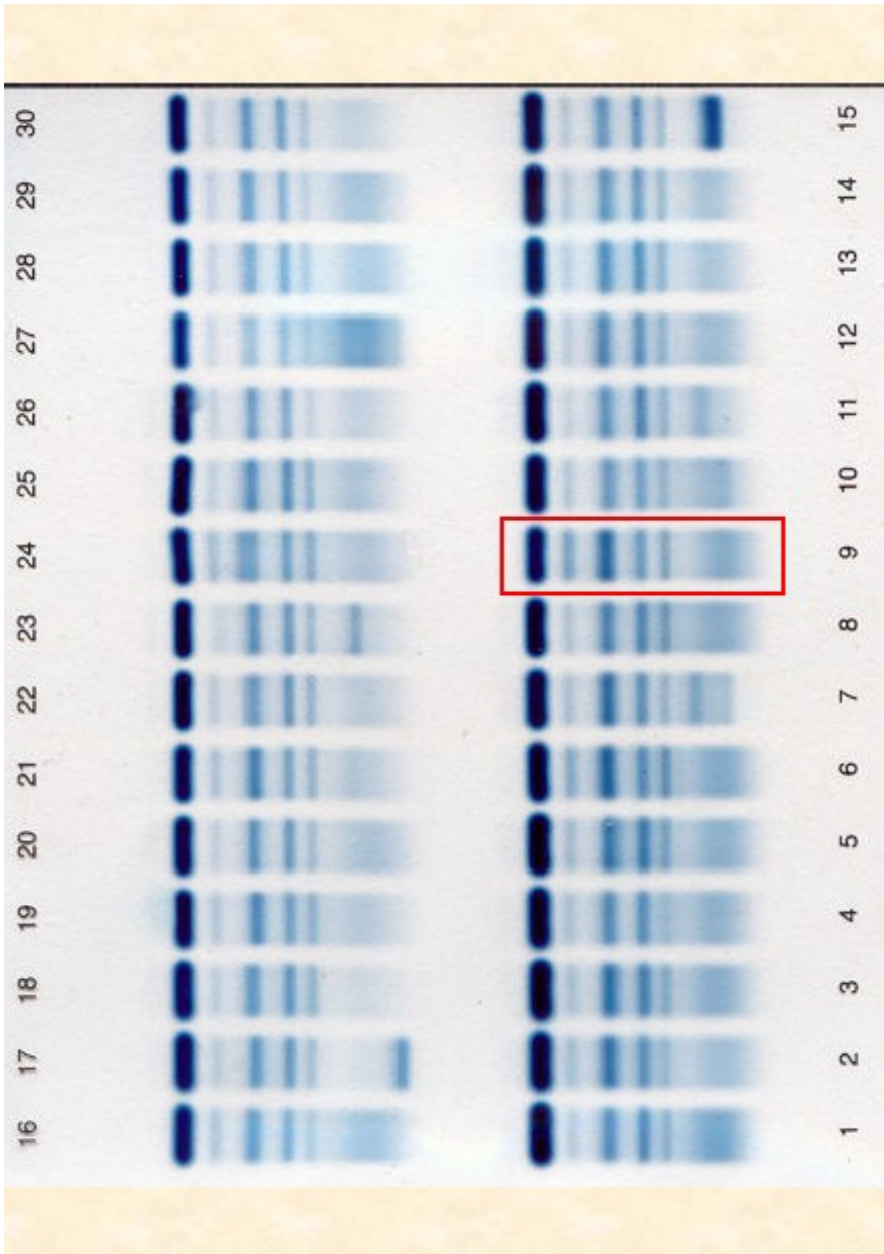
$\alpha$ 1-antitrypsin deficiency

- LL ref range: 1-3 g/L
- suggest phenotyping if <0.6
- PiZZ genotype: 10%  $\alpha$ 1-AT

*Combined with* ↓ albumin

- liver disease
- malnutrition
- protein loss





Increased alpha-2  
globulins

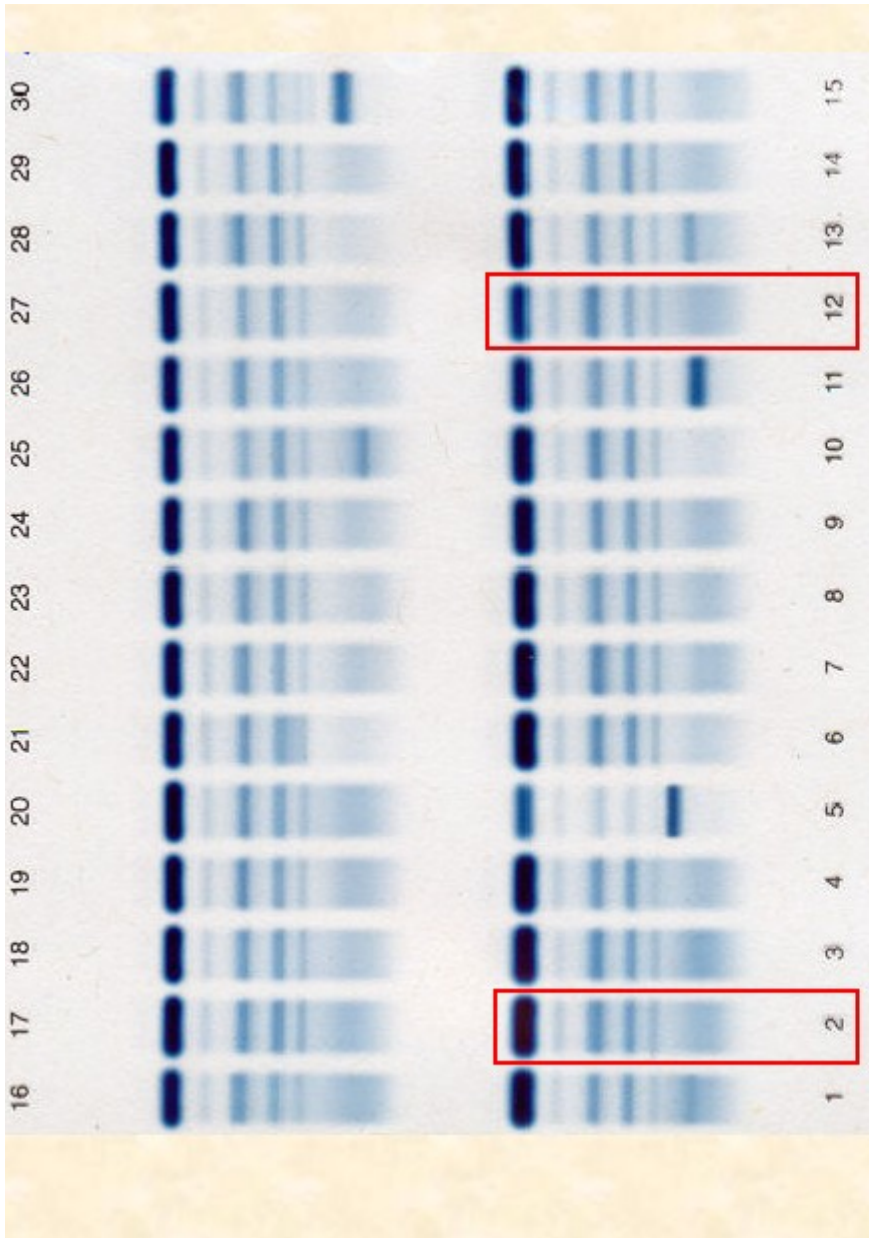


- hemolysis
- ? monoclonal protein

*Combined (eg with  $\uparrow$   $\alpha$ 1)*

- acute phase response
- selective protein loss





## Acute inflammation

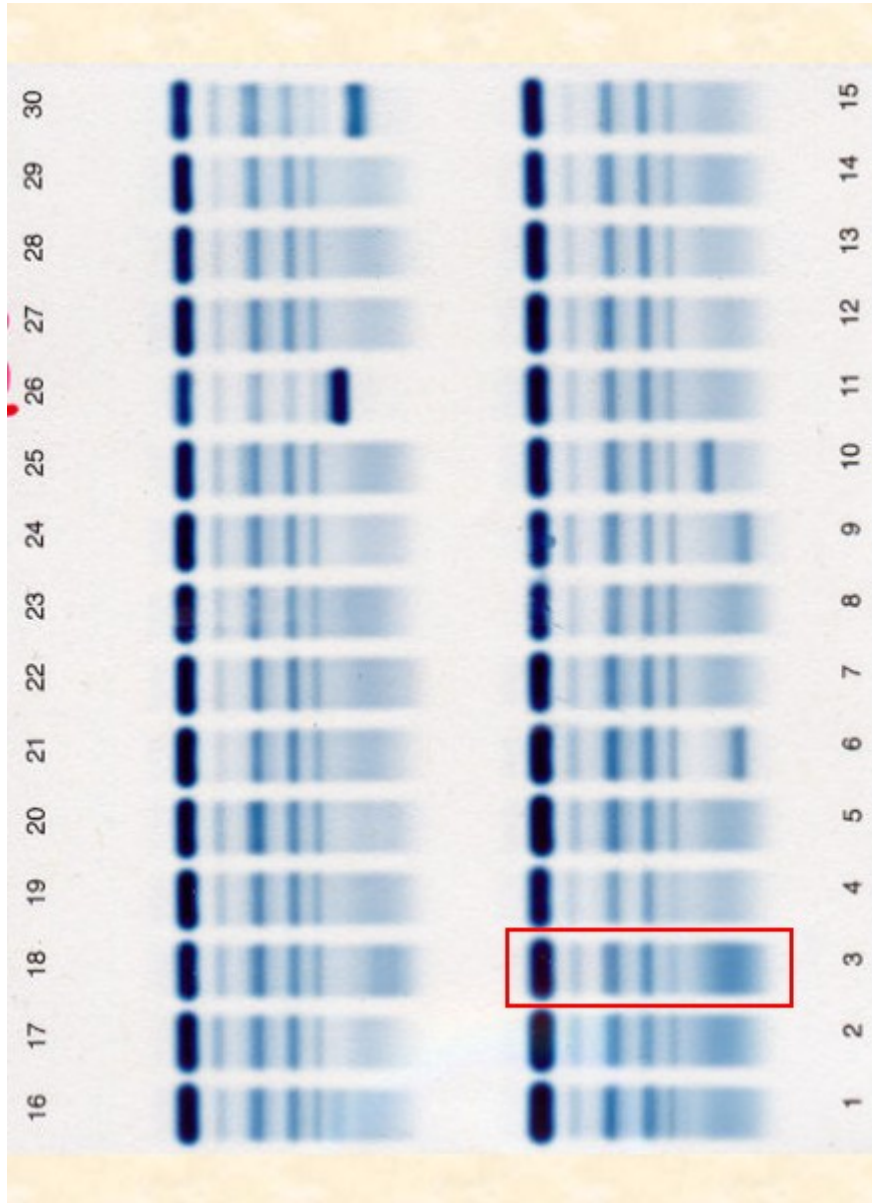


↑  $\alpha$ 1- and ↑  $\alpha$ 2-globulins

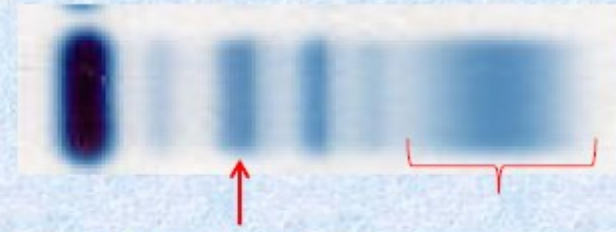
Often with decreased albumin, as shown in #12

- infection
- injury
- surgical trauma





## Chronic inflammation



↑ ↑  $\gamma$ -globulins  
 ↑  $\alpha_2$ -globulins  
 ± ↑  $\alpha_1$ -globulins  
 ± ↓ albumin

- autoimmune disease
- chronic liver disease
- chronic infection
- malignancy





# VELAMMAL MEDICAL COLLEGE HOSPITAL AND RESEARCH INSTITUTE

Anuppanadi, Madurai – 625009

## Department of Biochemistry

Certificate of Participation

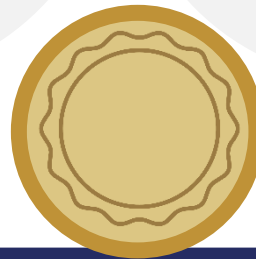
NAME

Has successfully completed the course on  
Serum Protein Electrophoresis on 31.05.2018

PROGRAM COORDINATOR

VICE PRINCIPAL & HOD Biochemistry

DR.P.K.Mohanty



DEAN

DR. R.M. Raja Muthaiah





**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009

**Report on Certificate Course on Serum Protein Electrophoresis**

**Topic:** Serum Protein Electrophoresis

**Date:** 31.05.2018

**Venue:** Biochemistry Demonstration Room

**Target Audience:** Faculties and M.B.B.S., students

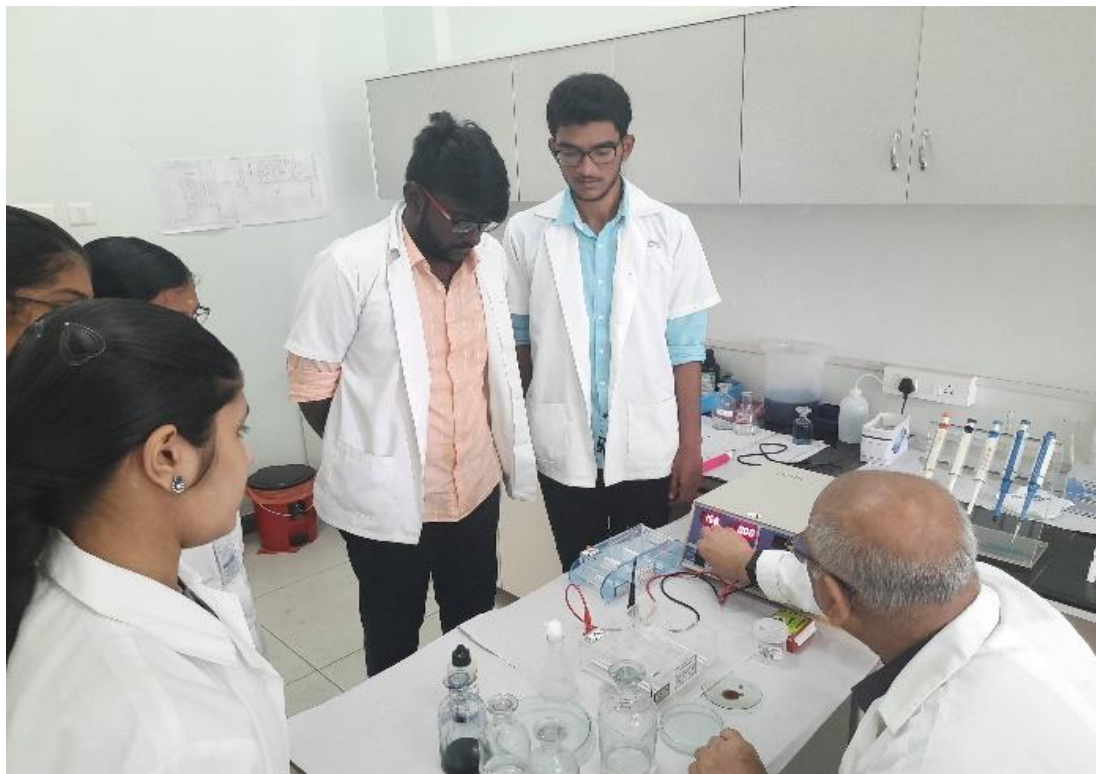
**Number of Participants:** 135

**Event Report:** The event started with the welcome address by Dr.K.Suganthy. Following that Dr. P.K. Mohanty started the lecture on serum protein electrophoresis. He explained the basics of electrophoresis and steps in performing serum protein electrophoresis and followed by this he showed a live demonstration of steps in serum protein electrophoresis and its interpretation.

  
Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
Dean  
Velammal Medical College Hospital  
and Research Institute  
"Velammal Village"  
Madurai-Tuticorin Ring Road  
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MADURAI - 625009





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MADURAI - 625009



**Outcome:** Participants should be able to do the serum protein electrophoresis by themselves and interpret the serum protein electrophoresis.

# Fractures and its management

**PROFESSOR DR.RAVIRAMAN**

# Introduction

- An *open fracture* is defined as a breach in soft tissue envelope over or near the fracture such that the underlying bone communicates directly with the environment
- The term *compound fracture* is non-specific and hence not used
- When wound occurs in same limb segment as a fracture , the # must be considered open until proven otherwise.

# HISTORY

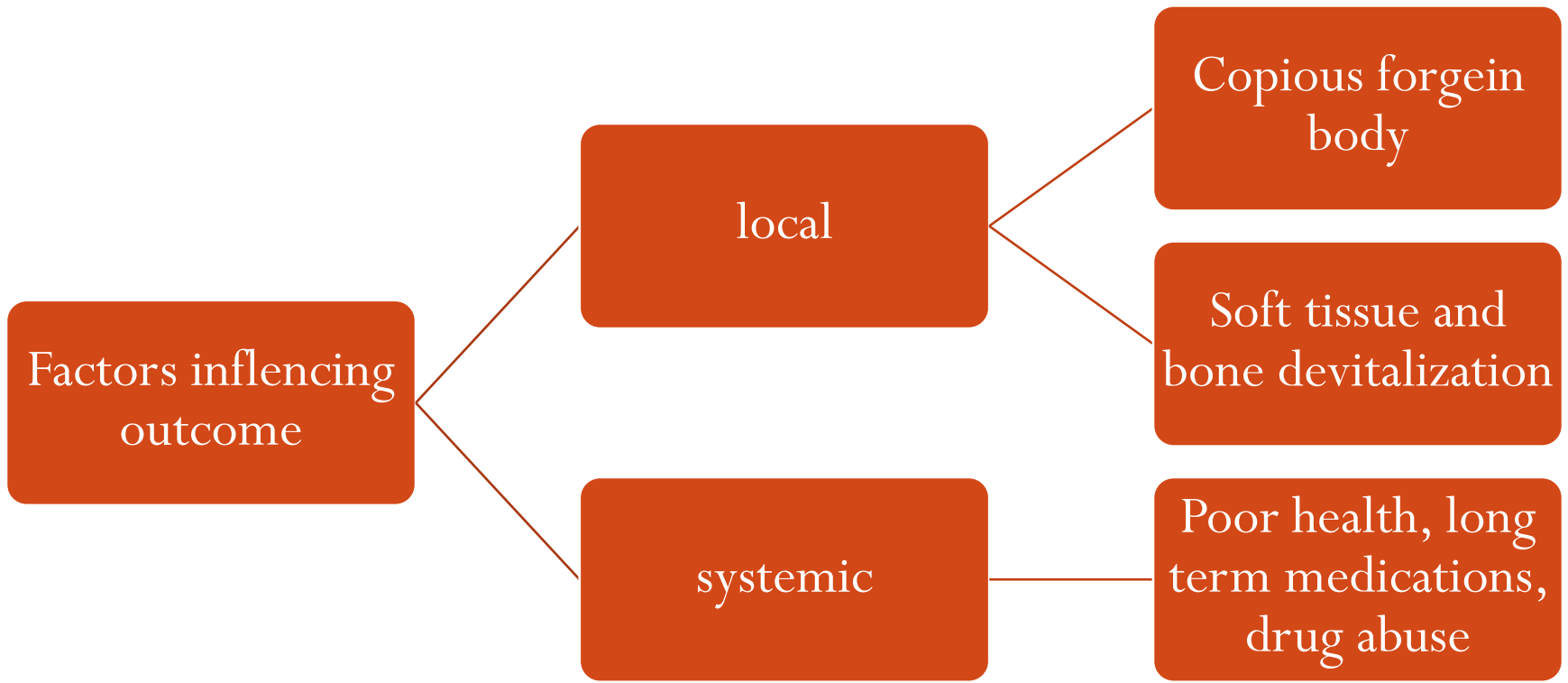
- Hippocrates-- greatest contribution-- recognition that surgeons can only facilitate healing they cannot impose it.
- Brunschwig and Botello, removal of nonvital tissue from wounds .
- Desault, 18th century, to reestablish the making of a deep incision to explore a wound, remove dead tissue, and provide drainage. coined term debridement.

# Principles of management

- Application of sterile dressing(anticipating debridement in operation set up)
- Immobilization
- Early antibiotics and tetanus
- Urgent operative wound debridement, irrigation,skeletal stabilization either provisionally or definitively
- Repeated debridement, definitive skeletal stabilization,soft tissue coverage as soon as possible

# Etiology

- Usually high energy trauma, sometimes low energy trauma depending upon anatomical site
- Greater soft tissue damage- susceptible to infection
- Bacteria from environment enter into wound but little correlation between contamination during injury and culture during surgery



Factors influencing  
outcome

local

Copious foreign  
body

Soft tissue and  
bone devitalization

systemic

Poor health, long  
term medications,  
drug abuse

# Management

- In emergency department
  - ATLS protocol



# LIFE SAVING MEASURES

- A Airway and cervical spine immobilisation
- B Breathing
- C Circulation (treatment and diagnosis of cause)
- D Disability (head injury)
- E Exposure (musculo-skeletal injury)

# History

- Time, nature, pattern of injury... to determine severity and also for documentation
- Any history suggestive and systemic involvement other than skeletal system
- Personnel history of the patient
- patient's medical condition
- Any h/o pain at fracture site prior to injury suggesting pathological fracture

- Any history of chronic medications, eg anticoagulants leading to continuous bleeding
- h/o intake of phenytoin for epilepsy

# Physical examination

- Compartment syndrome should be overlooked since compartment syndrome can also occur in open fracture
- Any wound in the In the fracture site or even with in the same limb segment should be assumed to represent an open fracture until other wise prooven
- Description of the wound— location, length, configuration, orientation, skin condition and abrasions

- Prolonged period of joint displacement or fracture displacement leads to pressure on the underlying skin converting closed fracture into open fracture
- Open fracture in pelvis may lead to organic contamination... carefully watched may necessitate colostomy or other surgical procedure to prevent contamination

- Neurovascular examination at and distal to the fracture site

- VASCULAR

- Warmth
- Pulse
- Colour
- Capillary filling
- Venous filling
- Arterial blood gas analysis

- Neurological

- Motor
- Sensory
- Autonomous

- Moist saline dressing,
- splint(non circumferntial, stabilizing atleast one joint above and below)
- evaluation of wound,
- photograph ,
- tetanus,
- antibiotics

# Radiological examination

- Plain radiograph
  - Initially atleast anteroposterior and lateral view of fractured bone
  - At least one joint above and below to look for any joint subluxation or dislocation
  - To identify fracture
  - To classify fracture
  - For furthur surgical plan
- Air in subcutaneous tissue, muscle(gas gangrene), E.coli and cl.perfringens
- CT
- MRI(not commonly used)

# Open fracture equivalents

- Open joints secondary to traumatic arthrotomies
- Diagnosed by saline challenge test

# Impending open fracture

- Laterally displaced fracture dislocation of ankle
- Displaced calcaneal tuberosity fracture
- Talar neck fracture (type III and IV)
- High energy tibial plateau fracture

# METHODS OF CLASSIFICATION

- GRADING SYSTEM – focus on severity of limb injury only  
Eg: Gustilo Anderson , Tscherne and Gotzen, Byrd and Spicer etc
- SCORING SYSTEM – focuses on limb injury and general health; also give ‘amputation score’.  
Eg: MESS , NISSA , LSI, PSI etc
- COMPREHENSIVE SYSTEM – combines the above two systems  
Eg: AO system , Ganga hospital score

# ***GRADING SYSTEMS***

# GUSTILO ANDERSON SYSTEM

- In 1976 , Gustilo and Anderson treated 1025 open fractures based on his grading system that offered prognosis about outcome of infected fractures
- In 1984, it was modified and was based on
  - a. Size of wound
  - b. Periosteal soft tissue damage
  - c. Periosteal stripping
  - d. Vascular injury
- Recently it has emerged that injured limbs are appropriately categorised by the system after wound excision (*Yang EC, Eisler J.2003*)

TYPE	DESCRIPTION
I	<1cm clean wound
II	>1cm laceration with no extensive soft tissue damage, skin flap or avulsion
IIIa	Extensive soft tissue damage or flaps , but adequate coverage of bone or high energy trauma regardless of size of the wound (like comminuted or segmental fractures even those of <1cm)
IIIb	Extensive soft tissue loss with periosteal stripping and bone exposure (usually massively contaminated)
IIIc	With arterial injury that requires repair regardless of size of soft tissue wound

- Simple and hence it is widely used
- Poor interobserver reliability especially with inexperienced surgeons
- gun shot injuries , wounds in farm yards are always grade III











# Classification— Why do we care?

- Classification correlates with prognosis

<b>GRADE</b>	<b>I</b>	<b>II</b>	<b>IIIA</b>	<b>IIIB</b>	<b>IIIC</b>
<b>INFECTION</b>	<b>0-2%</b>	<b>2-7%</b>	<b>10-25%</b>	<b>10-50%</b>	<b>25-50%</b>
<b>FRACTURE HEALING</b>	<b>21-28 WKS</b>	<b>26-28 WKS</b>	<b>30-35 WKS</b>	<b>30-35 WKS</b>	

# TSCHERNE SYSTEM

GRADE	DESCRIPTION
1	Skin laceration mostly inside out injury with little or no contusion of skin
2	Skin laceration with circumscribed skin or soft tissue contusion with moderate contamination
3	Fractures with severe soft tissue injury often with NV injury , severe bone comminution or compartment syndrome
4	Sub total (remaining soft tissue not exceeding $\frac{1}{4}$ of limb circumference) or total amputation

➤ This system includes compartment syndrome which is not included in other grading systems

# BYRD AND SPICER

TYPE	DESCRIPTION
I	Both endosteal and periosteal supply intact and surrounding soft tissue is healthy
II	Endosteal supply interrupted but periosteal supply maintained by surrounding soft tissues
III	Devascularised bone fragment and requires flap coverage

- This system lacks sophistication and hence not widely used

# ***SCORING SYSTEMS***

# MANGLED EXTREMITY SEVERITY SCORE (MESS)

TYPE	CHARACTERISTICS	INJURIES	POINTS
SKELETAL/ SOFT-TISSUE GROUP			
1	Low energy	Simple closed #, small calibre gun shot	1
2	Medium energy	Open # , D/L , multiple level #	2
3	High energy	Shot gun blast , high velocity gun shot	3
4	Massive crush	Rail road, oil rig accidents	4
SHOCK GROUP			
1	Normotensive	BP stable in field and OT	0
2	Transiently hypotensive	BP unstable in field but responds to IV fluids SBP<90 in field and responding to IV fluids only	1
3	Prolonged hypotensive	in OT	2

# MESS Contd...

TYPE	CHARACTERISTICS	INJURIES	POINTS
<b>ISCHEMIA GROUP</b>			
1	None	Pulsatile limb w/o signs of ischemia	0*
2	Mild	Diminished pulses w/o signs of ischemia	1*
3	Moderate	No pulse, sluggish capillary refill, paraesthesia, motor activity	2*
4	Advanced	Pulseless, cool, paralysed, numb, no capillary refill	3*
<b>AGE GROUP</b>			
1	< 30 yrs		0
2	30 – 50 yrs		1
3	> 50 yrs		2

\* If ischemia time > 6 hrs, add 2 points.

## MESS Contd...

- It was developed to identify those patients who will be benefited by primary amputation
- In retrospective analysis, the outcome of injured limb was either salvage or amputation
- A score of 7 or greater is predictive of amputation
- MESS is found to be specific but lacks some sensitivity which infers that score predicting limb salvage ( $<7$ ) is more reliable than score predicting amputation ( $>$  or  $=7$ ) (*Bosse MJ JBJS 83A:412,2001*)

# OTHER SCORING SYSTEMS

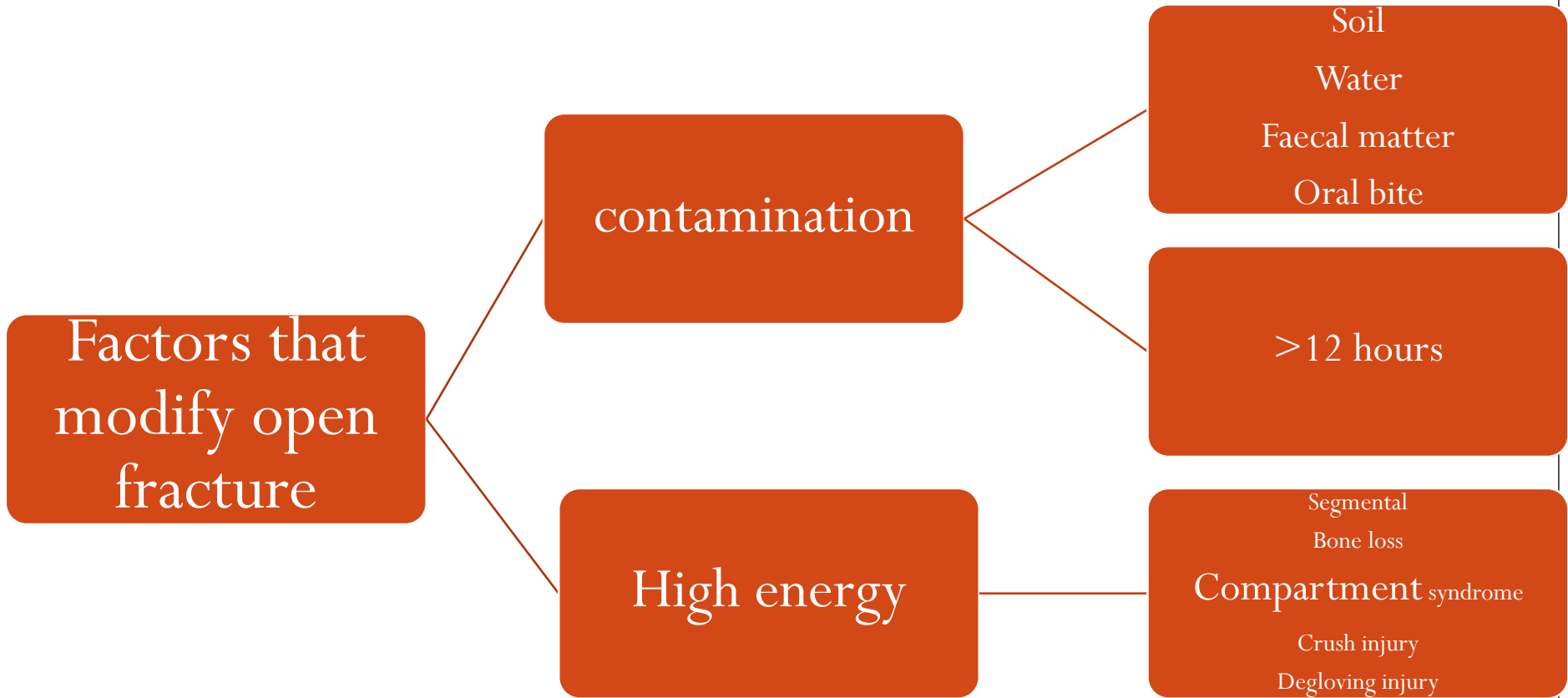
- NISSA – Nerve injury Ischemia Soft tissue injury Skeletal injury Shock Age , is more sensitive and more specific than MESS.
- LSI – Limb Salvage Index
  - a. This index is applied to limbs with arterial injury
  - b. Warm ischemia time together with scores for injured skin , muscle , bone , NV are added to give total score
  - c.  $LSI > \text{ or } = 6$  and grade IIIc gustilo with major nerve injury are amputated

***COMPREHENSIVE  
SYSTEMS***

- AO System :
  - Skin lesions , muscle -tendon , NV , bone injuries are graded separately
  - AO system allows better prediction of outcome when compared to Gustilo
  - Due to its complexity not widely accepted
- Ganga hospital score :
  - Includes additional criteria like age  $>65$  , DM , cardio-respiratory disease , trauma chest/abdomen, farmyard/sewage contaminations, delay in debridement  $>12h$

# Injury severity score

- Abbreviated Injury Scale (AIS) global severity scoring system that classifies each injury in every body region according to its relative severity on a six point ordinal scale:
  - 1 Minor,
  - 2 Moderate,
  - 3 Serious,
  - 4 Severe,
  - 5 Critical,
  - 6 Maximal (currently untreatable).
- nine body regions: 1 Head, 2 Face, 3 Neck, 4 Thorax, 5 Abdomen, 6 Spine, 7 Upper Extremity, 8 Lower Extremity, 9 External and other.
- A major trauma (or polytrauma) is defined as ISS > 15.



# Timing of treatment

- Debridement within 6 hours of injury
- Threshold for common organism to multiply to  $10^5$  is around 5.17 hours . So debridement should be started earlier

# TIMING OF PROCEDURES

- SUBSEQUENT DAYS
  - 48 hrs = Debride Again, Plan Closure
  - 4-5 days = Cover Wound
  - Convert to Definitive Fixation

# antibiotics

- Preoperative prophylactic antibiotics
- Immediately upon arriving in emergency department, continued for 24 hours, and for every 24 hours after subsequent debridement
- No supporting evidence for continuing antibiotics further
- Not used to prevent pin tract infection

Antibiotics	type I	type II	type III	organic
Cefazolin 1 gm/8 hrs	x	x	x	
Aminoglycoside 5 mg/kg/day	x	x	x	
Penicillin and metronidazole				x

# Wound cultures

- Initial culture --- not of much value
  - 76 %--- no growth
  - 24 %---only skin flora
- Cultures after debridement and during subsequent debridement is useful

# Operative management

- Gross contamination, foreign body removed immediately
- Wound covered with saline gauze dressing and not with iodine soaked gauze due to
  - Blood and fat inactivate the antibiotic activity of iodine
  - Systemic toxicity in case of large wounds
  - Staining of tissues leads to difficulty while debridement
  - Affects wound healing at cellular level

- Once patients general condition is stable, patient shifted to operative room and anaesthesia given
- If compartment syndrome is suspected regional block is given rather than general anaesthesia because to examine the part post operatively

# Operative set up

- Separate table for debridement instruments and separate table for skeletal stabilization instruments in order to prevent contamination
- Tourniquets are placed in position but not inflated because the tissues are already hypoxic

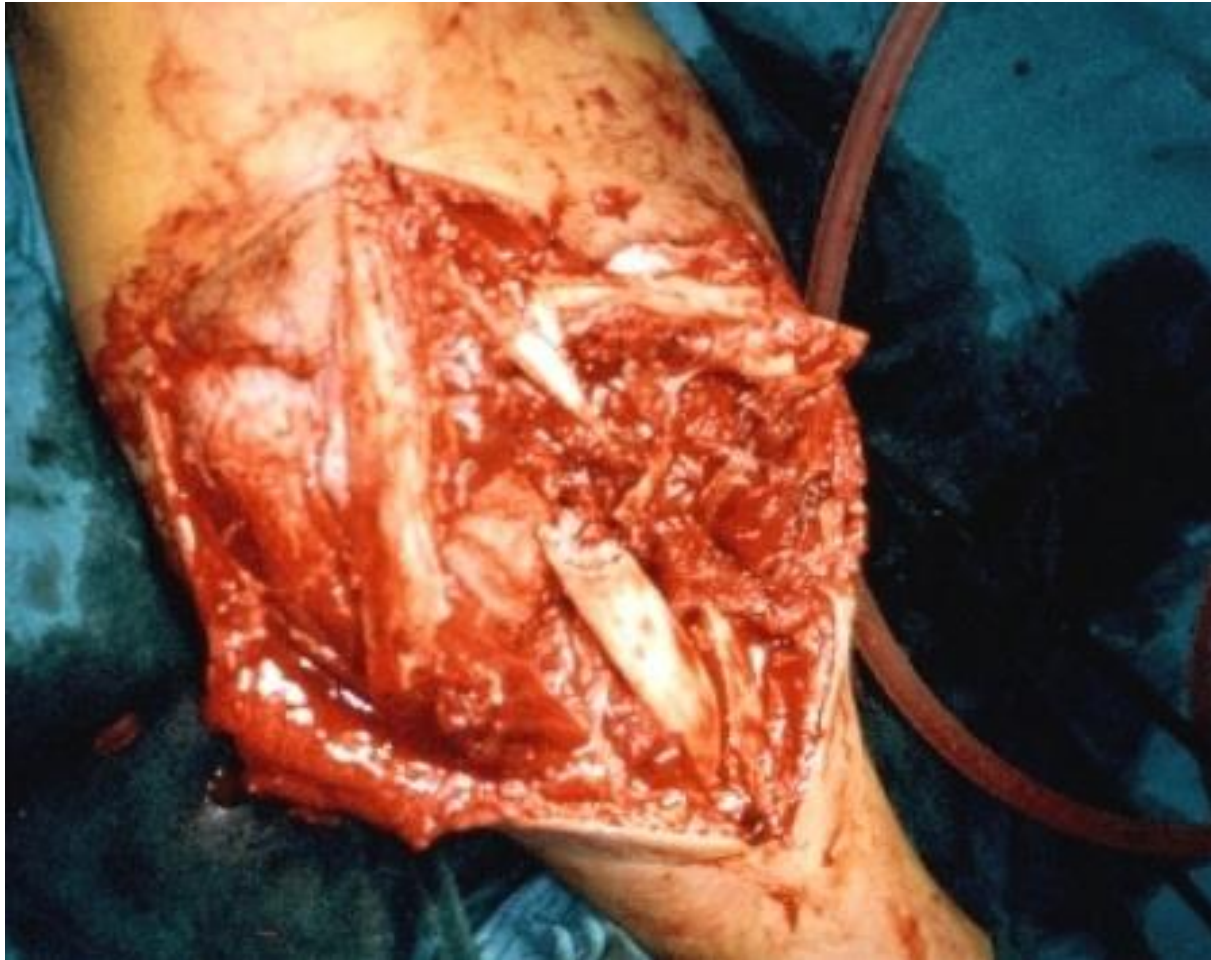
# Debridement and irrigation

- Debridement -----
  - Superficial
  - deep

- Superficial

- Wound extended
- Final classification of fracture made
- Outside to inside
- Longitudinal incision
- Necrotic skin and subcutaneous tissue excised
- Not to detach skin from subcutaneous tissue
- Marginal skin left for further debridement
- Both non viable and marginally viable fascia excised
- Prophylactic fasciotomy (if required)

# Extensile longitudinal skin incision



- Deep

- Soft tissue..dynamic and evolving(not static)
- Hydraulic damage to muscle due to high water content(so damage even if skin not involved)
- When in doubtful of viability ,debridement is safest



- Viability of muscles
  - 4 “c” s
    - Colour-- beefy red;
    - Contractility--
    - Consistency--- firm, not easily disrupted
    - Capacity to bleed
- Vascular anatomy of muscles
  - Small arteries that lie in epimyseal layers

- Histologic zones
  - Inner zone ... necrotic and no swelling
  - Partial viable muscle with ischemic zone that swells subsequently
  - Outer normal muscle where no swelling
- So superficial muscle may be viable than deep muscles which are necrotic
- Debridement of muscles must be overlooked. Done by spreading muscle fibre along its line with hemostat

- Tendons

- Preserved
- Covering of tendon with peritendon is essential

- Intact arteries ,veins, nerves should not be debrided

- Bones

- Small pieces of bone completely devoid of soft tissue debrided
- Large fragment retained for skeletal stabilization and removed subsequently
- Articular fragment.. Retained
- Bone ends cleared off from haematoma with currette and saline



# Irrigation

- Done after debridement
- Decreases bacterial count, float out remaining debris
- High pressure pulsatile lavage
- Low pressure gravity lavage
- No advantage of using antibiotic solution
- Ideal volume ... 6 to 12 litres of normal saline

# PULSE LAVAGE



# Skeletal stabilization

- Benefits
  - Minimizing motion at fracture site so decreasing soft tissue injury
  - Immediate vascular inflow
  - Decrease pain
  - Mobilization of patients out of recumbence
- Restoration of bony anatomy by fracture reduction and stabilization...healing of soft tissue

- Articular stabilization leads to immediate joint motion .. Good long term outcome

# Types

- Extraosseous immobilization
  - Pop
  - Fibreglass
  - Skeletal traction
- For eg ... minimally displaced compound grade I wounds
- Humeral shaft fractures with minimal laceration

# External fixation

- Temporary or definitive
- Done by placing transcutaneous half pins remote from fracture site
- Frames applied
- Reduction done
- Clamps tightened
- Allow early limb and patient mobilization



- While doing external fixation definitive procedure should be considered
- Pin tract infection ... post operative infection
- Pins should not be in joints
- If pins placed through soft tissue → soft tissue instability → infection → pin loosening
- Predrilling rather than self drilling to avoid thermal necrosis

- **Complication**

- Pin loosening
- Reduction loss
- Osteomyelitis
- Sepsis

- Pin tract infection → contraindication for intramedullary nailing

- External fixation as definitive treatment of open fracture leads to high rate of infection(15%)
  - Non union(3 – 10 %)
  - Malunion(36%)
  - Pin tract infection (50%)

# Internal fixation

- Primary focus is avoiding infection
- For many years— internal fixation discouraged—
  - Metal acts as foreign body
  - Biofilm formation
  - Bone devascularization by extramedullary implants

# Plates and screws

- Important is minimizing complication of acute plate fixation
  - Pre operative antibiotics
  - Sterile dressing'
  - Splinting of fracture
  - Meticulous debridement
  - Minimal periosteal stripping
  - Screws usually unicortical to avoid reamer interface

# Internal fixation – plates and screws



# Intramedullary nailing

- Most common amenable is tibial shaft fracture
- Previous history
  - Intramedullary nailing and reaming → damage to osseous blood supply → contamination of medullary cavity
- Chapmans trial -- → open fracture fixed with
  - Plate --- 19% infection
  - External fixation ---- 3 % infection
- Infection decreased with imil nailing

# IM Nail



- External fixation leads to
  - secondary procedures
  - Malalignment
  - Non union
- Currently acute imil nailing with limited reaming preferred except those with deep extensive contamination, vascular damage, pre xting deformity

# Reaming vs unreaming

- During fracture both endosteal and periosteal blood supply disturbed
- While reaming endosteal blood supply further disturbed but has secondary rebound phenomenon on periosteal perfusion

- Benefits of reaming

- Enhances periosteal blood supply
- Large diameter nails
- Reaming of isthmus.. Large segment of bone nail contact

- In femur ,safer to have imil nailing due to increased musculature and increased blood supply

# Reamed vs. Nonreamed Nails

	<u>Reamed</u>	<u>Non-Reamed</u>
# pts.	73	63
Nonunion	4%	11%
Malunion	4%	3%
Broken Bolts	3%	16%

# Wound closure and coverage

- Wound without skin loss
- Wound with skin loss

- Without skin loss

- Previously delayed closure but lead to complications
- Currently – primary wound closure with tension free closure
- Delayed closure

- Indications

- >12 hours
- >12 hours with out antibiotics
- Deep seated contamination
- Neurovascular injury
- Immunocompramised
- Inability to achieve tension free closure

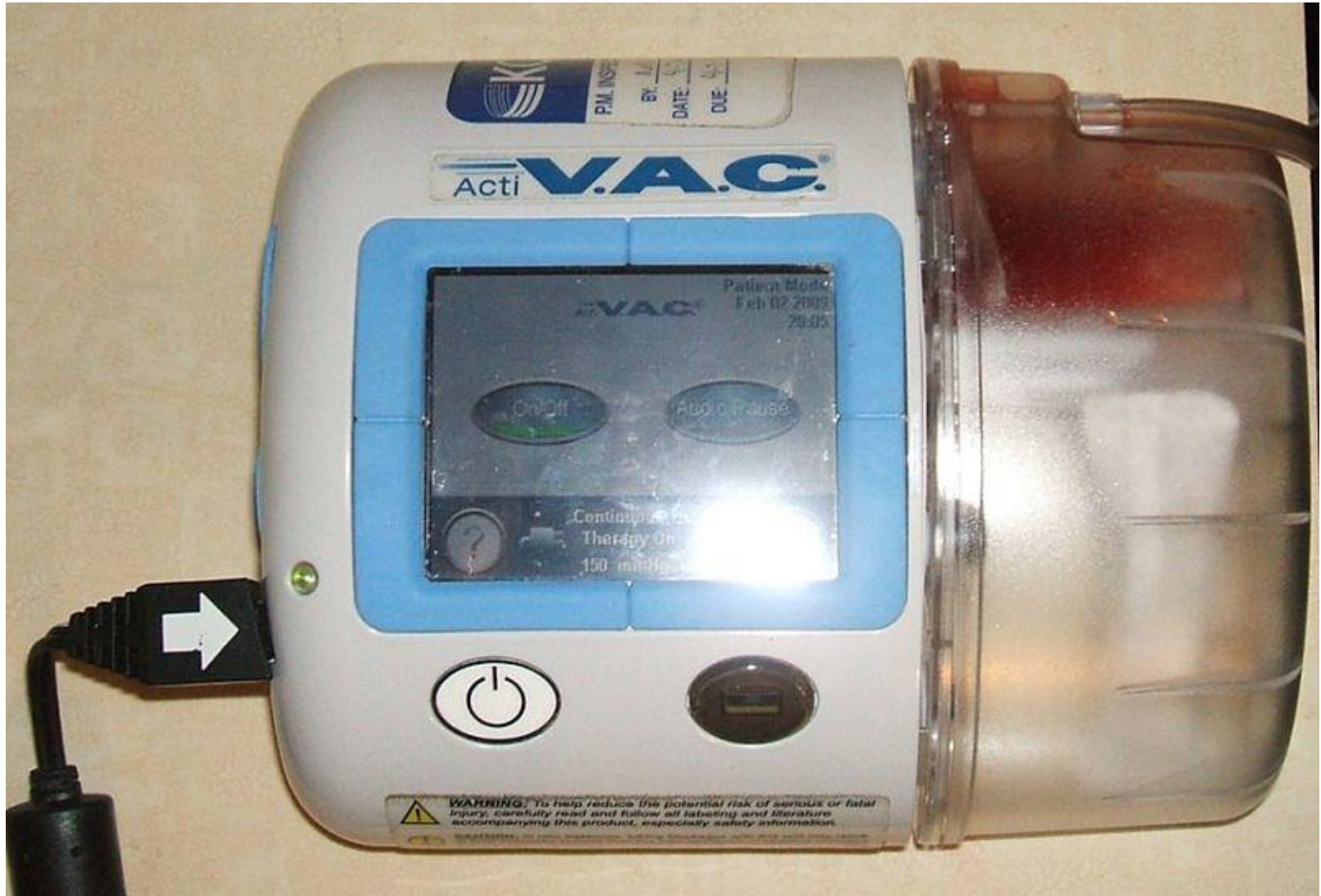
- With skin loss

- Healing by secondary intention but no bone, tendon, neurovascular structure should be in its bed
- Fascial layers closed.. Wed to dry gauze dressing---until granulation—epithelialization
- Negative pressure foam dressing-----decreases edema---increases granulation but requires patients compliance and good nursing care
- Releasing or relaxing incision

- Antibiotic eluting PMMA / calcium based cements----high local concentration
- Beads placed deeper in wound and skin closed
- Or beed pouch used
- Elution higher in initial days and gradually decreases
- Cement substrate acts for dead space management
- Calcium sulfate/hydroxyapatite used in chronic condition where secondary removal not done



- **Negative pressure wound therapy (NPWT)**, also known as **topical negative pressure, sub-atmospheric pressure dressings** or **vacuum sealing technique**,
- A vacuum source is used to create sub-atmospheric pressure in the local wound environment.





# Gunshot Wounds

- Low energy missiles rarely require debridement and can often be treated like closed injuries
- Fractures due to high energy missiles (eg assault rifle or close range shot gun) treated as standard open injuries



# Complications

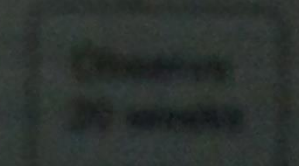
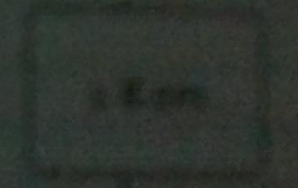
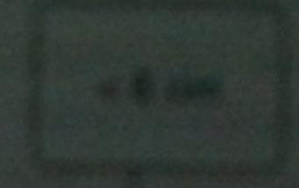
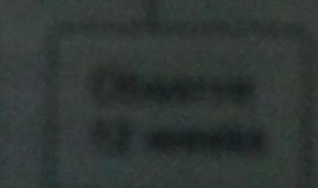
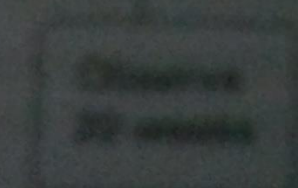
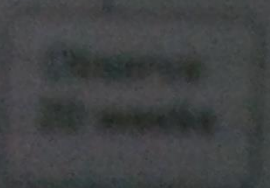
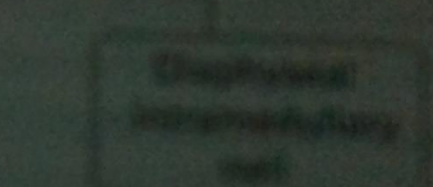
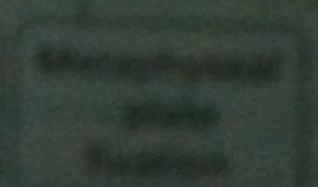
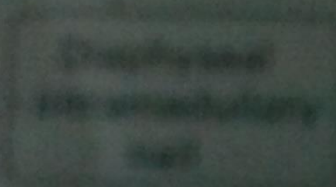
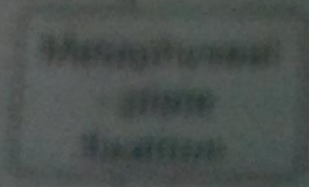
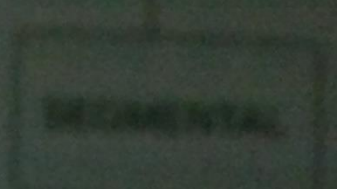
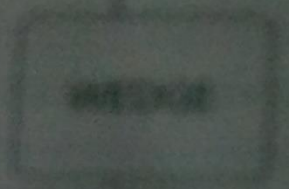
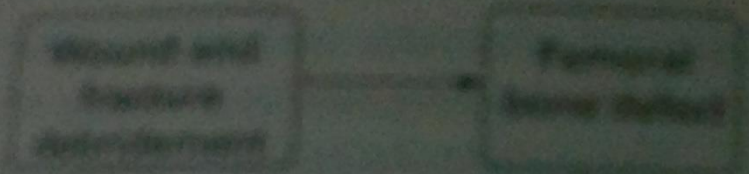
- Infection
- Systemic inflammatory response syndrome
- Acute respiratory distress syndrome
- Systemic sepsis
- Chronic osteomyelitis
- Non union
- Malunion
- Soft tissue damage, neural, vascular damage
- 



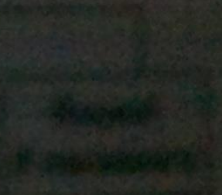
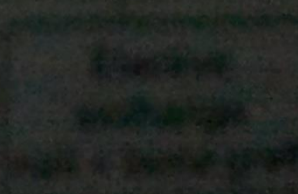
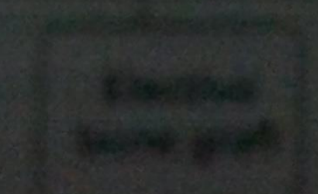
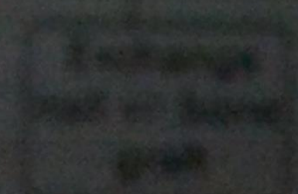
# Recent journals

- Jbjs dec 2010
- Study of treatment of open femoral fractures with bone loss
- Created an algorithm





Management of ear radiological evidence of hearing



**From**

Dr. V. Raviraman  
Department of Orthopaedics  
Velammal Medical College Hospital and RI  
Madurai

**To:**

The Dean  
Velammal Medical College Hospital and RI  
Madurai

**Respected Sir:**

We from the department of Orthopedics are planning to conduct a Certificate Courses – **“LIMB SALVAGE IN ORTHOPAEDICS”** on 04/01/2018 involving Ortho posting M.B.B.S Students & CRRIs. We kindly request you to give permission to conduct the same. Kindly do the needful.

Thanking You


Date: 22.12.2017

Place: Madurai

Yours sincerely,

  
Dr. V. Raviraman

Head of the Department

  
Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
Dean  
Velammal Medical College Hospital  
and Research Institute  
"Velammal Village"  
Madurai-Tuticorin Bypass Road  
Anuppanadi, Madurai, T.N.-625 009



**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009

**Certificate Course**

**Topic:** Limb Salvage In Orthopaedics

**Date :** 04.01.2018,

**Time :** 11.00 am to 01.00 pm

**Venue :** Ortho Opd Demo Hall

**Participant's List**

Sl.No	Faculty Name
1.	Dr. V. Raviraman
2.	Dr. S. Shanmuganathan
3.	Dr. Ganesan G Ram
4.	Dr. K. N. Subramanian
5.	Dr. M. Subbiah
6.	Dr. R. Hari sudhan
7.	Dr. Muthu kumar . S
8.	Dr. S. Lokesh Kumar
9.	Dr. E. Vijaya raja
10.	Dr. M.J. Krishna kumar
11.	Dr. S. Dheepan Kumar
12.	Dr. V Janarthanan
	<b>CRRIS</b>
13.	Brindha Devi M
14.	Deepasri J C
15.	Dhanalakshmi P
16.	Dharani K
17.	Gautham Kumar S
18.	Gayathri M
19.	Gokularasu M
20.	Gowshika T
21.	Hareeshma S G
22.	Hariganesh K
23.	Hariharan P
24.	Hariharasudhan S
25.	Hariharasudhan T


*T. Thirunavukkarasu*  
Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
Dean

Velammal Medical College Hospital  
and Research Institute  
"Velammal Village"  
Madurai-Tuticorin Ring Road  
Anuppanadi, Madurai-625 009, T.M.



**VELAMMAL MEDICAL COLLEGE**  
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MADURAI - 625009

26.	Haripreetha R
27.	Harishma Devi G
28.	Harishma I
29.	Haritha K
30.	Heera J
31.	Hiresan A M
32.	Ishwarya Lakshmi G
33.	Iswarya S
34.	Jayakumar M
35.	Jeash Narayan K S
36.	Jebisha J B
37.	Jeydhip Sankar K S R

  
Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
Dean  
Velammal Medical College Hospital  
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**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009

**Report**

**Topic** : Certificate course on Limb Salvage In Orthopaedics

**Date** : 04.01.2018

**Venue** : Ortho OPD Demo Hall, VMCH & RI

**Target Audience** : CRRIs

**Number of participants:** 25

A certificate course on Limb Salvage In Orthopaedics was organised by the Department of Orthopaedics to CRRIs on 04.01.2018. 25 CRRIs participated in the course. The program began by 08.30 am with a pretest followed by Introduction, Patient selection, Surgical principle, prosthesis fixation, Complications and Assessment. The program concluded with a vote of thanks. Pretest and posttest were conducted to sensitize the students with topic content and grade their knowledge gain of the course.

**Outcome:**

Students learnt about the concepts of Limb Salvage In Orthopaedics. The gained idea about Limb Salvage In Orthopaedics.

Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,  
*Dean*

Velammal Medical College Hospital  
and Research Institute  
"Velammal Village"  
Madurai-Tuticorin Ring Road  
Anuppanadi, Madurai-625 009, T.N.



**VELAMMAL MEDICAL COLLEGE**  
HOSPITAL AND RESEARCH INSTITUTE  
MADURAI - 625009

## Department of Orthopaedics



**VELAMMAL  
HOSPITAL**  
the healing touch



**Certificate Course**  
Title:  
**Limb Salvage in  
Orthopaedics**

### AGENDA

TIME	TOPIC	SPEAKER
8.30 am to 09.00 am	Introduction	Dr. S Lokesh kumar
9.00 am to 11.00 am	Patient selection	Dr.K.N.Subramanian
11.00 am to 01.00 pm	Surgical principle	Dr.N.Jerry
01.00 pm to 01.30 pm	Prosthesis fixation	Dr.A.Sivakumar
01.30 pm to 02.30 pm	Lunch	
02.30 pm to 03.30 pm	Complications	Dr.S.Manoh
03.30 pm to 04.30 am	Assessment	Dr.S.Muthukumar



**Date: 04.01.2018**



**Day: Tuesday**



**Venue: Lecture Hall**



**Duration: 8 hours**

Teaching method power point presentation,  
interactive discussion



# VELAMMAL MEDICAL COLLEGE HOSPITAL AND RESEARCH INSTITUTE MADURAI - 625009



GPS Map Camera



**Chinthamani, Tamil Nadu, India**

IDA Scudder Trader Centre, Velammal Medical College  
Campus, Ring Road, Chinthamani, Tamil Nadu 625009,  
India

Lat 9.887117°

Long 78.150652°

04/01/18 12:44 PM GMT +05:30



GPS Map Camera



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Campus, Ring Road, Chinthamani, Tamil Nadu 625009,  
India

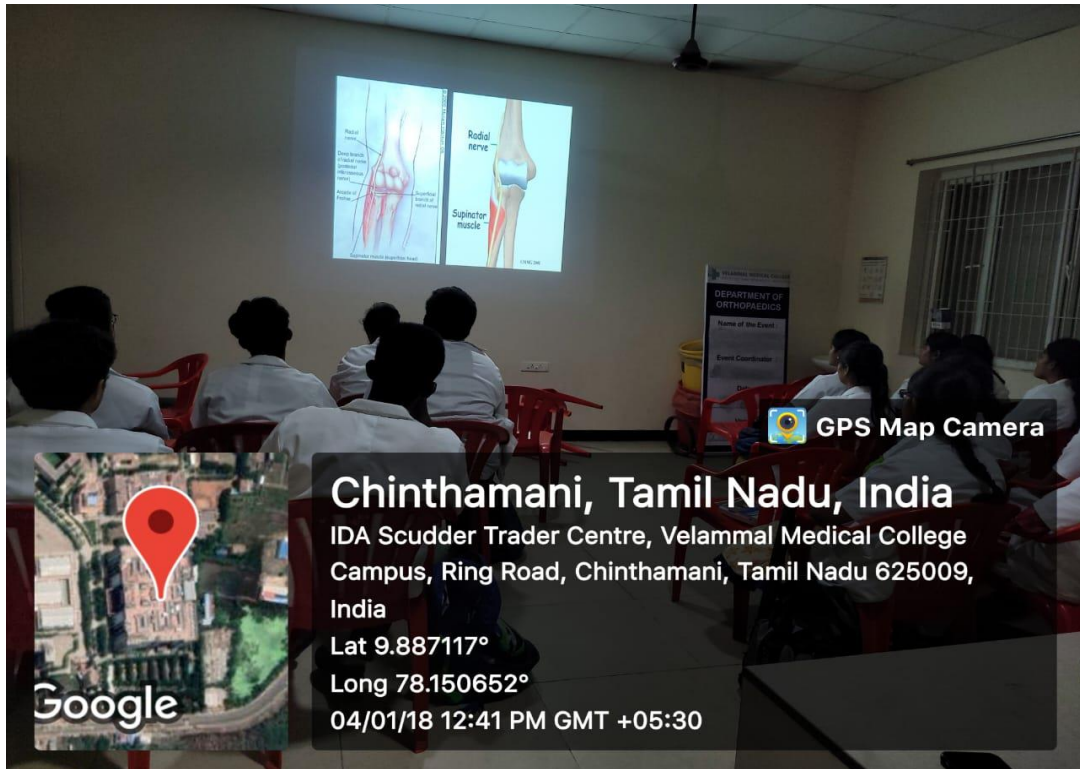
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# VELAMMAL MEDICAL COLLEGE HOSPITAL AND RESEARCH INSTITUTE MADURAI - 625009



# LIMB SALVAGE IN ORTHOPAEDICS

DR. GANESH G RAM

# DEFINITION

- A set of surgical techniques that have been developed to restore the skeletal continuity following the en bloc resection of bone and soft tissue neoplasm.

# GOALS OF LIMB SLAVAGE

- Painless limb
- Functional , tumor free limb

# SCORING SYSTEMS TO DECIDE ON LIMB SALVAGE

- Mangled extremity syndrome index
- Predictive salvage index (PSI)
- Mangled extremity severity score
- Limb salvage index (LSI)

# LIMB SALVAGE OR AMPUTATION

- SIMON – 4 ISSUES
  - Survival (mortality)
  - Morbidity – short and long term
  - Function – compared to prosthesis
  - Psychosocial consequences

# LIMB SALVAGE – Complications

- More extensive surgical procedure
- Flap necrosis
- Blood loss
- DVT

# LONG TERM COMPLICATIONS

- Periprosthetic fracture
- Prosthetic loosening or dislocation
- Allograft fracture
- Non-union of graft-host junction
- Future leg length discrepancy



VELAMMAL MEDICAL COLLEGE  
HOSPITAL & RESEARCH INSTITUTE

**DEPARTMENT OF RADIODIAGNOSIS**

# **IMAGING ANATOMY OF BRAIN**

**CERTIFICATE COURSE**

VELAMMAL MEDICAL COLLEGE HOSPITAL & RESEARCH INSTITUTE



# IMAGING ANATOMY OF BRAIN

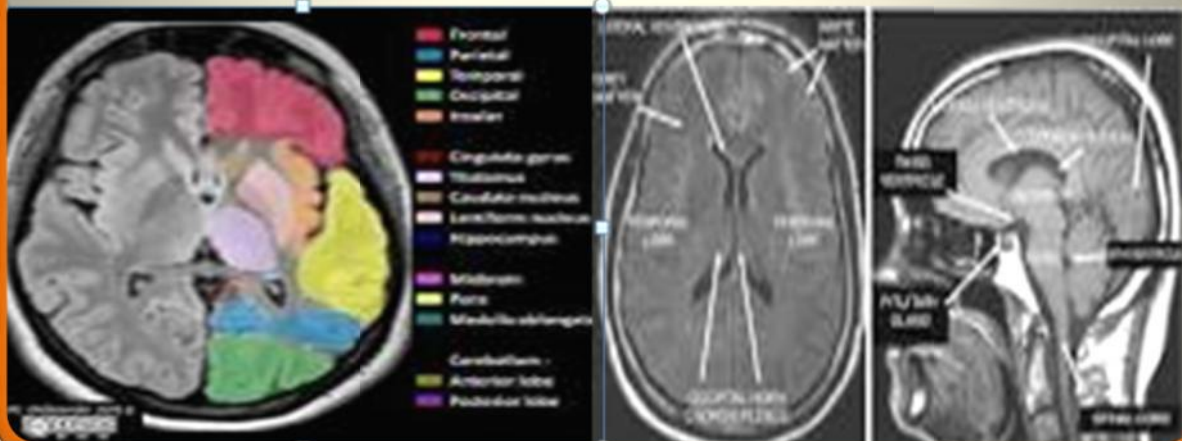
CERTIFICATE COURSE

Department of Radiodiagnosis

Date: 13.06.2018

Time: 10 am – 12 pm

MBBS Students (Final Year)







# ANATOMY OF BRAIN

---

## In short

- The Patient Is Placed In A Magnetic Field.
- A Radio Frequency Wave Is Sent In.
- The Radio Frequency Wave Is Turned Off.
- The Patient Emits A Signal.
- Which Is Received And Used For Reconstruction Of The Picture.

- MRI is based on the principle of nuclear magnetic resonance (NMR)
- Two basic principles of NMR
  1. Atoms with an odd number of protons or neutrons have spin
  2. A moving electric charge, either positive or negative, produces a magnetic field
- Body has many such atoms that can act as good MR nuclei ( $^1\text{H}$ ,  $^{13}\text{C}$ ,  $^{19}\text{F}$ ,  $^{23}\text{Na}$ )

## Why Hydrogen ions are used in MRI

Every hydrogen nucleus has an unpaired proton, which is positively charged.

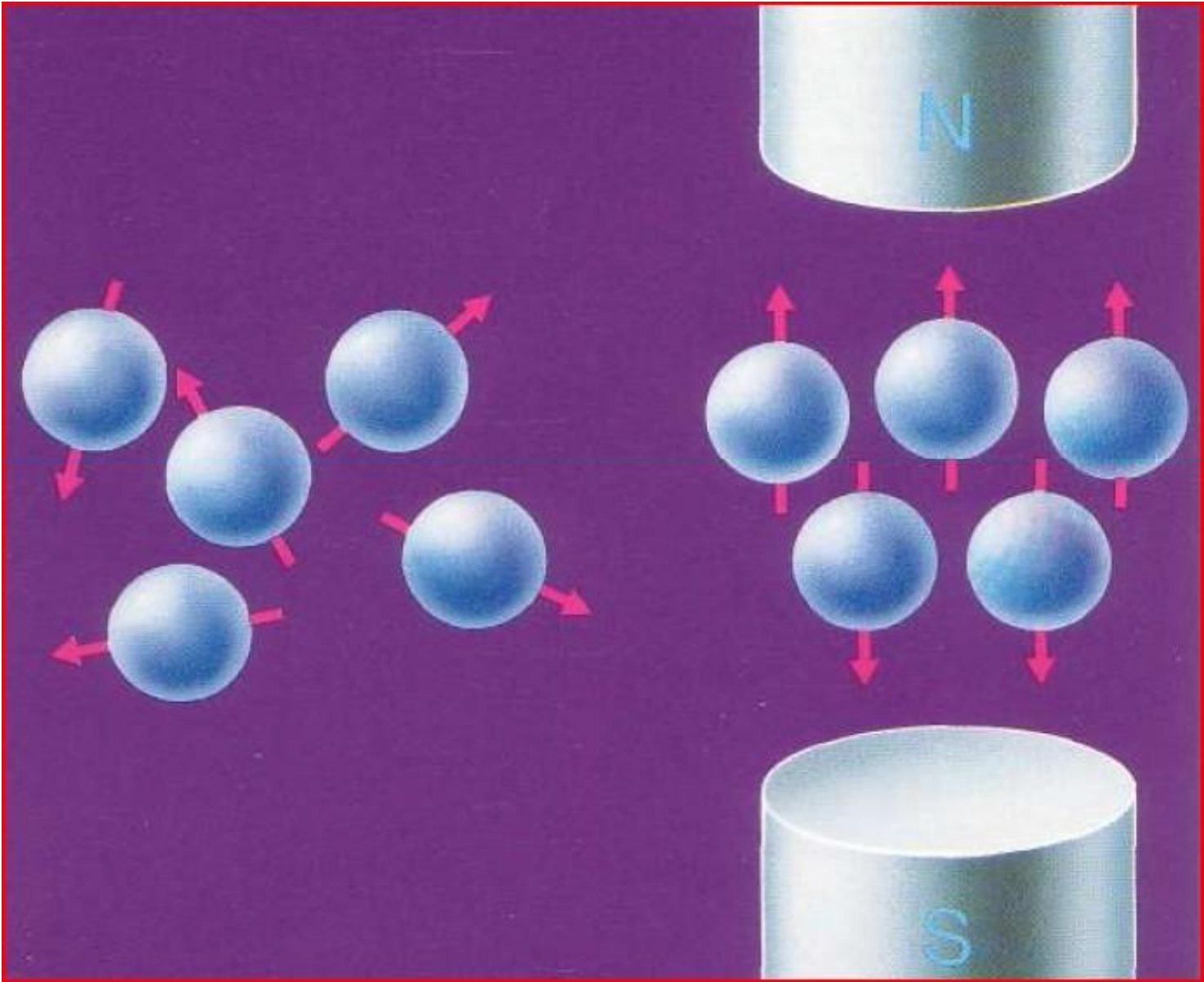
- a tiny magnet which produces small but noticeable magnetic field

Hydrogen is abundant in the body in the form of water and fat

Essentially all MRI is hydrogen (proton) imaging

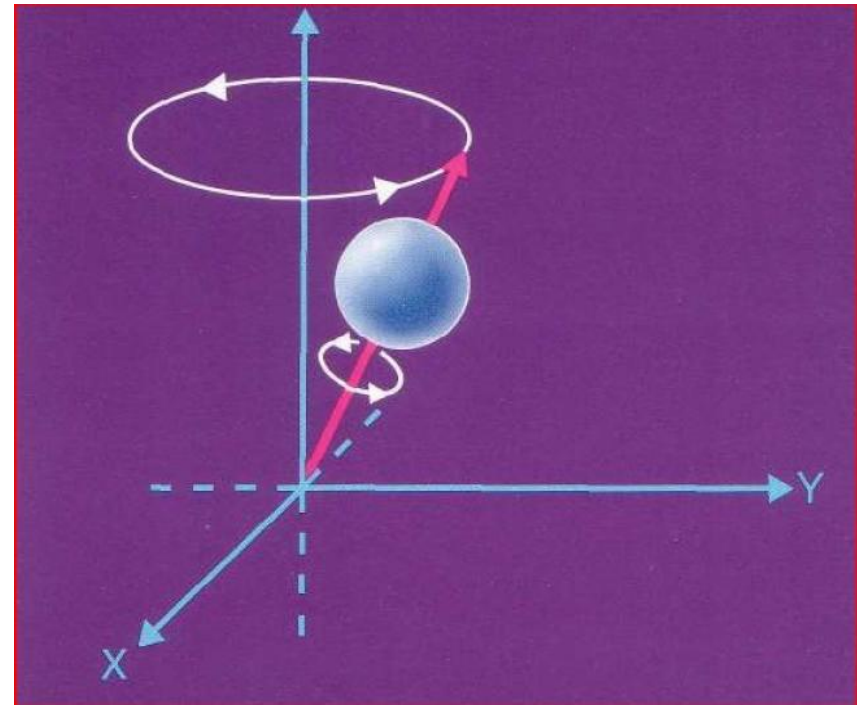
## Body in an external magnetic field ( $B_0$ )

- In our natural state Hydrogen ions in body are spinning in a haphazard fashion, and cancel all the magnetism.
- When an external magnetic field is applied protons in the body align in one direction.



# Net magnetization vector (M)

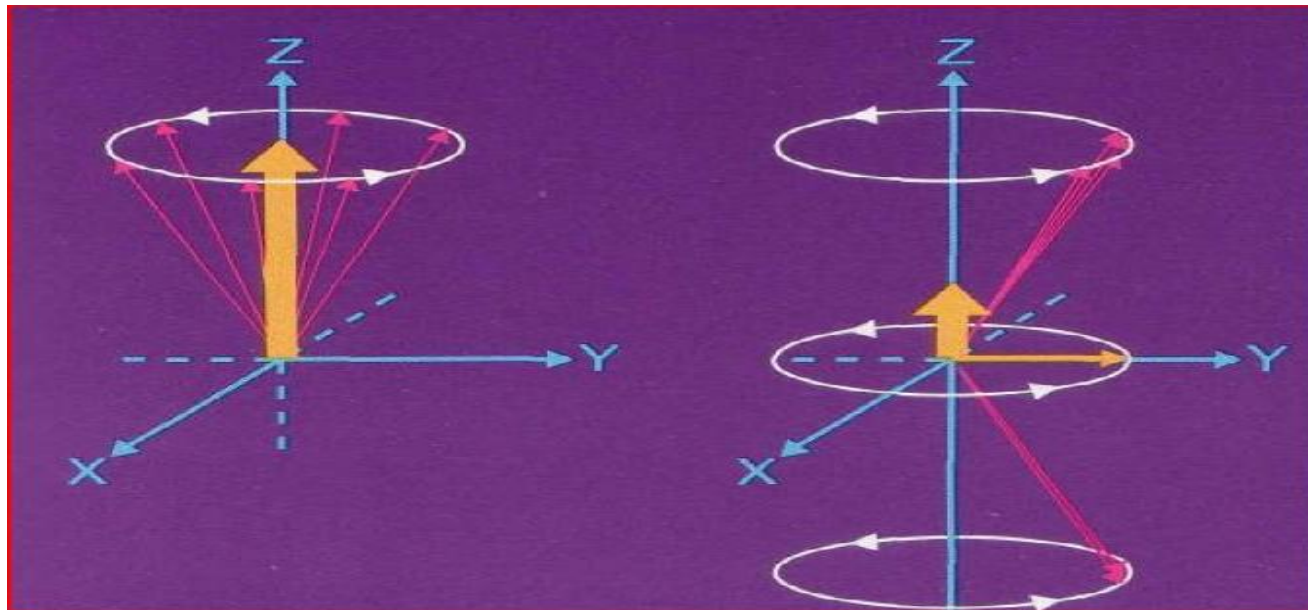
- Half of the protons align along the magnetic field and rest are aligned opposite
- 
- At room temperature, the population ratio of anti-parallel versus parallel protons is roughly 100,000 to 100,006 per Tesla



- These extra protons produce net magnetization vector (M)

# Manipulating the net magnetization

- RF waves are used to manipulate the magnetization of H nuclei
- the RF pulse causes longitudinal magnetization to decrease, and establishes a new transversal magnetization.



# Turning off the RF wave.

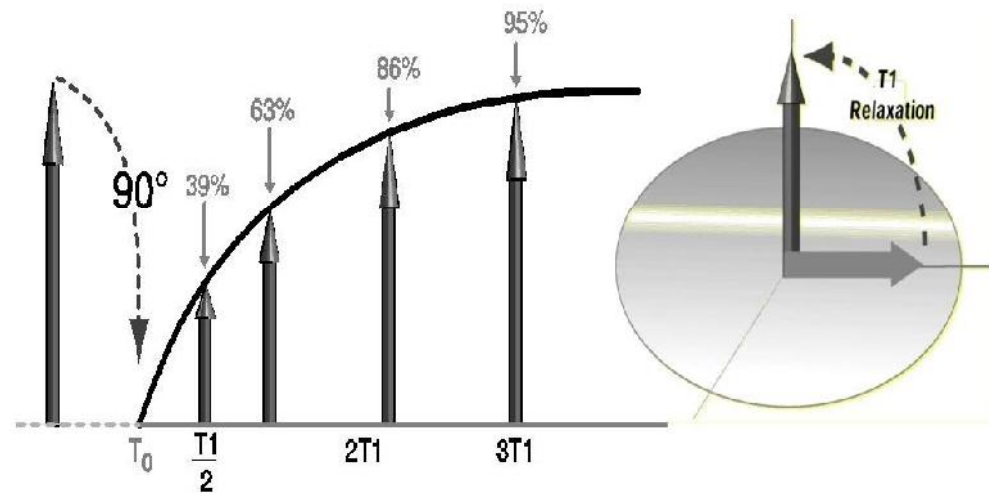
- When RF pulse is stopped higher energy gained by proton is retransmitted and hydrogen nuclei relax by two mechanisms
- **T1 or spin lattice relaxation-**  
by which **original magnetization (longitudinal)** begins to recover.
- **T2 relaxation or spin spin relaxation –**  
by which magnetization in X-Y plane (**transversal**) decays towards zero in an exponential fashion.

# T1 relaxation (spin-lattice relaxation)

After protons are Excited with RF pulse They move out of Alignment .

But once the RF Pulse is stopped they Realign after some Time And this is called t1 relaxation

## T1 Recovery (Relaxation)

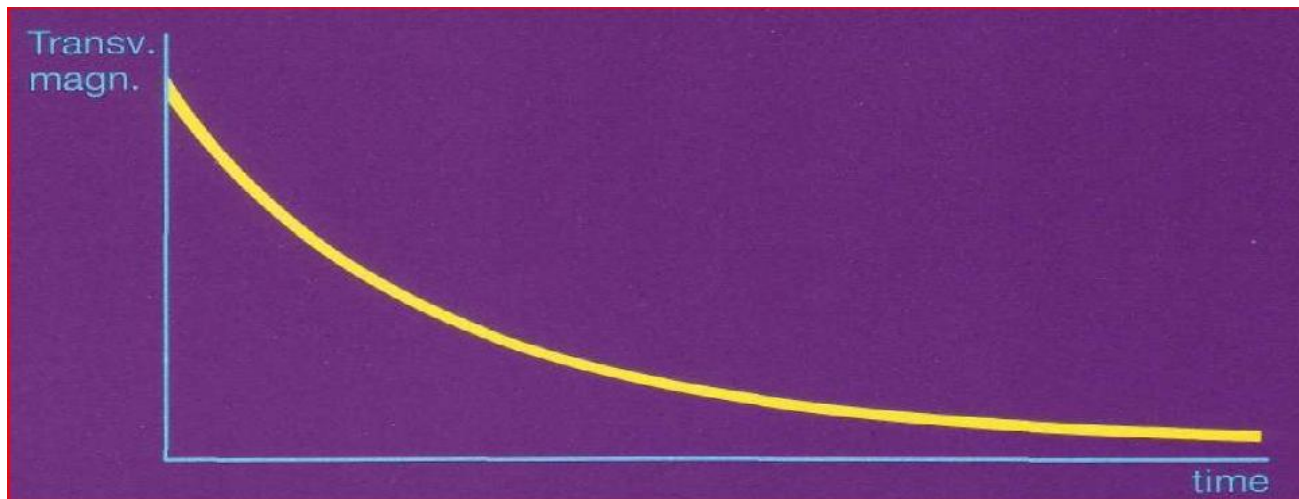


<http://www.mri.tju.edu>

T1 is defined as the time it takes for the hydrogen nucleus to recover 63% of its longitudinal magnetization

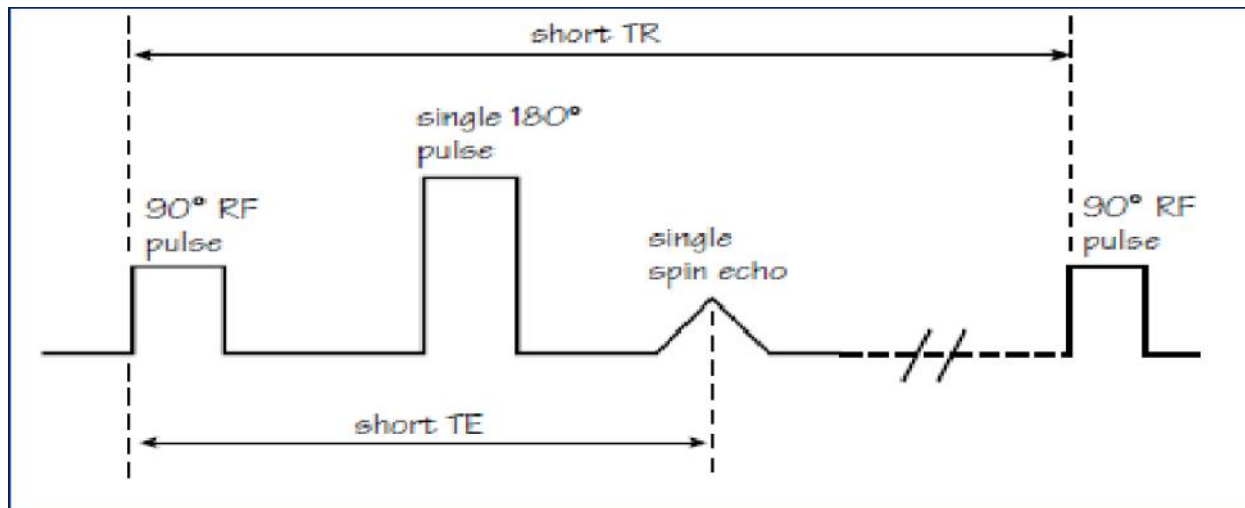
# T2 relaxation (spin-spin relaxation)

- When RF wave is switched off transversal magnetization decreases and disappears; this transversal relaxation is described by a time constant T2.
- **T2 relaxation time** is the time for 63% of the protons to become dephased owing to interactions among nearby protons.



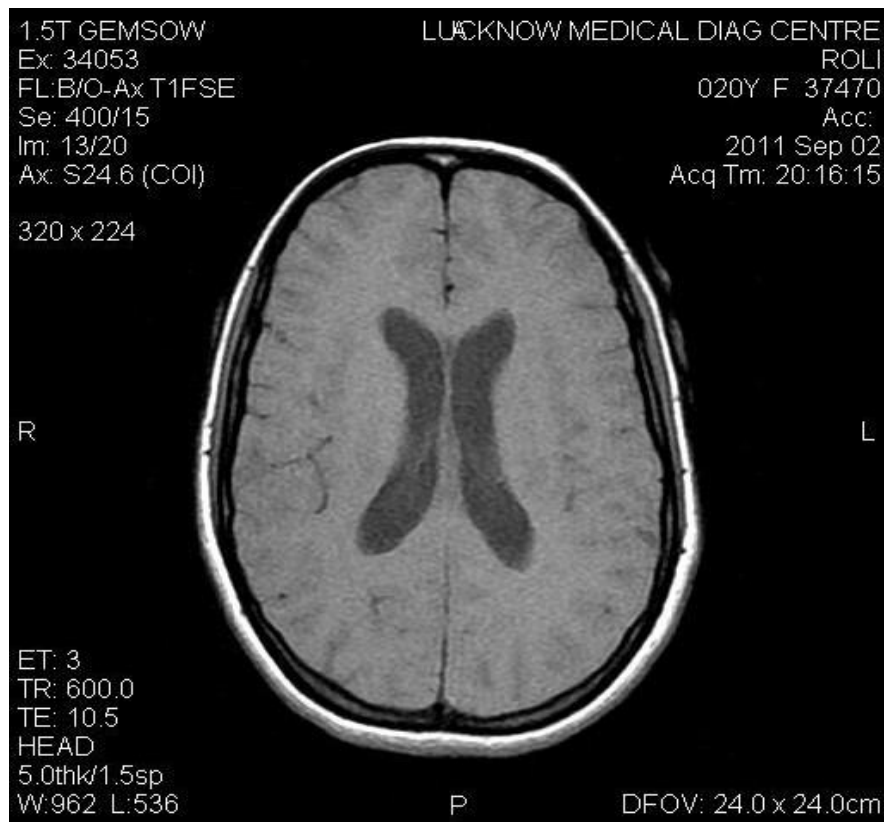
# TR & TE

- TR (**Repetition Time**): is the length of time from one  $90^\circ$  RF pulse to the next  $90^\circ$  RF pulse.
- TE (**Echo Time**): This is the time between an RF excitation pulse and the collection of the signal.



# Short TR+ short TE= T 1 weighted image

- In general a short TR (<1000ms) and short TE (<45 ms) scan is T1WI.

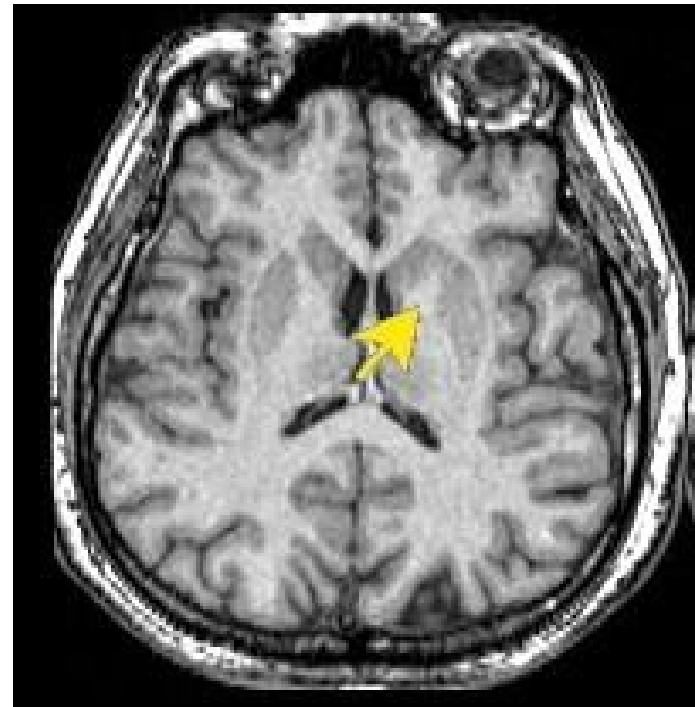


# T1 Characteristics

- White matter **brighter** than Gray.

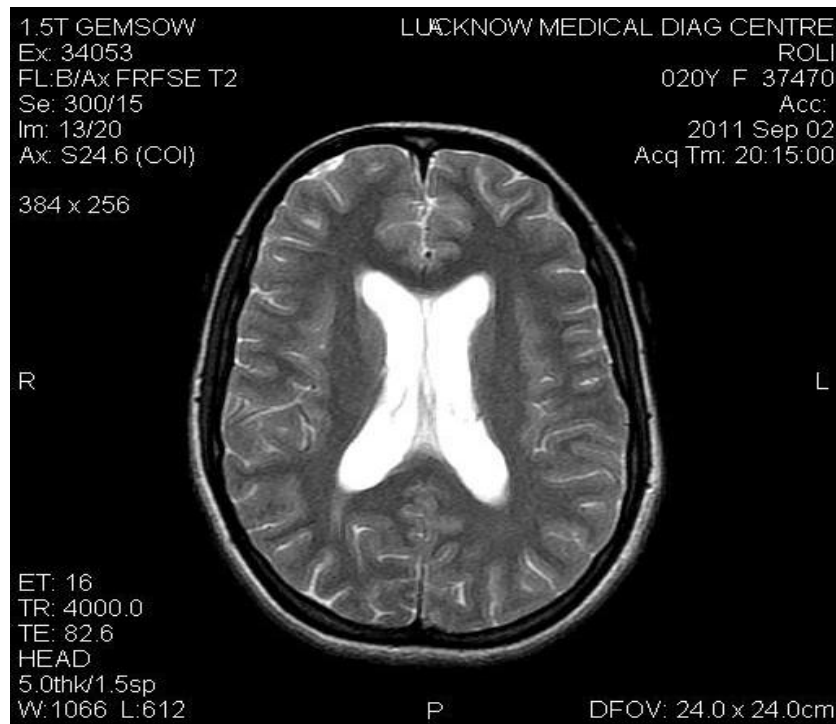
Dark on T 1: **CSF**, Edema, tumor, infection, inflammation, **hemorrhage (hyper acute, chronic)**, Low proton density, calcification.

Bright on T1: **Fat, sub acute hemorrhage**, melanin, protein rich fluid, Slowly flowing blood, **Paramagnetic substances (gadolinium, copper, manganese)**.



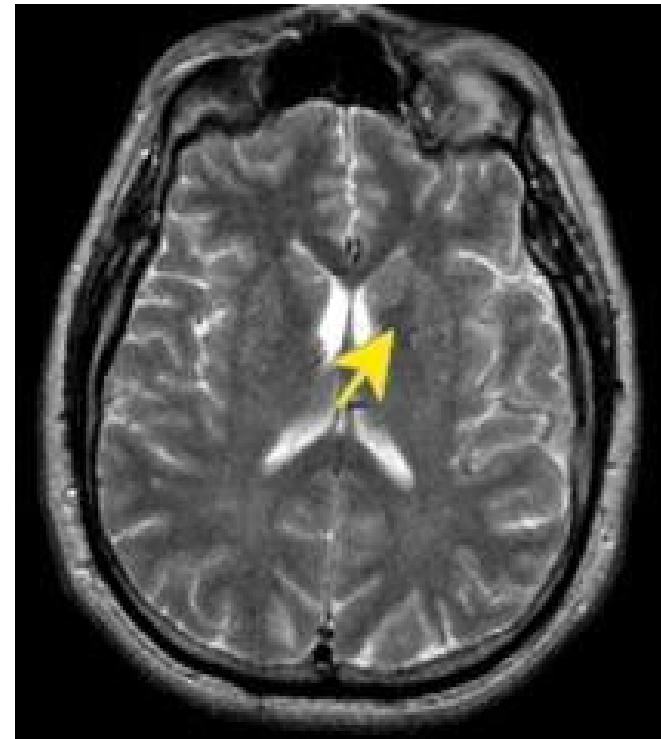
# Long TR+ Long TE= T 2 weighted image

- Long TR (>2000ms) and long TE (>45ms) scan is T2WI.



# T 2 Characteristics

- Gray matter brighter than white.
- **Bright on T 2: CSF, Edema, tumor, infection,** inflammation, subdural collection, Methemoglobin in late sub acute hemorrhage
- **Dark on T2: calcification,** fibrous tissue, deoxyhemoglobin, methemoglobin (intracellular), iron, hemosiderin, melanin



# Fluid-attenuated inversion recovery (FLAIR)

- T2-weighted imaging is well suited for **lesion detection** in the brain because most lesions appear **hyperintense** with this sequence.
- However CSF also appears hyperintense on T2-weighted spin-echo (SE) images.
- Therefore, lesions at CSF interfaces, such as cortical sulci and ventricles, may be mistaken for extensions of CSF or partial volume effects.

- The conventional FLAIR technique employs a 180 degree RF pulse to flip the net magnetization vector 180° and null the signal from a particular entity (eg, water in tissue).
- When the RF pulse ceases, the spinning nuclei begin to relax. When the net magnetization vector for water passes the transverse plane (the null point for that tissue), the conventional 90° pulse is applied, and the SE sequence then continues as before.
- The interval between the 180° pulse and the 90° pulse is the TI ( Inversion Time).

- FLAIR imaging **suppresses signal from free water in CSF** and maintains hyperintense lesion contrast.
- FLAIR image have a long TR and TE and an inversion time (TI) that is tailored to null the signal from CSF.
- FLAIR sequences are particularly useful in evaluation of **MS, infarcts, SAH**

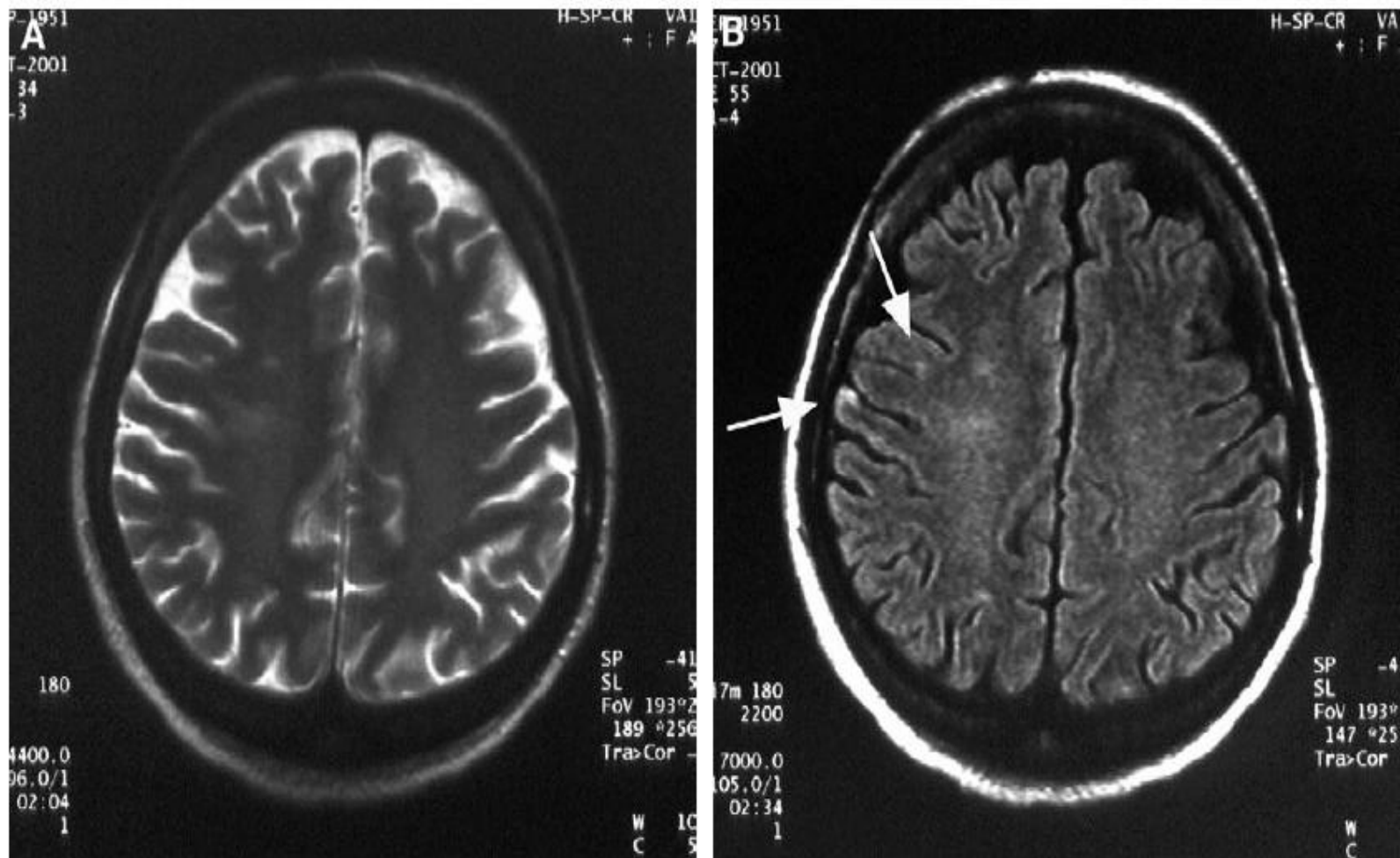


Fig. 10. A 68-year old patient with embolic infarcts in the right middle cerebral artery distribution. Small hyperintense cortical lesions are virtually imperceptible on an axial fast spin-echo T2-weighted image (A), whereas they are clearly visualized on a fast spin-echo FLAIR image (arrows) (B).

# Which scan best defines the abnormality

## **T1 W Images:**

- Subacute Hemorrhage
- Fat-containing structures
- Anatomical Details

## **T2 W Images:**

- Edema
- Demyelination
- Infarction
- Chronic Hemorrhage

## **FLAIR Images:**

- Edema,
- Demyelination
- Infarction esp. in Periventricular location

# Diffusion-weighted MRI


- Diffusion-weighted MRI is an example of endogenous contrast, using the motion of protons to produce signal changes
- DWI images are obtained by applying pairs of opposing and balanced magnetic field gradients (but of differing durations and amplitudes)
- The primary application of DW MR imaging has been in brain imaging, mainly because of its exquisite sensitivity to early detection of ischemic stroke

# Other Causes of Positive DWI

- Bacterial abscess, Epidermoid tumor
- Acute demyelination
- Tumors undergoing central necrosis
- Tumors with high nuclear:cytoplasmic ratios
- Acute encephalitis
- Jacob Creutzfeld disease
- Elevated protein or subacute blood
- T2 shine-through (High ADC)

# Apparent Diffusion Coefficient

- It is a measure of diffusion
- Calculated by acquiring two or more images with a different gradient duration and amplitude
- The lower ADC measurements seen with early ischemia

- 
- The ADC may be useful for estimating the lesion age and distinguishing acute from subacute DWI lesions.
  - Acute ischemic lesions can be divided into hyperacute lesions (low ADC and DWI-positive) and subacute lesions (normalized ADC).
  - Chronic lesions can be differentiated from acute lesions by normalization of ADC and DWI.

# Approaching , MRI FILM

- **Image Delineation**

for normal anatomy –preferred scan is T1W

for any pathology Preferred scan is T2W→T1W


Usual Order: Axial> Sagittal>Coronal.

- **Skull**

- Soft tissue
- Diploic Spaces

- **Ventricles, cisterns & sulci**

- Size: Hydrocephalus
- Shape: Mass Effect
- Symmetry.

- 
- Symmetry of Intracranial contents
    - Normal grey-white differentiation,
    - Deep nuclei
    - Brainstem & cerebellum
    - Sinus and blood vessels
  - Focal abnormalities
    - Space Occupying Lesion
    - Signal Intensity Changes



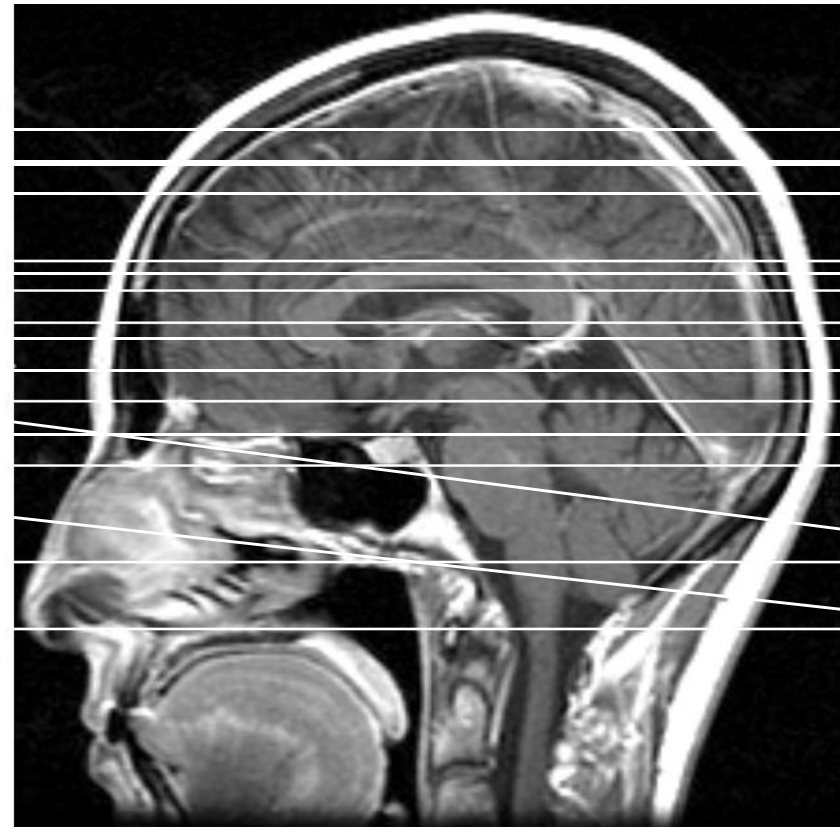
# NORMAL RADIOLOGICAL ANATOMY ON MRI

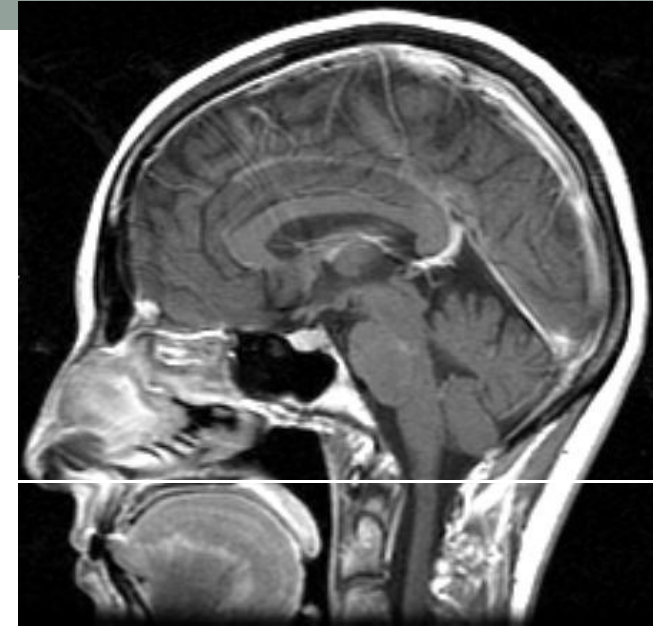
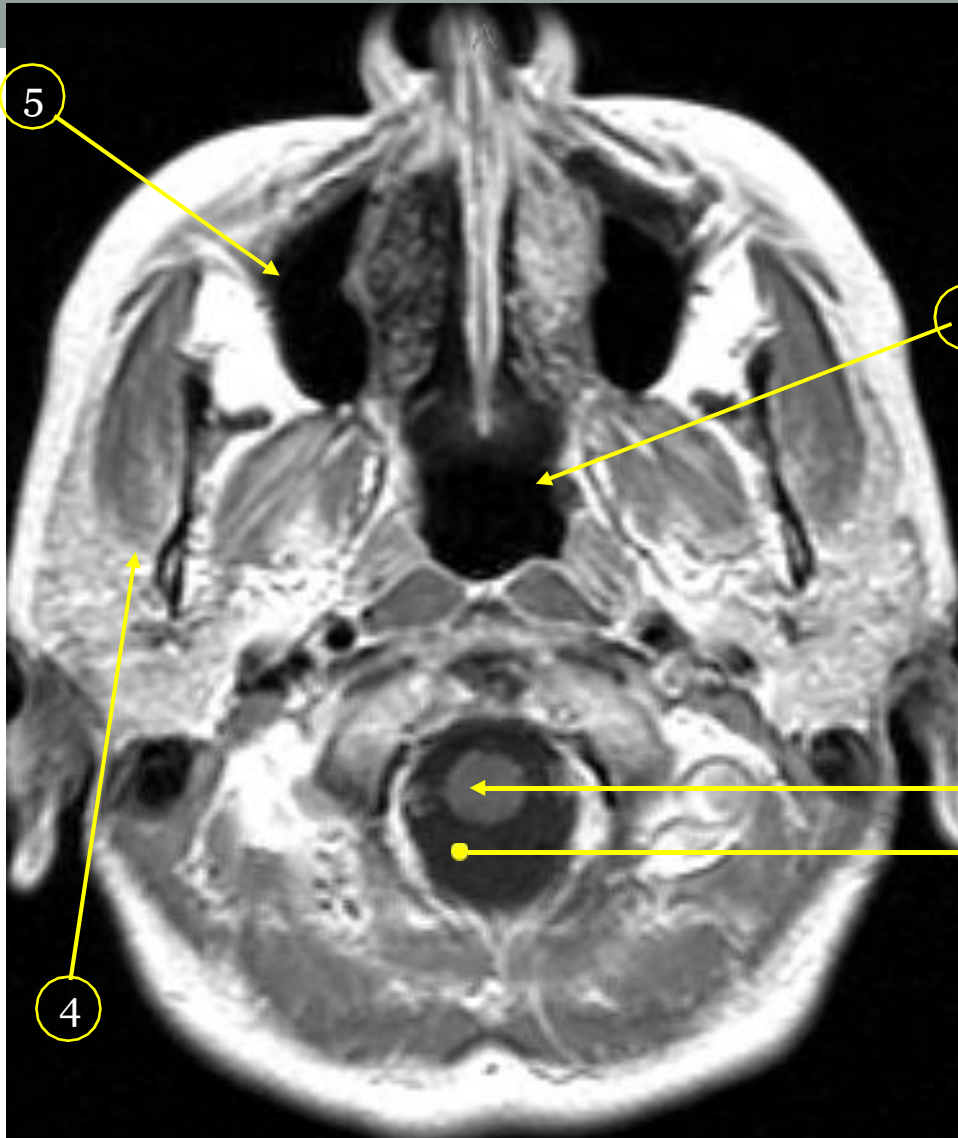
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# Axial Sections

- **At level of**

1. Foramen magnum.
2. Medulla .
3. Pons .
4. Midbrain .
5. Third ventricle
6. Thalamus.
7. Body of lateral ventricle.
8. Body of corpus callosum.
9. Above lateral ventricle





1. Cisterna Magna
2. Cervical Cord
3. Nasopharynx
4. Mandible
5. Maxillary Sinus

**Axial T1 Weighted M.R.I.  
Section at the level of Foramen  
Magnum**

# At the level of caudal medulla

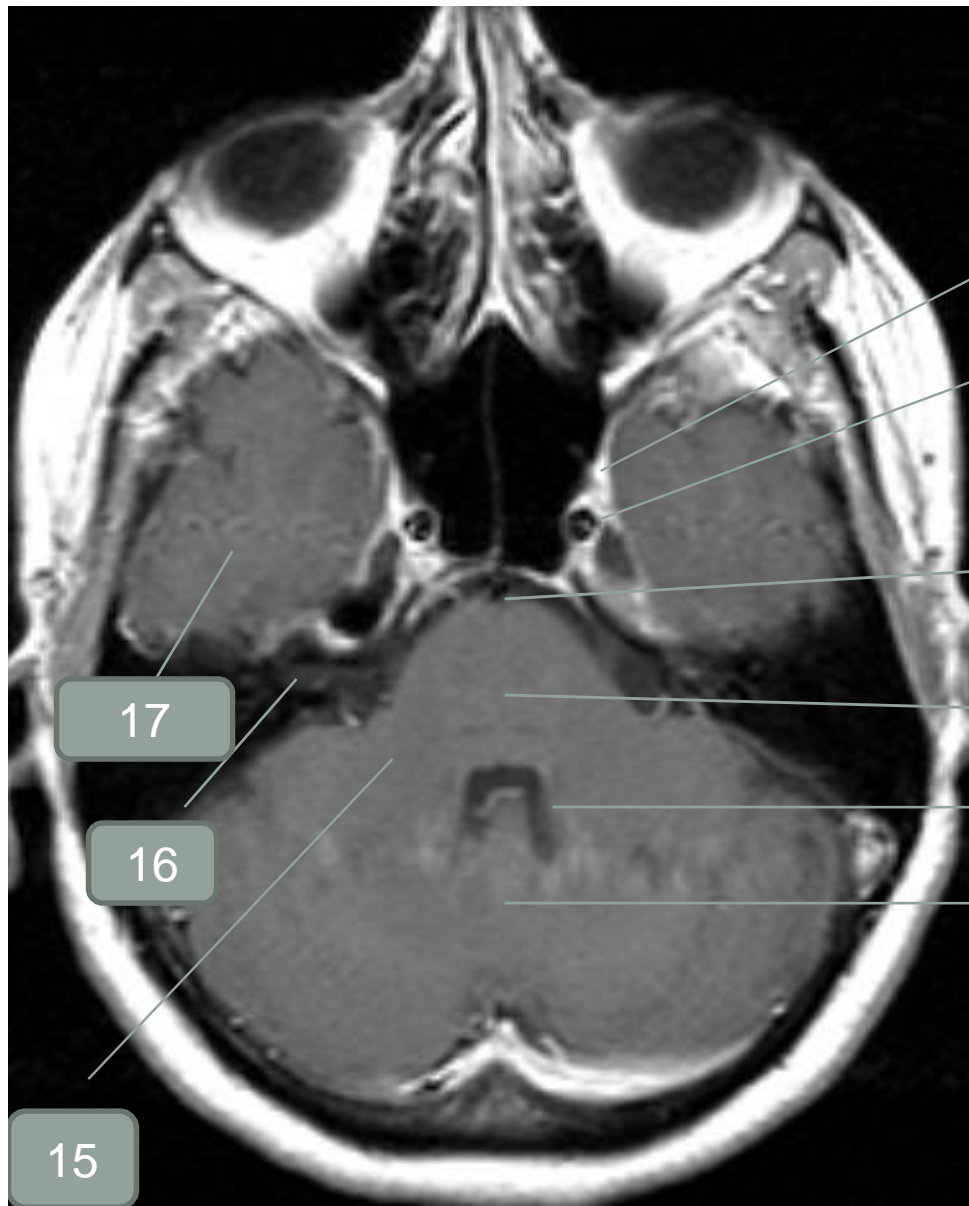


Maxillary  
sinus

Flow void of vertebral A.

Medulla oblongata

cerebellum



14

ICA

12

11

10

9

17

16

15

- 9. Vermis
- 10. IV Ventricle
- 11. Pons
- 12. Basilar Artery
- 13. Internal Carotid Artery
- 14. Cavernous Sinus
- 15. Middle Cerebellar Peduncle
- 16. Internal Auditory Canal
- 17. Temporal Lobe

• **Post Contrast Axial T1W MR Image of the brain at pons**



20

16

17

15

19

18

14

13

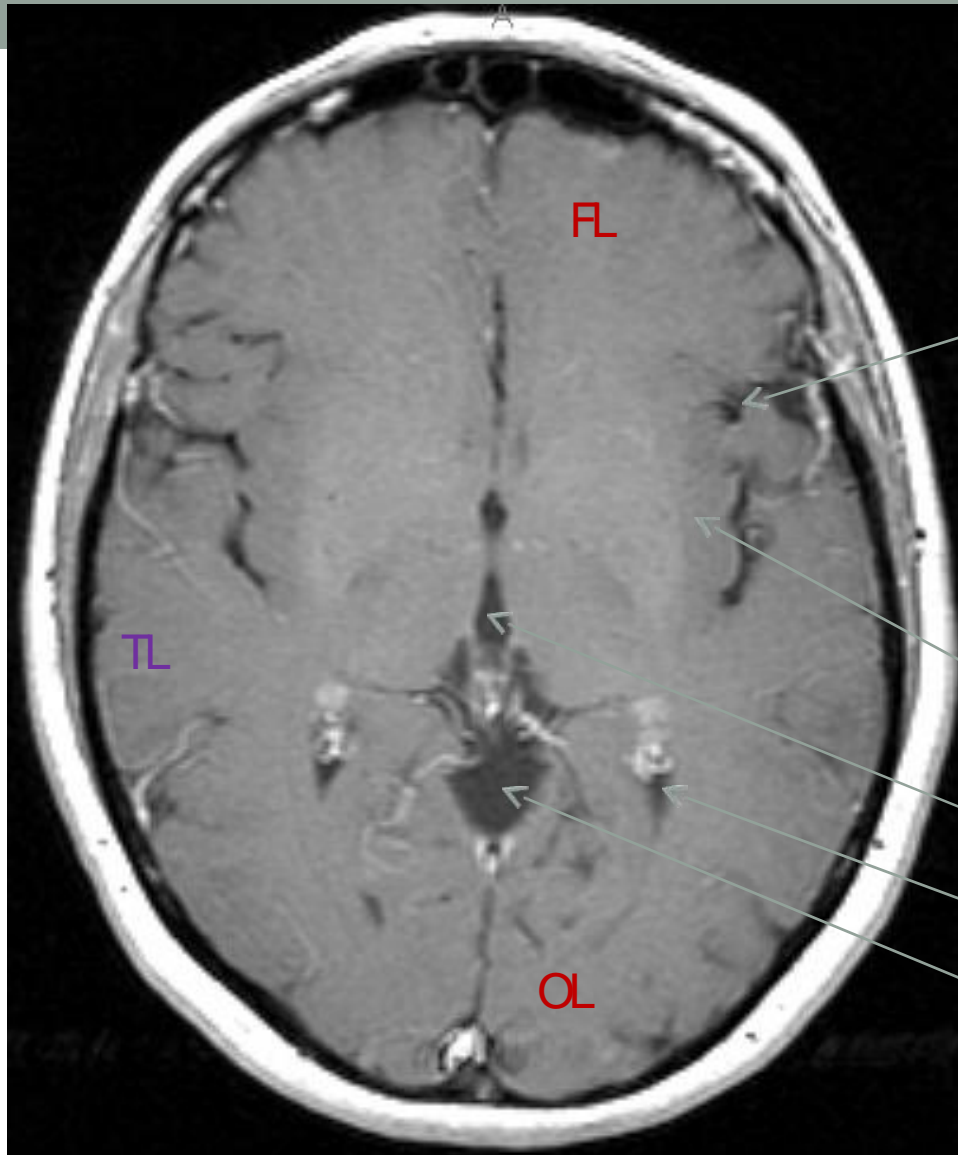
12

22

21

- 12. SUP CEREBELLAR CISTERN
- 13. QUADRIGEMINAL CISTERN
- 14. PERIMESENCEPHALIC SUPRACELLAR CISTERN
- 15. SUPRASELLAR CISTERN
- 16. OPTIC CHIASMA
- 17. INFUNDIBULUM
- 18. AQUEDUCT OF SYLVIVS
- 19. MIDBRAIN
- 20. ORBITS
- 21. POSTERIOR CEREBRAL ARTERY
- 22. MIDDLE CEREBRAL ARTERY

**Fig. 1.4 Post Contrast Axial T1W MR Image of the brain AT THE LEVEL OF MID BRAIN**



Sylvian  
Fissure

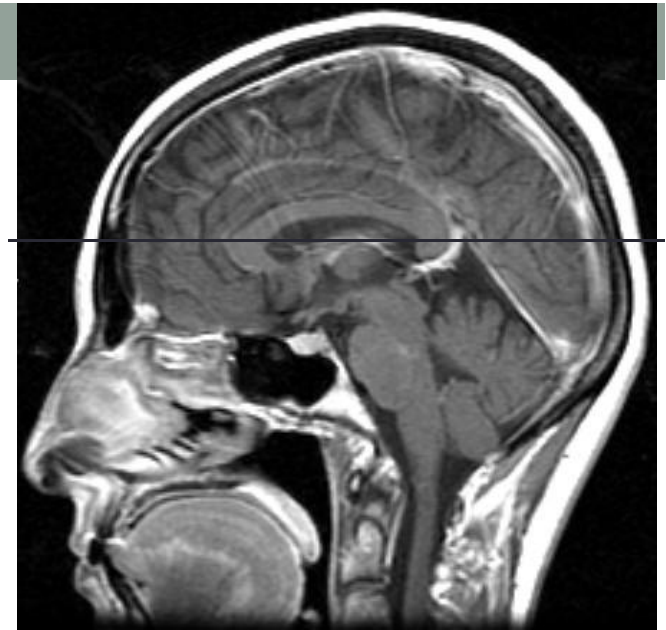
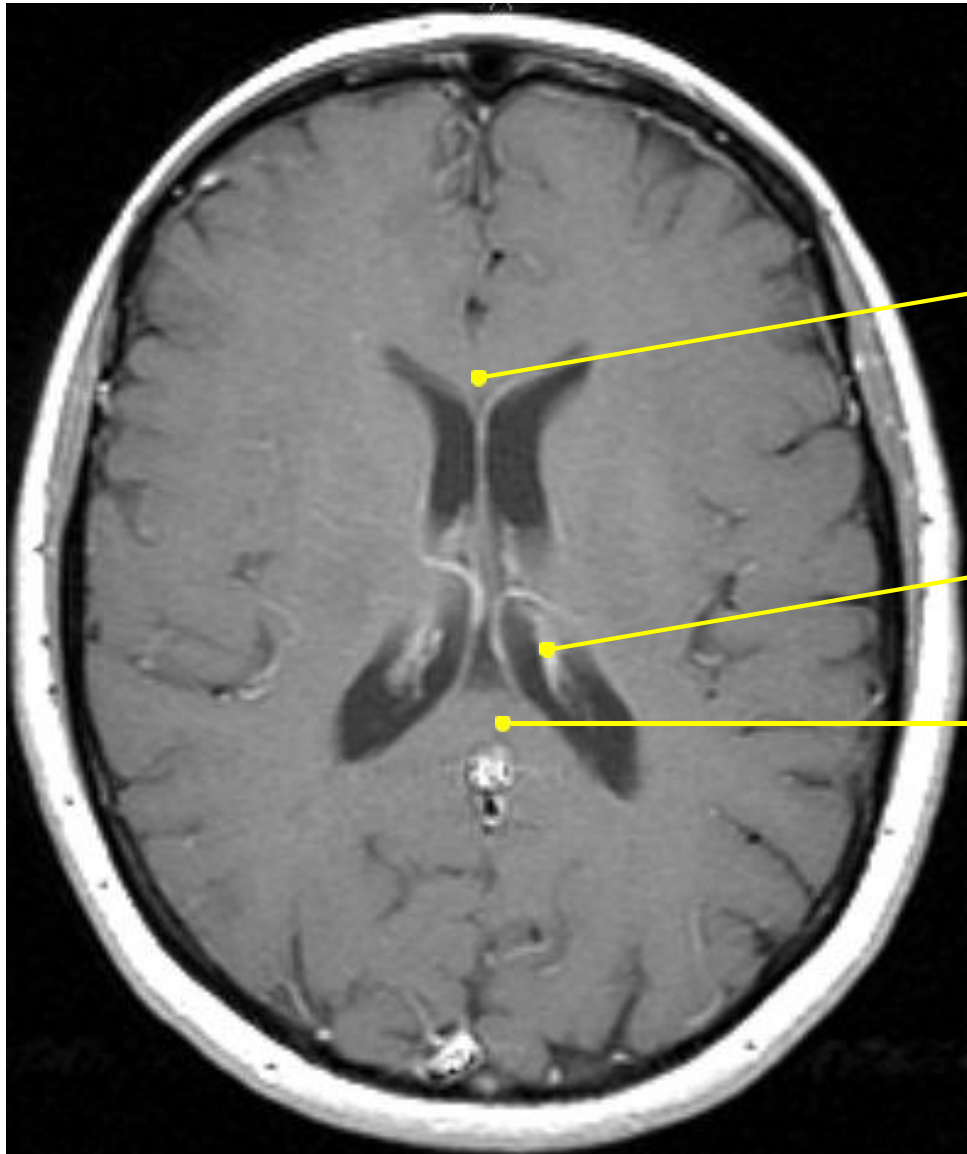
Insular cortex

Third ventricle

Temporal horn

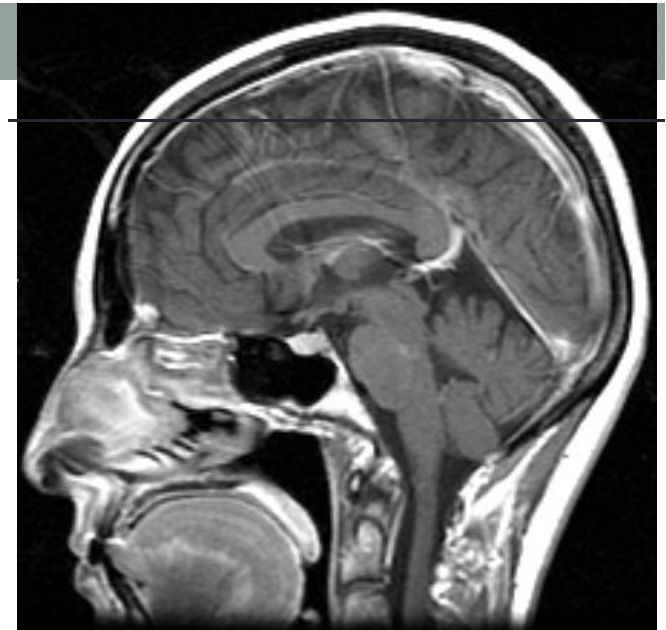
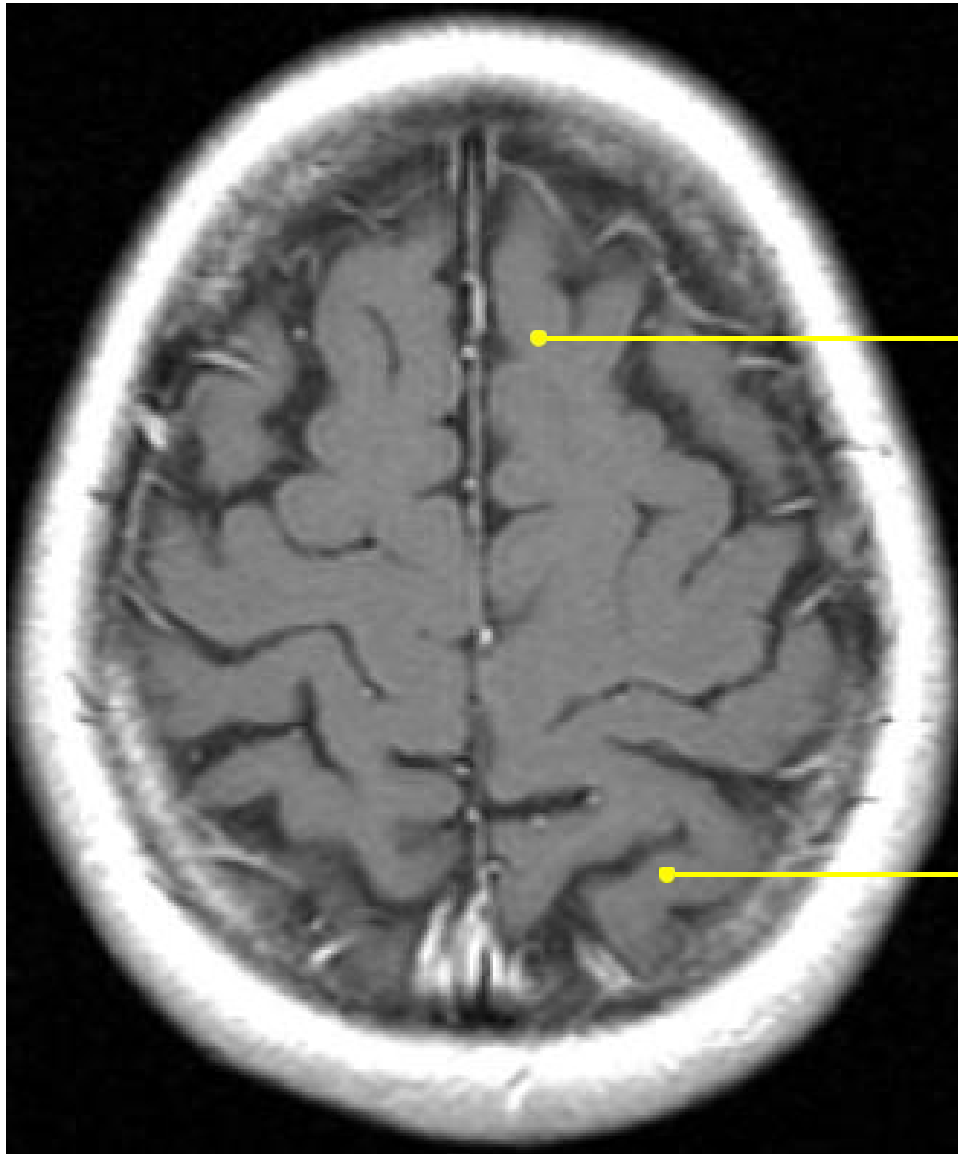
Sup cerebellar cistern

**Post Contrast Axial MR Image of the brain  
Section at the level of third ventricle**



**Post Contrast sagittal T1 W M.R.I.  
Section at midventricular level**

1. Genu of corpus callosum
2. Choroid plexus
3. Splenium of corpus callosum



2

1

1. Parietal Lobe

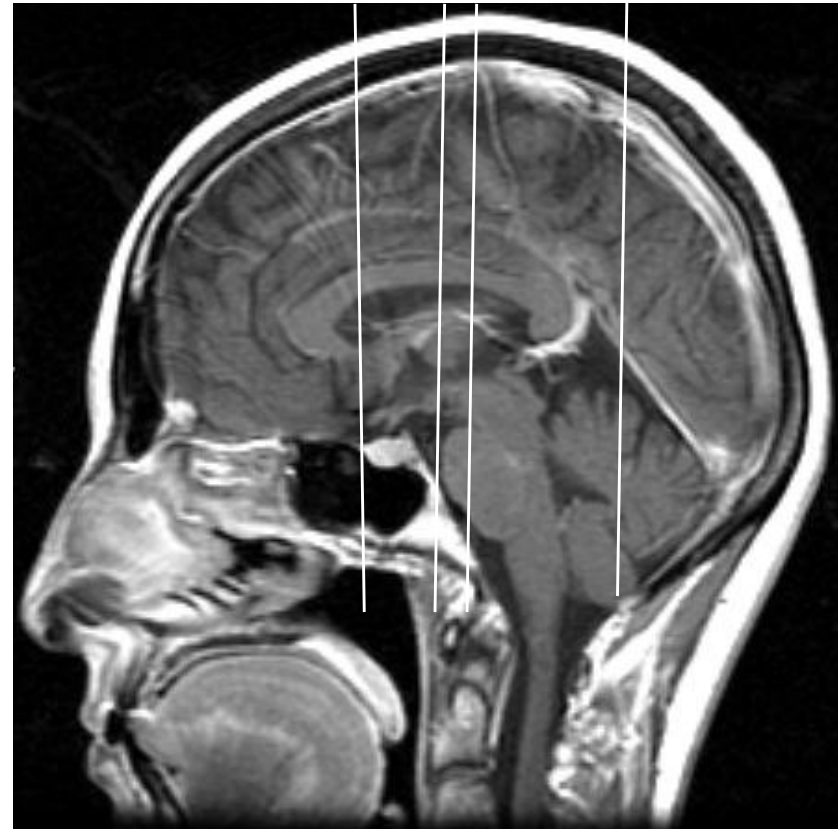
2. Frontal Lobe

**Post Contrast sagittal T1 Wtd  
M.R.I.  
Section above the Corpus  
Callosum**

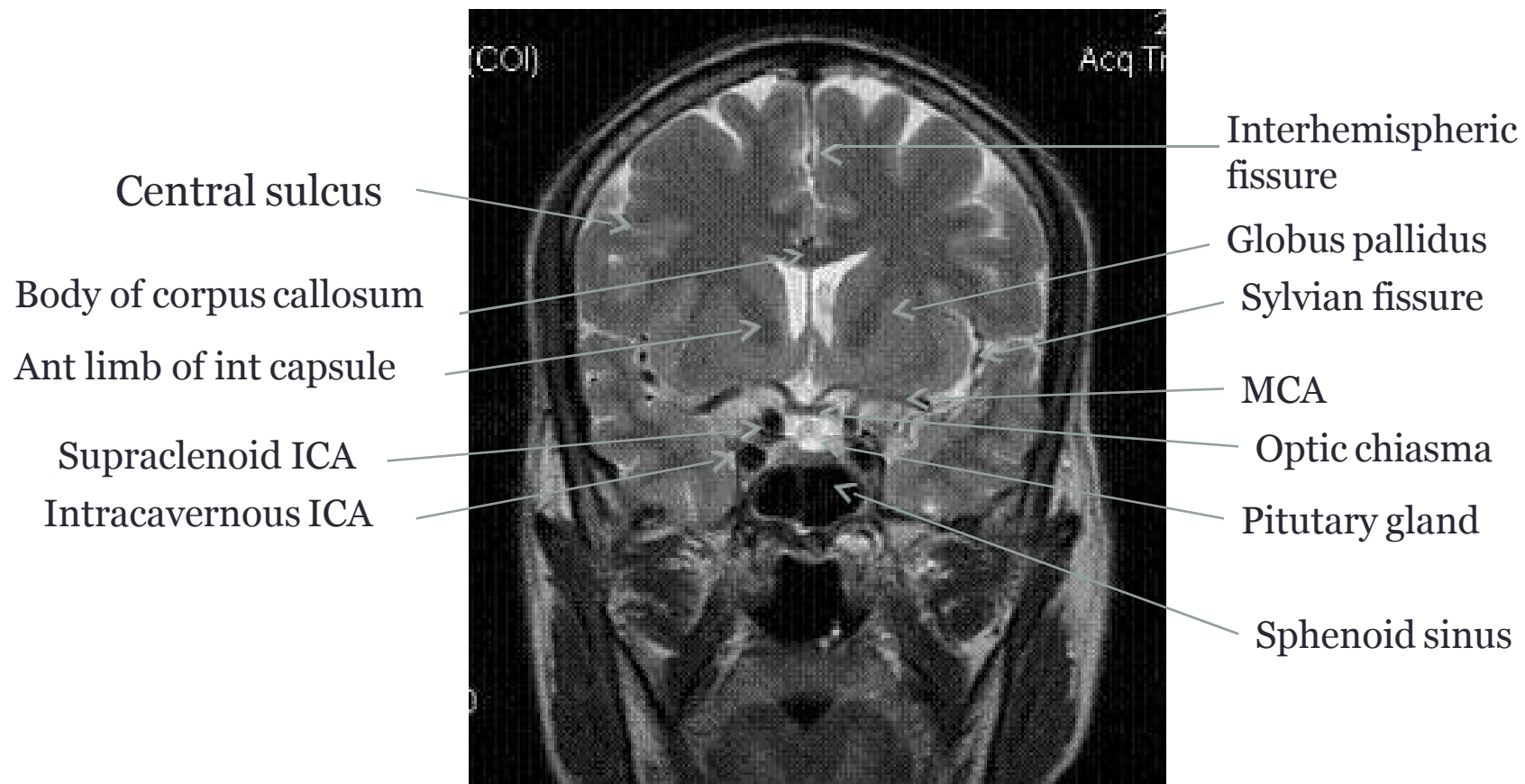
# Coronal sections of brain

At the level of-

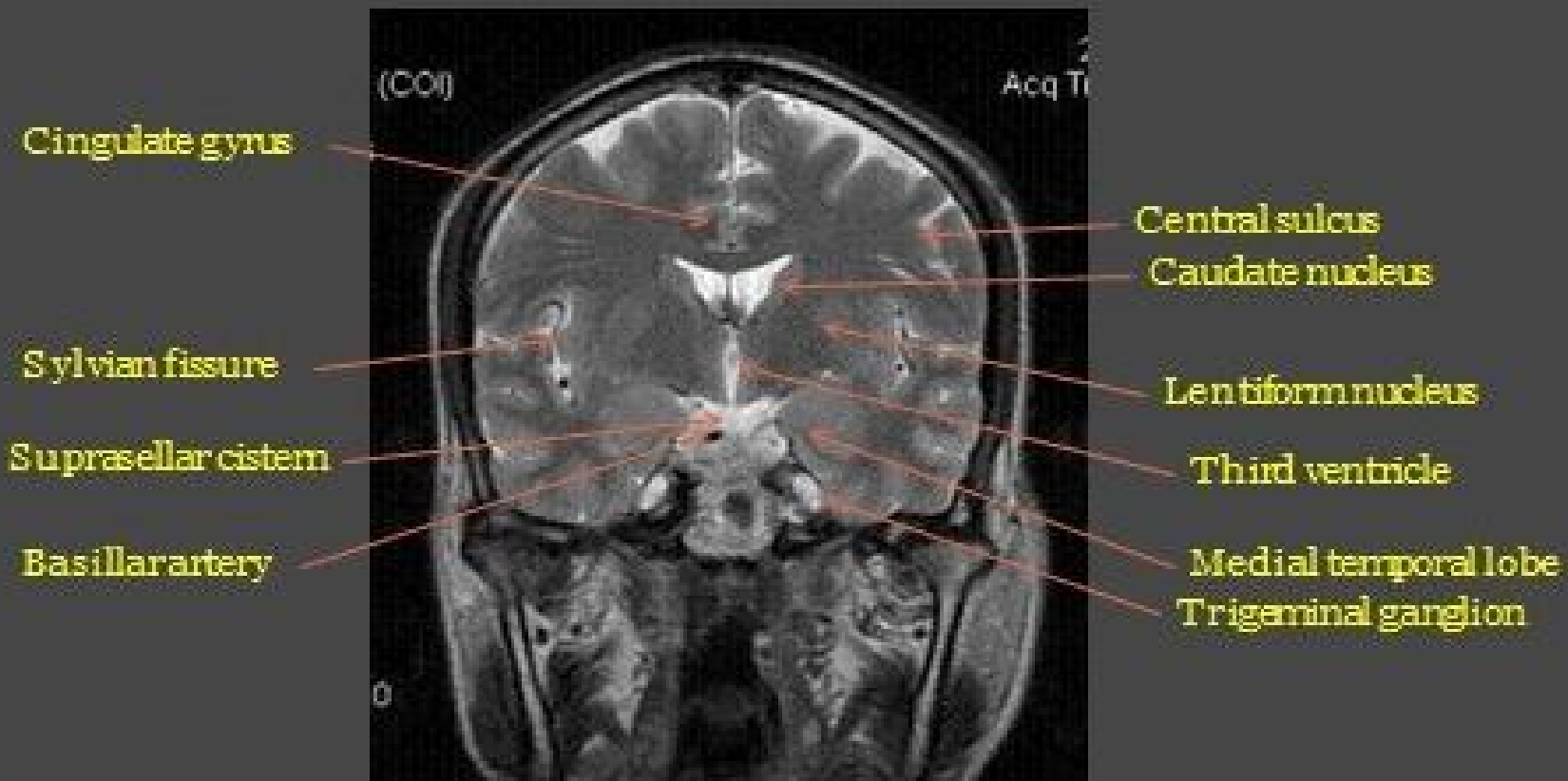
1. Frontal horns.
2. Third ventricle.
3. Mid-ventricular level.
4. Occipital horn level



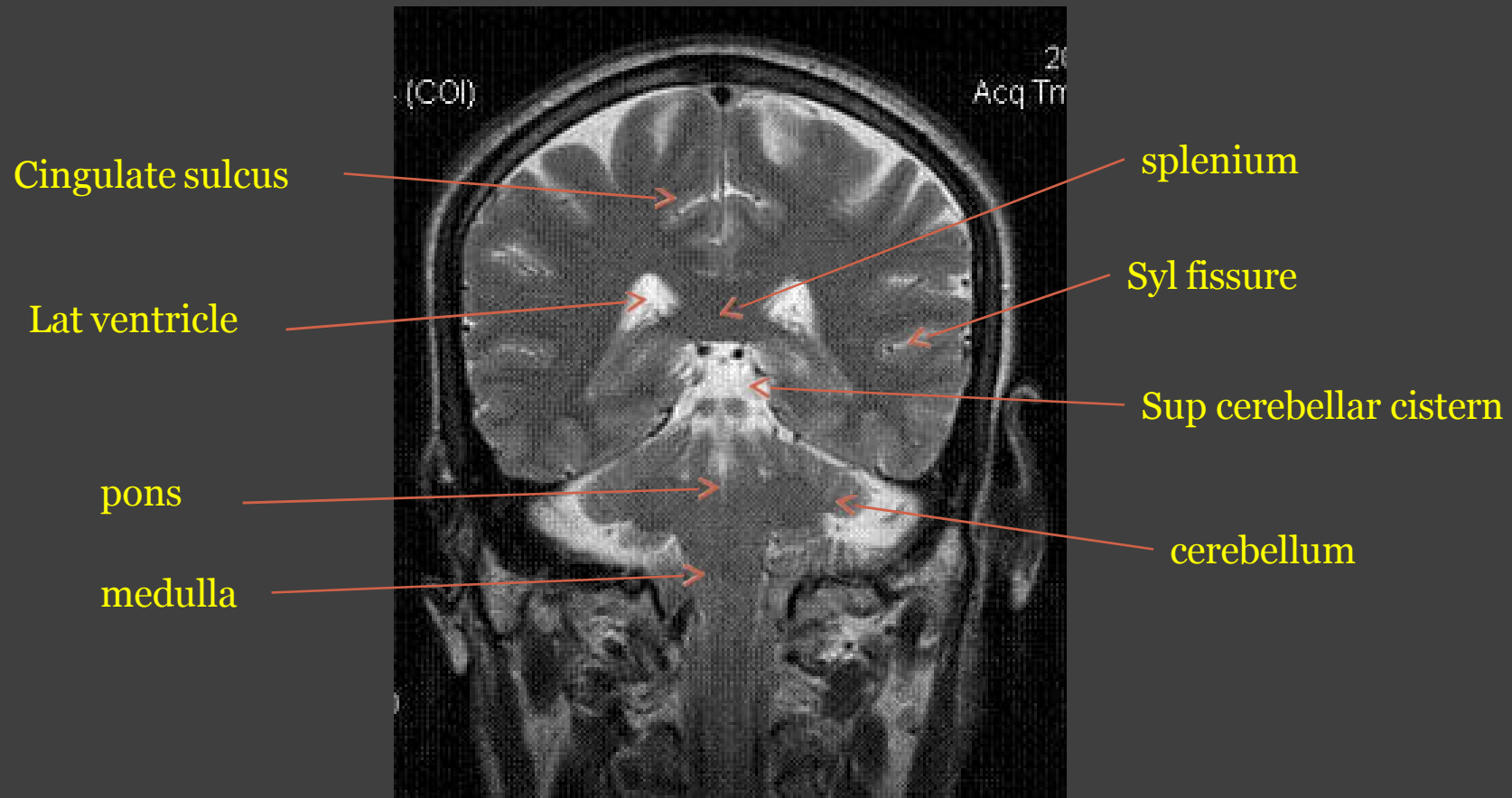
# CORONAL SECTION AT THE LEVEL OF FRONTAL HORN



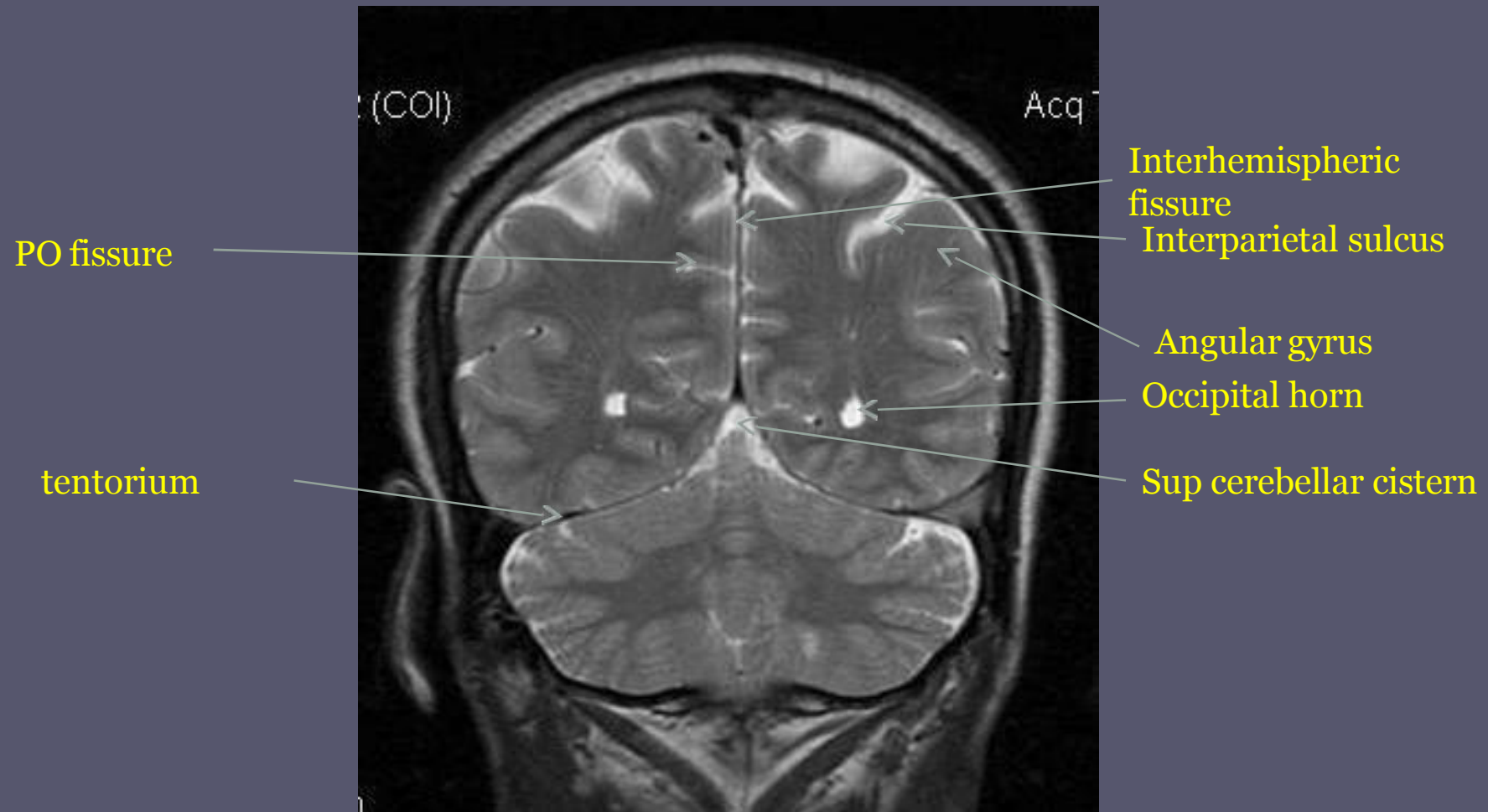
## CORONAL SECTION AT THE LEVEL OF THIRD VENTRICLE



# CORONAL SECTION AT MID VENTRICULAR LEVEL



# CORONAL SECTION AT THE OCCIPITAL HORN LEVEL



# Sagittal sections

- At the level –

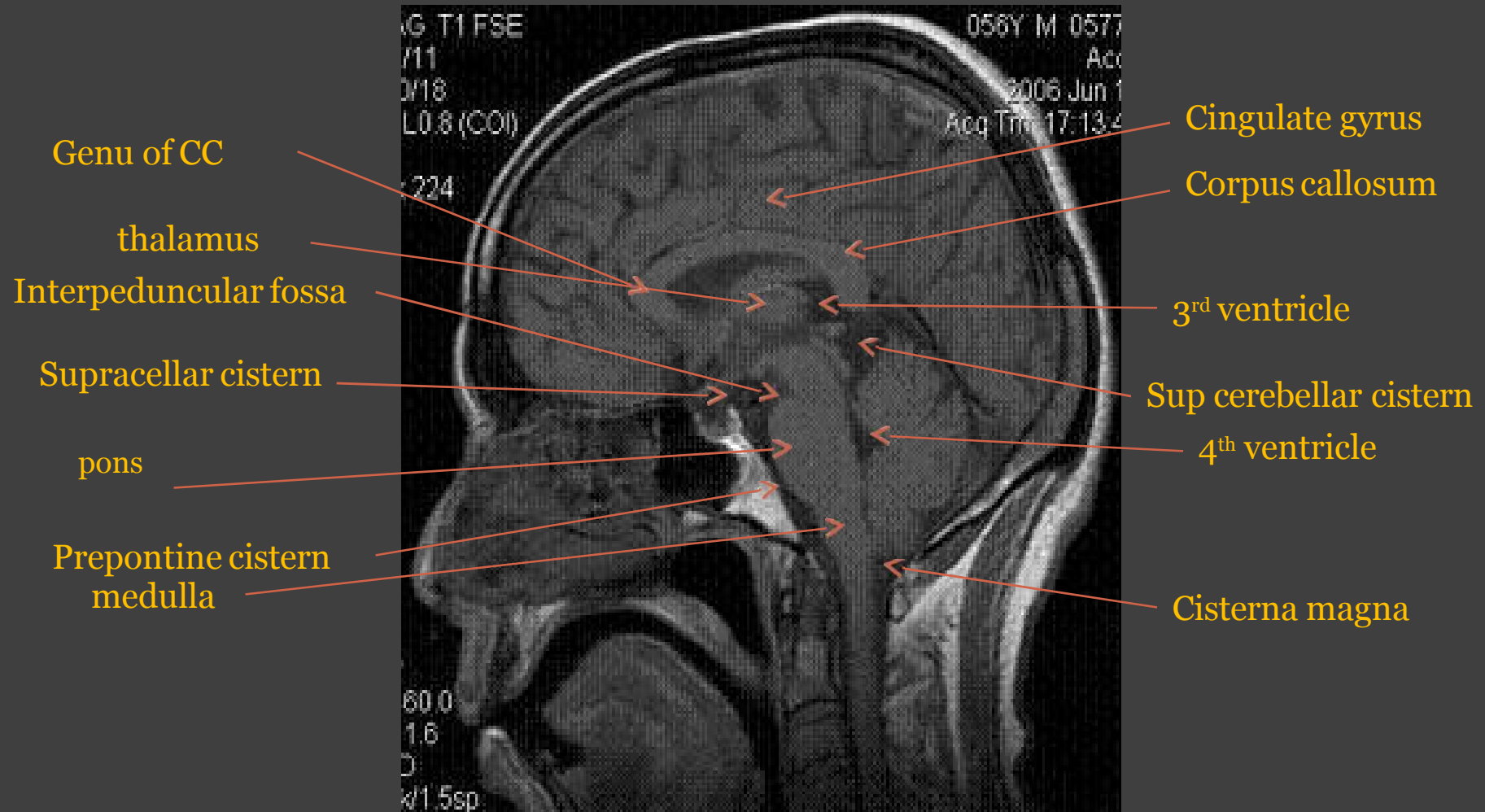
1. Midsagittal

2. Internal capsule

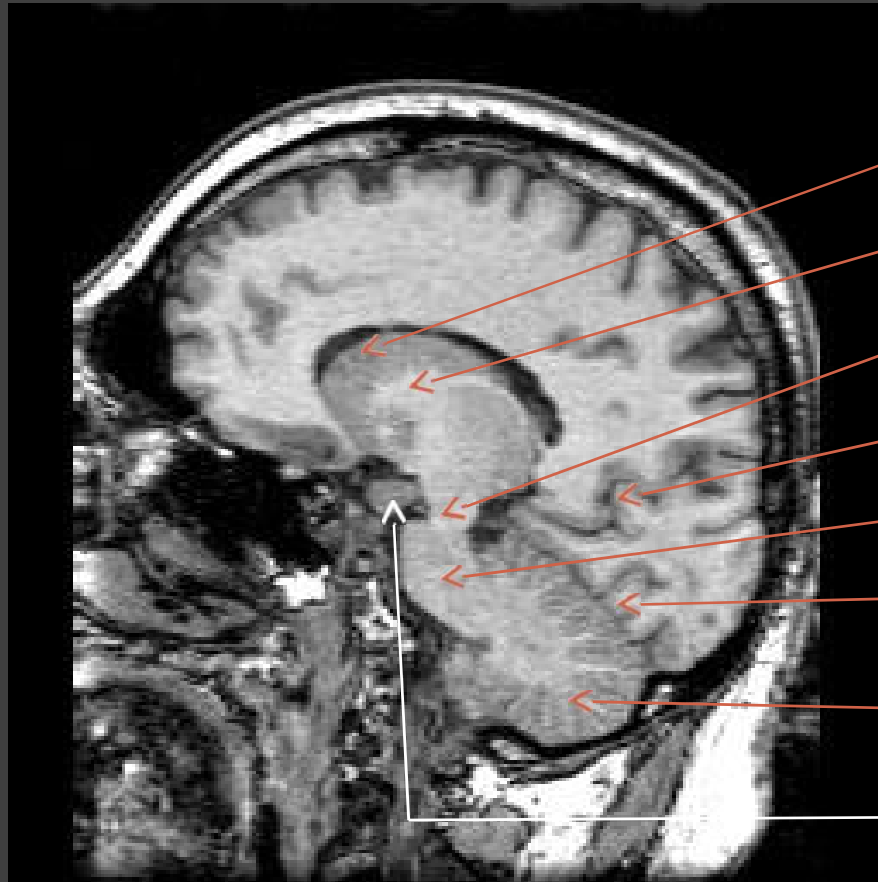
3. Parasagittal

4. Lateral orbital

# Sagittal section at mid sagittal level



# Sagittal section at internal capsule level



head of caudate

Internal capsule

cerebral peduncle

parieto occipital sulcus

middle cerebellar peduncle

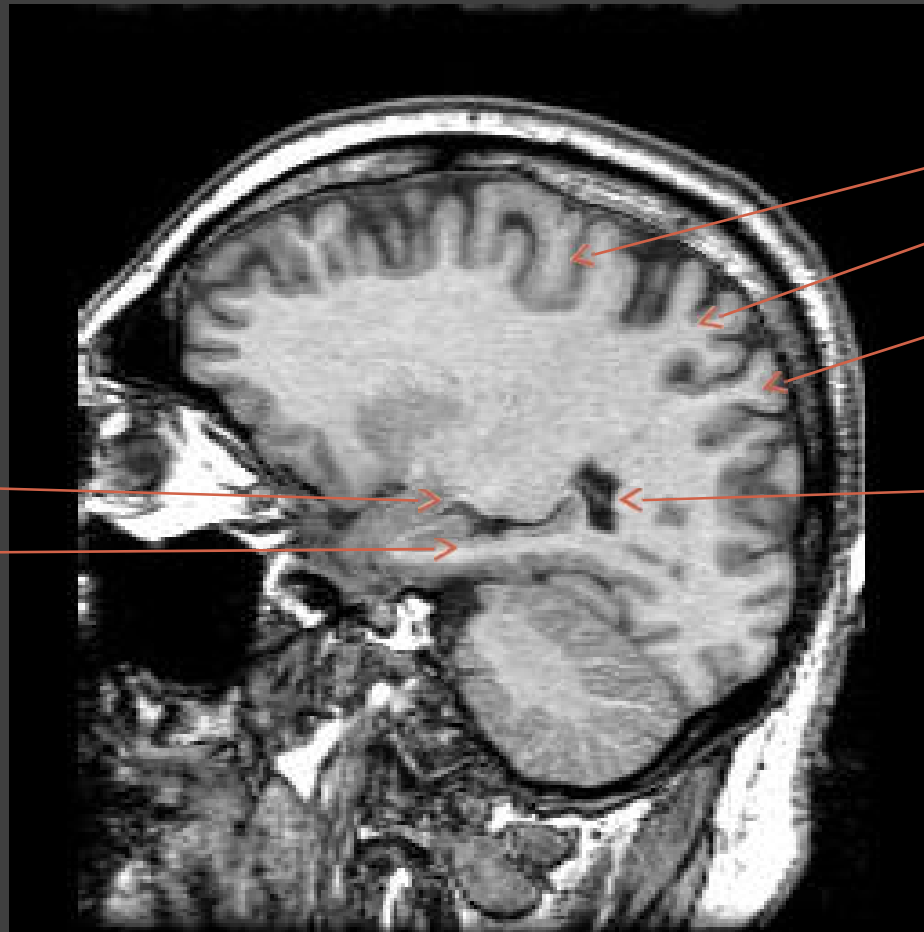
tentorium cerebelli

cerebellum

amygdala

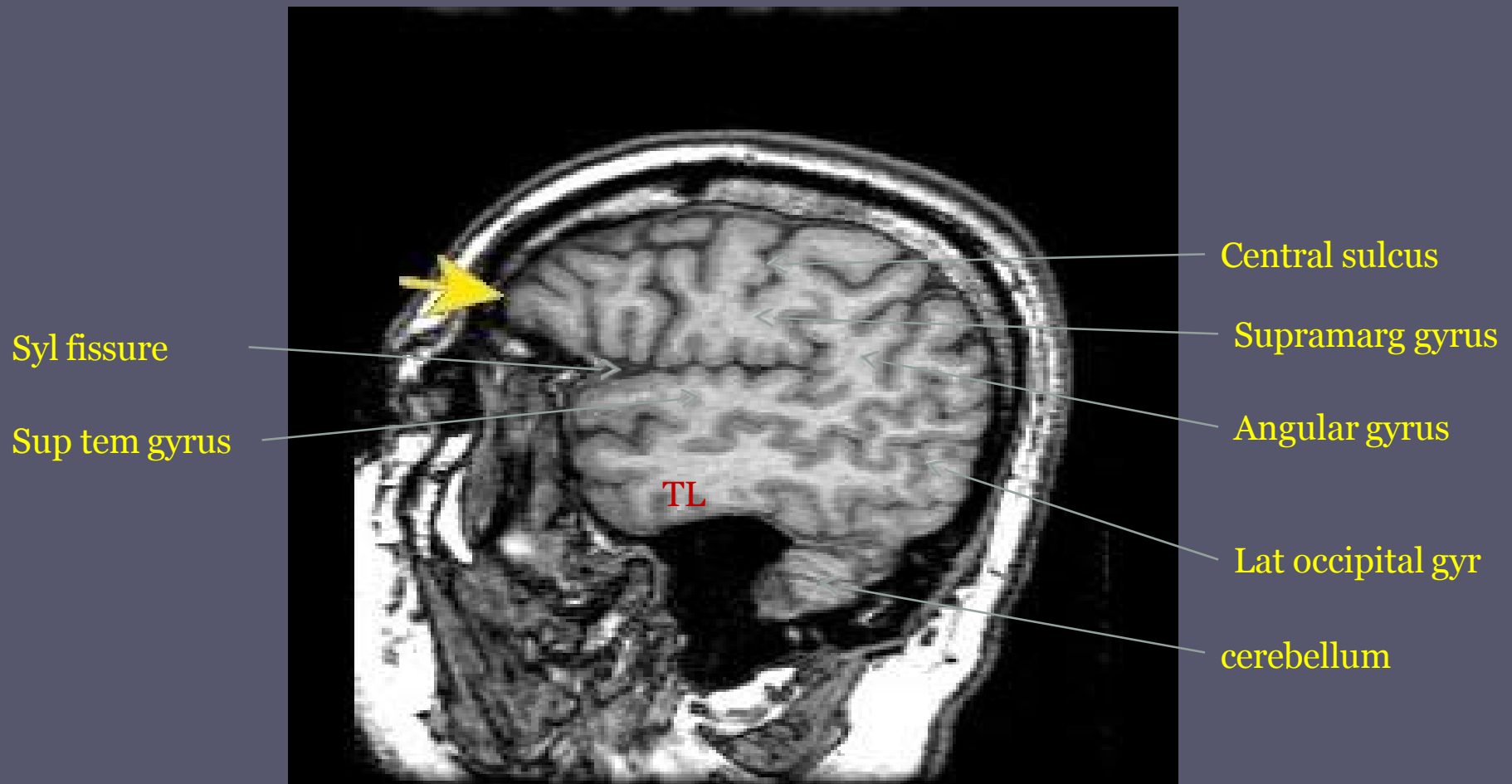
# At Parasagittal section

Sylvian fissure  
Temporal lobe



Central sulcus  
Supramarginal gyrus  
Angular gyrus  
Lat ventricle

# Sagittal section at lateral orbital level



# Contrast agents

- Principles of contrast uptake are same in CT and MRI i.e. enhancement of CNS pathology due to disruption of blood brain barrier
- Unlike contrast agents used in CT which are directly visualized those used in MRI produce local alteration in the magnetic environment that influences the MRI signal intensity.
- It is the effect of proton relaxation that appears on MRI and not the contrast itself

- Gadolinium is a paramagnetic agent responsible for T1 shortening of MR images
- Shortening of T1 leads to higher signal intensity on a T1WI hence areas of gad accumulation appear bright on T1
- Though it also shortens T2, effect is less as compared to T1
- Standard dose of Gad is 0.1 mmol/kg

# Normal contrast enhancing structures on MRI brain

- Pitutary stalk
- Median eminence
- Dural sinuses and cortical veins
- choroid plexus

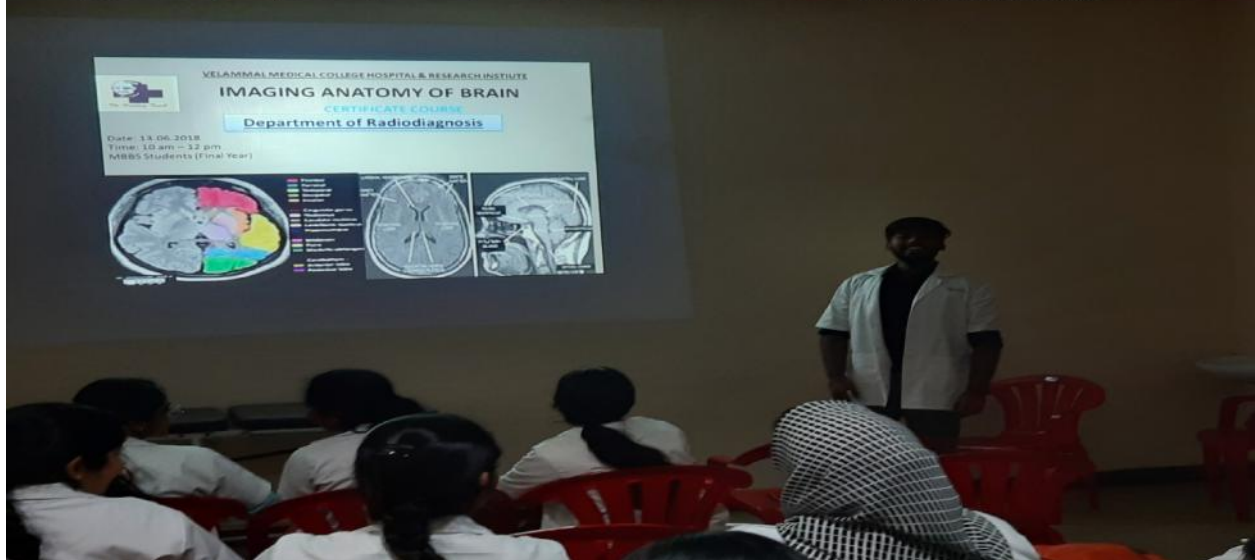
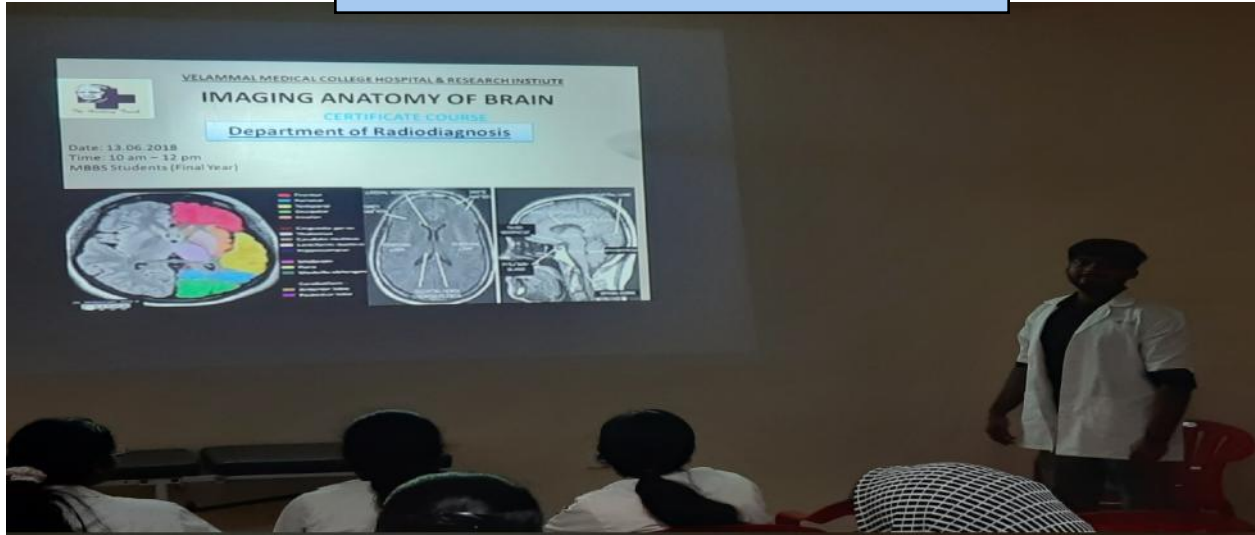
# Advantages of MRI

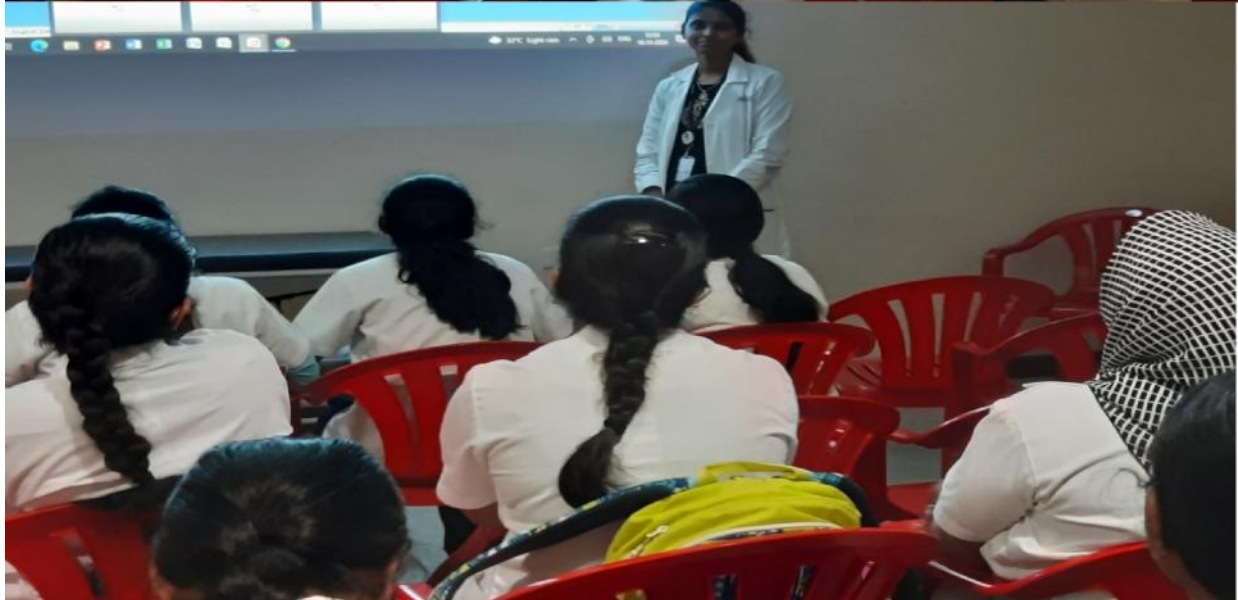
- No ionizing radiation & no short/long-term effects demonstrated
- Variable thickness, any plane
- Better contrast resolution & tissue discrimination
- Various sequences to play with to characterize the abnormal tissue
- Many details without I.V contrast
- No allergy ( as with Iodine)
- Can be used in renal impairment
- Pregnancy is not a contraindication

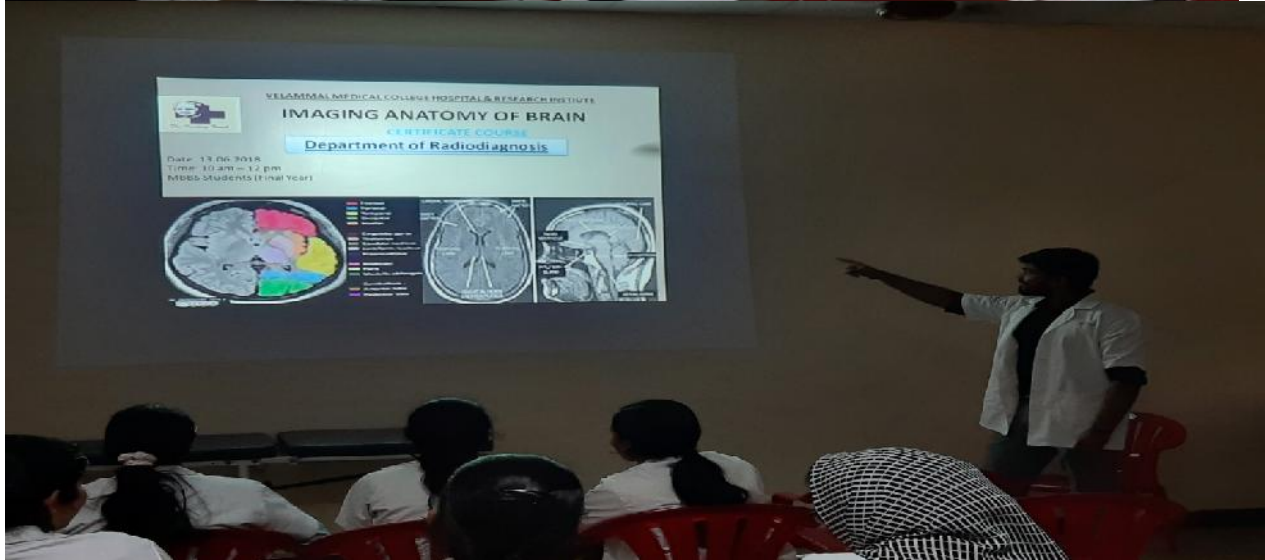
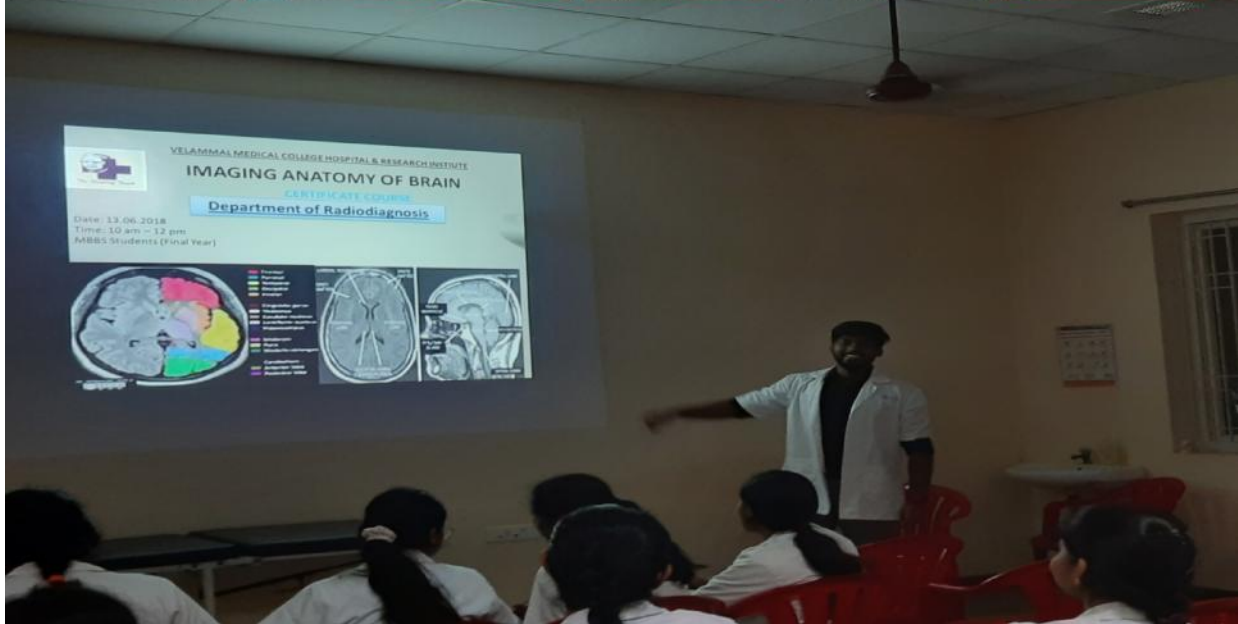
# Caveats of MR imaging

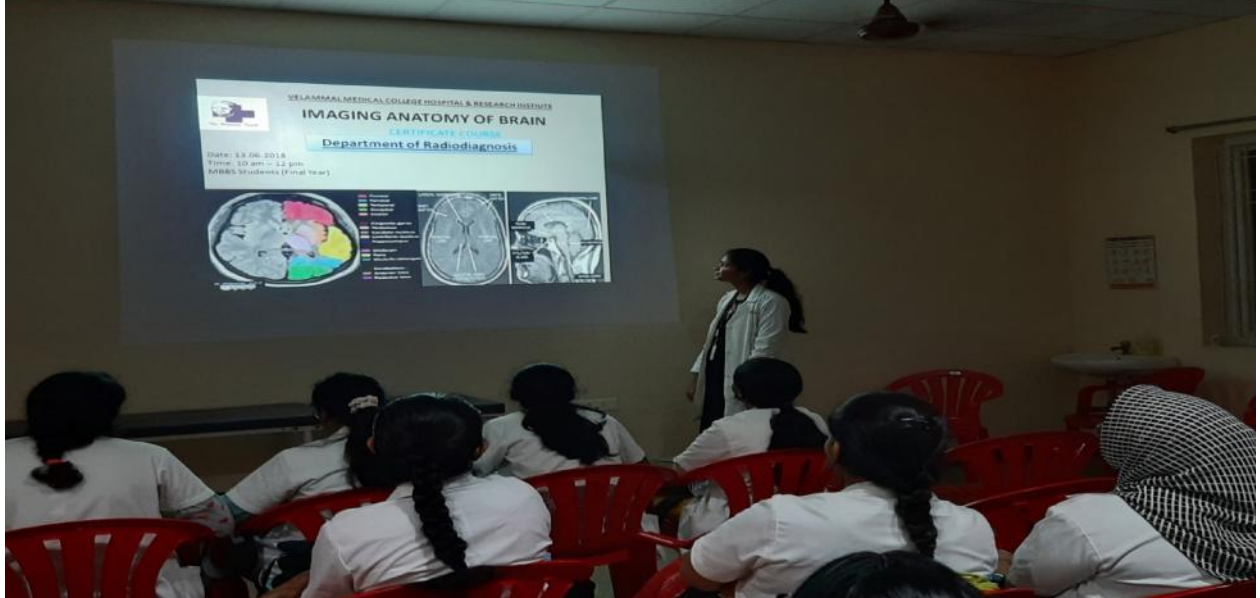
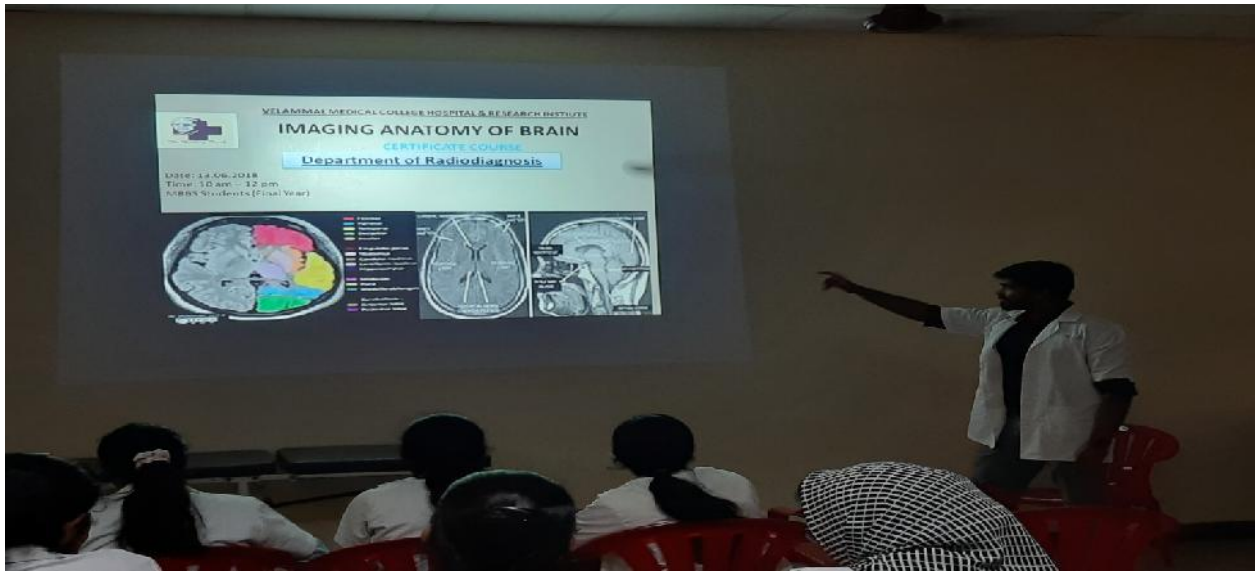
- Very sensitive to body movements
- Produces lots of noise during examination (The noise is due to the rising electrical current in the wires of the gradient magnets being opposed by the main magnetic field. The stronger the main field, the louder the gradient noise)
- Time taking
- Difficult to perform in claustrophobic pts
- Expensive
- Less sensitive for SAH
- Less sensitive for detection of calcification
- Relatively insensitive to bony cortical abnormalities
- Peoples with metallic implants can not be scanned

# PHOTOS











VELAMMAL MEDICAL COLLEGE  
HOSPITAL & RESEARCH INSTITUTE

**DEPARTMENT OF RADIODIAGNOSIS**

**STROKE IMAGING**

**CERTIFICATE COURSE**

**VELAMMAL MEDICAL COLLEGE HOSPITAL & RESEARCH INSTITUTE**



# STROKE IMAGING

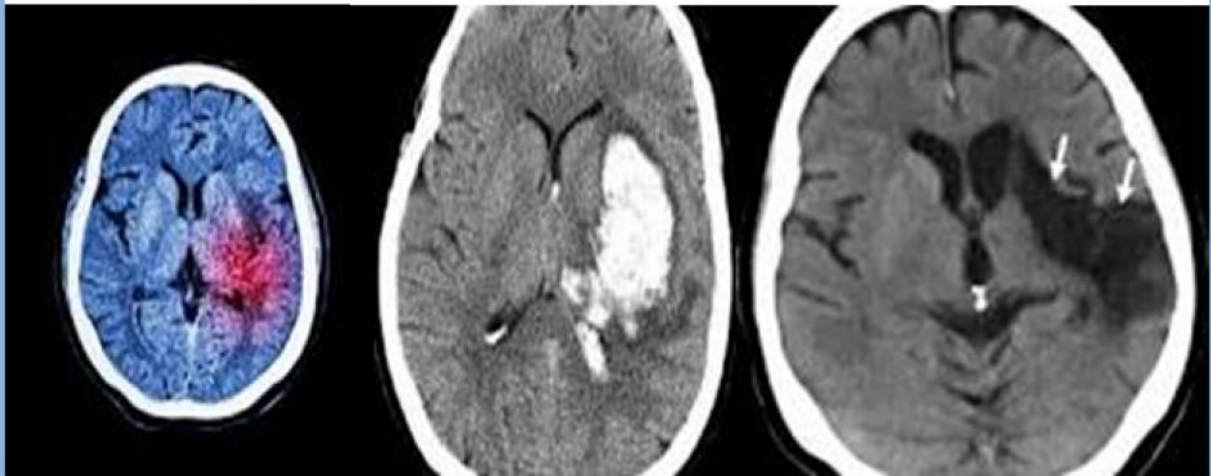
**CERTIFICATE COURSE**

**Department of Radiodiagnosis**

Date: 12.09.2018

Time: 10 am – 12 pm

MBBS Students (Final Year)





# **IMAGING IN STROKE**

# STROKE

- Stroke is an acute central nervous system injury with abrupt onset.
- This can occur following ischemia caused by blockage (thrombosis, arterial embolism) or a hemorrhage or tumors.
- Approximately 80% of all strokes are due to acute ischemia .

# **ETIOLOGY**

**Non vascular (5 %)**

- **Tumour, Hypoxia**

**Vascular (95 %)**

- **Ischemic Stroke**

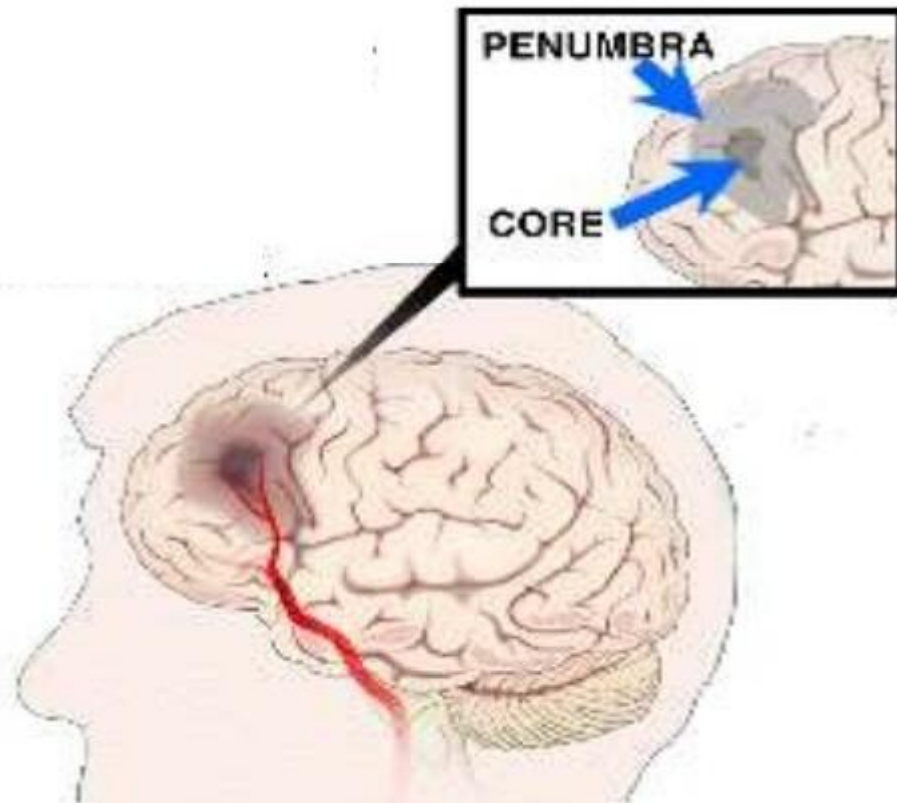
Thrombotic

Embolic

# PATHOPHYSIOLOGY

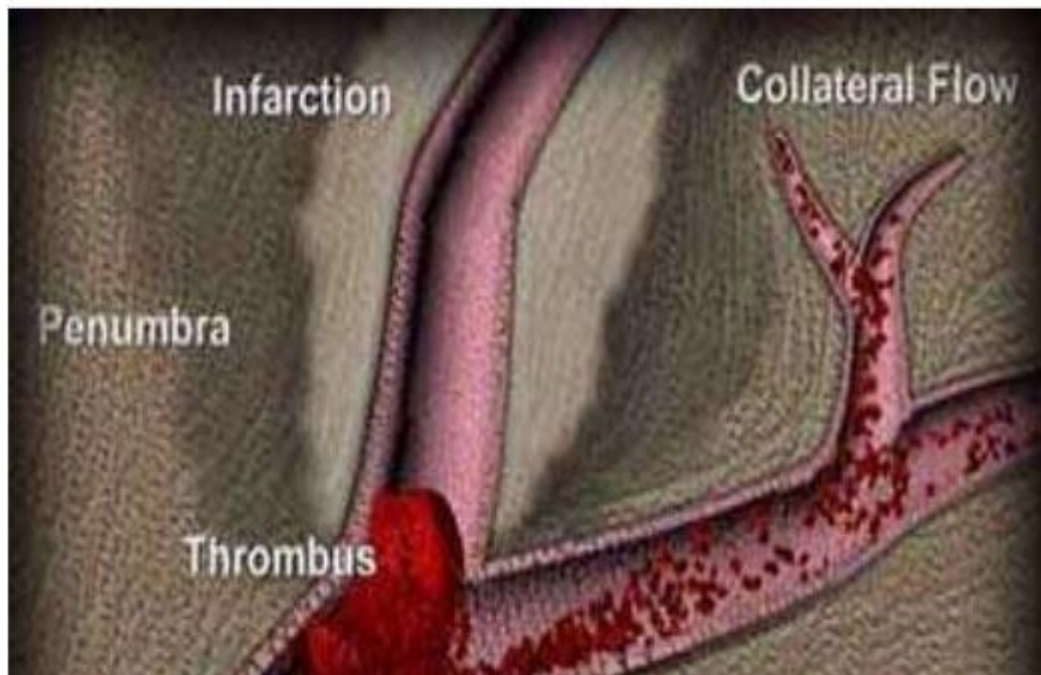
- Stroke progress in stages from ischemia to actual infarction.
- In artery occlusion, there is densely ischemic central focus & less densely ischemic “penumbra”.
- Cells within the densely ischemic area are usually damaged irretrievably unless reperfusion is quickly established.

# PENUMBRA



Penumbra is zone of reversible ischemia around core of irreversible infarction  
-Salvageable in first few hours after ischemic stroke

# PENUMBRA



Brain cells within the penumbra, a rim of mild to moderately ischemic tissue lying between tissue that is normally perfused and the area in which infarction is evolving, may remain viable for several hours. That is because the penumbral zone is supplied with blood by collateral arteries

- Ischemia produces energy depletion in the affected cells.
- Loss of ion homeostasis, accumulation of  $\text{Ca}^{++}$ ,  $\text{Na}^+$  &  $\text{Cl}^-$  along with osmotically obligated water & anaerobic glycolysis with production of intra & extracellular metabolic acidosis.
- Hypoxic-ischemic injury also leads to the accumulation of extracellular glutamate & free radicals.
- These changes are called ischemic cascade .a

- Secondary manifestation such as edema & mass effect eventually ensue.
- Imaging manifestations of cerebral ischemia vary significantly with time.
- Four distinct temporal phases can be identified,
  - Hyperacute
  - Acute
  - Subacute
  - Chronic

# **GOALS OF IMAGING**

- **To establish the diagnosis as early as possible.**
- **Give accurate information about intracranial vasculature and brain perfusion for guidance in selecting the appropriate therapy.**

# Imaging should target assessment of 4 P's:

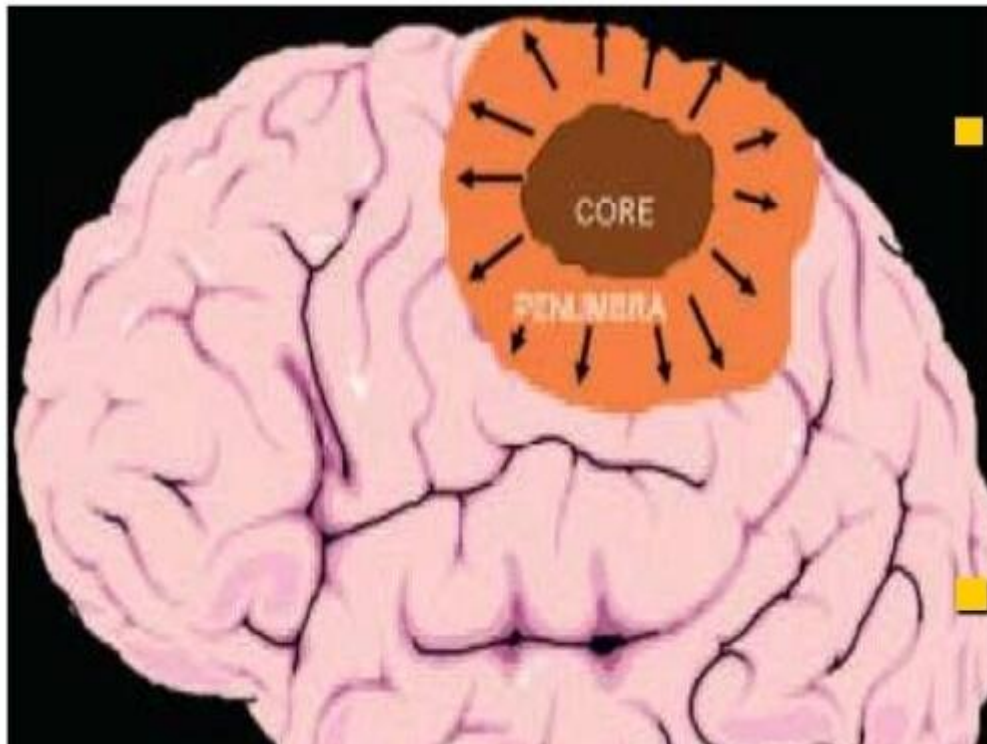
- **Parenchyma:**
  - Assess early signs of acute stroke, rule out hemorrhage
- **Pipes**
  - Assess extracranial circulation (carotid and vertebral arteries of the neck) and intracranial circulation for evidence of intravascular thrombus
- **Perfusion**
  - Assess cerebral blood volume, cerebral blood flow, and mean transit time

# OVERVIEW OF IMAGING MODALITIES

- **Unenhanced CT**
- **Can be performed quickly.**
- **Can help identify early signs of stroke, and can help rule out hemorrhage.**
- **CT angiography can depict intravascular thrombus**
- **CT perfusion imaging can demonstrate**

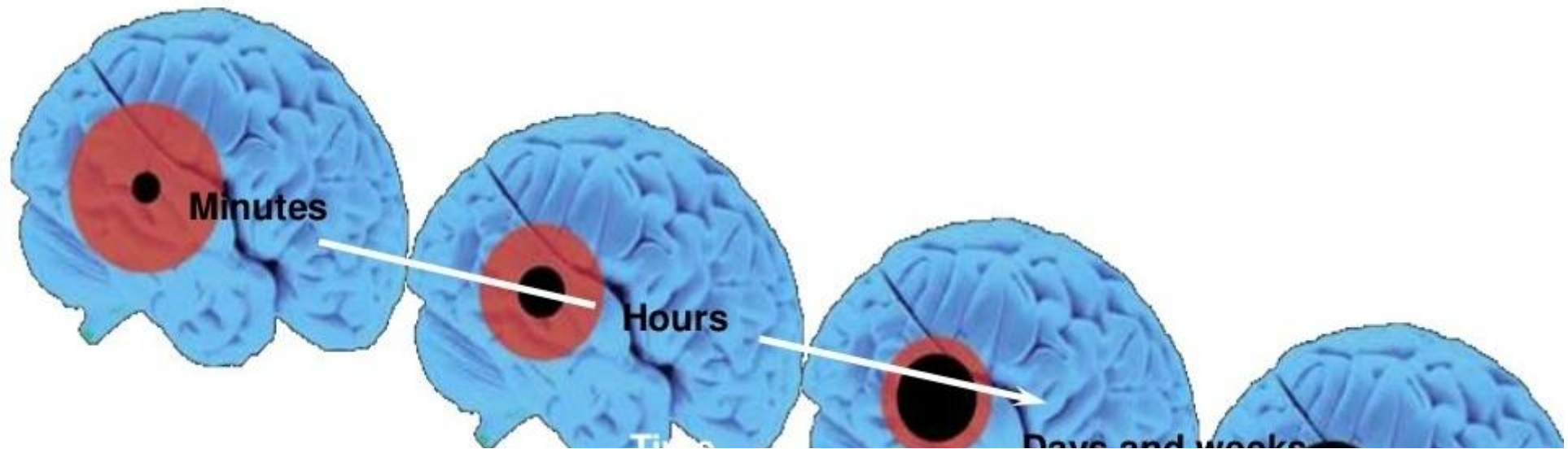
- **Acute infarcts may be seen early on conventional MR images, but diffusion-weighted MR imaging is more sensitive for detection of hyperacute ischemia.**
- **Gradient-echo MR sequences can be helpful for detecting a hemorrhage.**
- **MR Angiography – To evaluate the status of neck and intracranial vessels**

# ACUTE STROKE



- Acute cerebral ischemia may result in a central irreversibly infarcted tissue core surrounded by a peripheral region of stunned cells that is called as a penumbra
- This region is potentially salvageable with early

The transition from ischemia to irreversible infarction depends on both the severity and the duration of the diminution of blood flow



# **ISCHEMIC PENUMBRA**

- **IDENTIFIED BY**
  - **CT - ALTERED PARAMETERS IN PERFUSION**
  - **MR – PERFUSION /DIFFUSION MISMATCH**
  
- **PRESENCE OF PENUMBRA HAS SIGNIFICANT IMPLICATIONS IN PT MANAGEMENT**

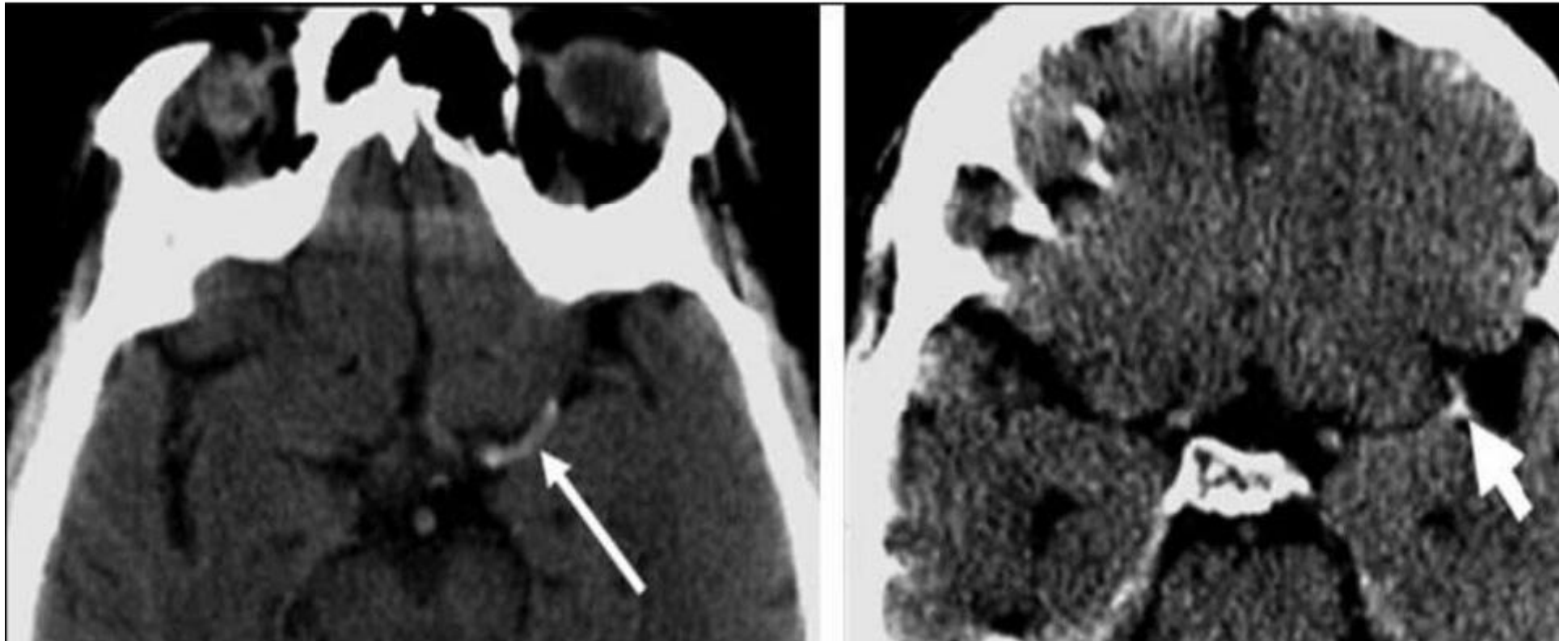
# NECT

- **Widely available.**
  - **Can be done quickly.**
  - **It not only can help identify a hemorrhage (a contraindication to thrombolytic therapy), but it also can help detect early-stage acute ischemia by depicting features such as –**
-

# **HYPERDENSE VESSEL SIGN**

- Acute thrombus has high attenuation value this feature is referred to as the hyperdense vessel sign.
- Highly specific but sensitivity is poor.
- FALSE POSITIVE
  - HIGH HEMATOGRIT LEVEL
  - MCA CALCIFICATION

# HYPERDENSE MCA



# **OBSCURATION OF LENTIFORM NUCLEUS**

- Lentiform nucleus appears hypoattenuated because of acute ischemia of the lenticulostriate territory , resulting in obscuration of the lentiform nucleus.
- This feature may be seen on CT images within 2 hours after the onset of a stroke .

## OBSCURATION OF LENTIFORM NUCLEOUS



# INSULAR RIBBON SIGN

- It is the local hypoattenuation of the insular cortex region due to cytotoxic edema as this region is susceptible to early and irreversible ischemic damage.

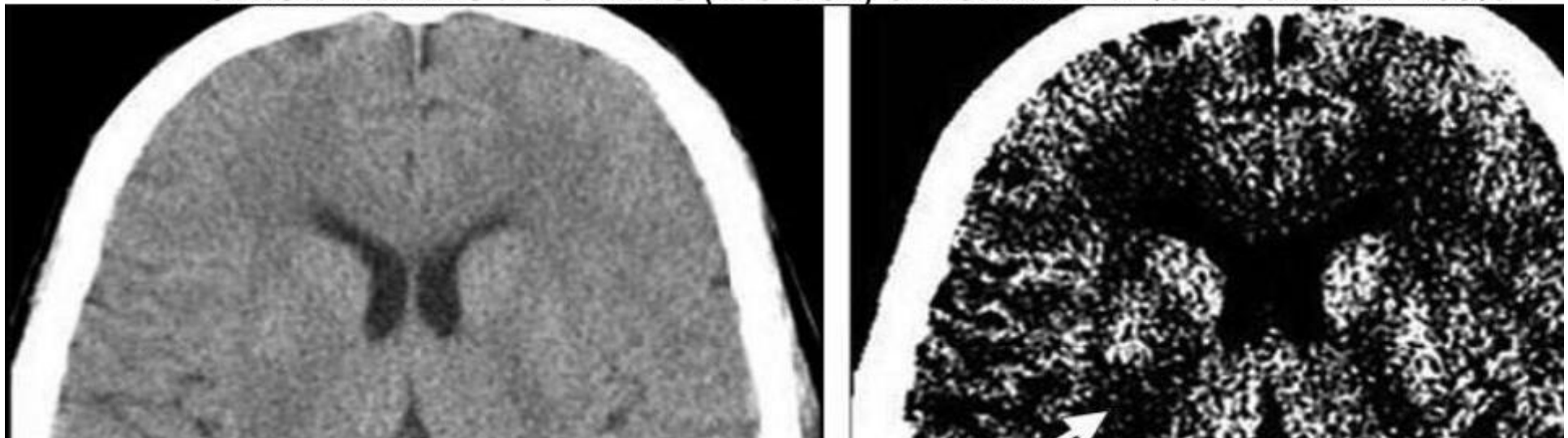
# INSULAR RIBBON



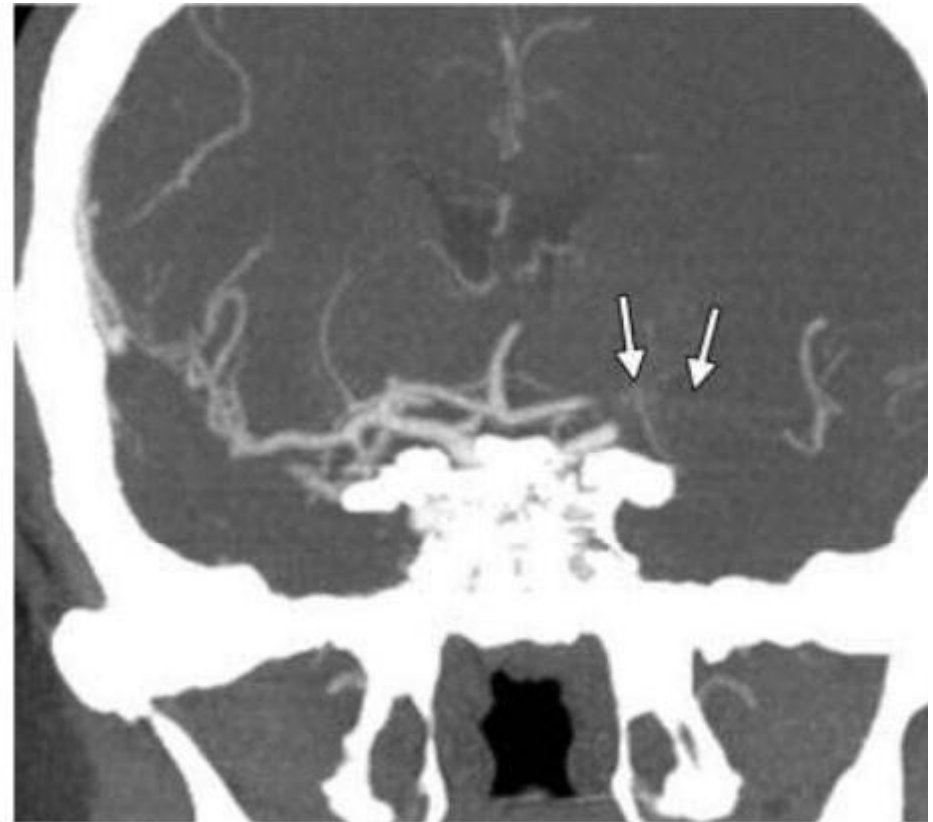
**Axial unenhanced CT image, obtained in a 73-year-old woman 2½ hour after the onset of left hemiparesis, shows hypoattenuation and obscuration of the posterior part of the right lentiform nucleus (white arrow) and a loss of gray matter–white matter definition in the lateral**

# WINDOW SETTING

- Detection of early acute ischemic stroke on unenhanced CT images may be improved by using variable window width and center level settings to accentuate the contrast between normal and edematous tissue
- STANDARD WINDOW SETTING (W80 C 20) – SENSITIVITY 57% SPECIFICITY 100%
- STROKE WINDOW SETTING (W8 C 32) SENSITIVITY 71% SPECIFICITY 100%



# CT ANGIOGRAPHY

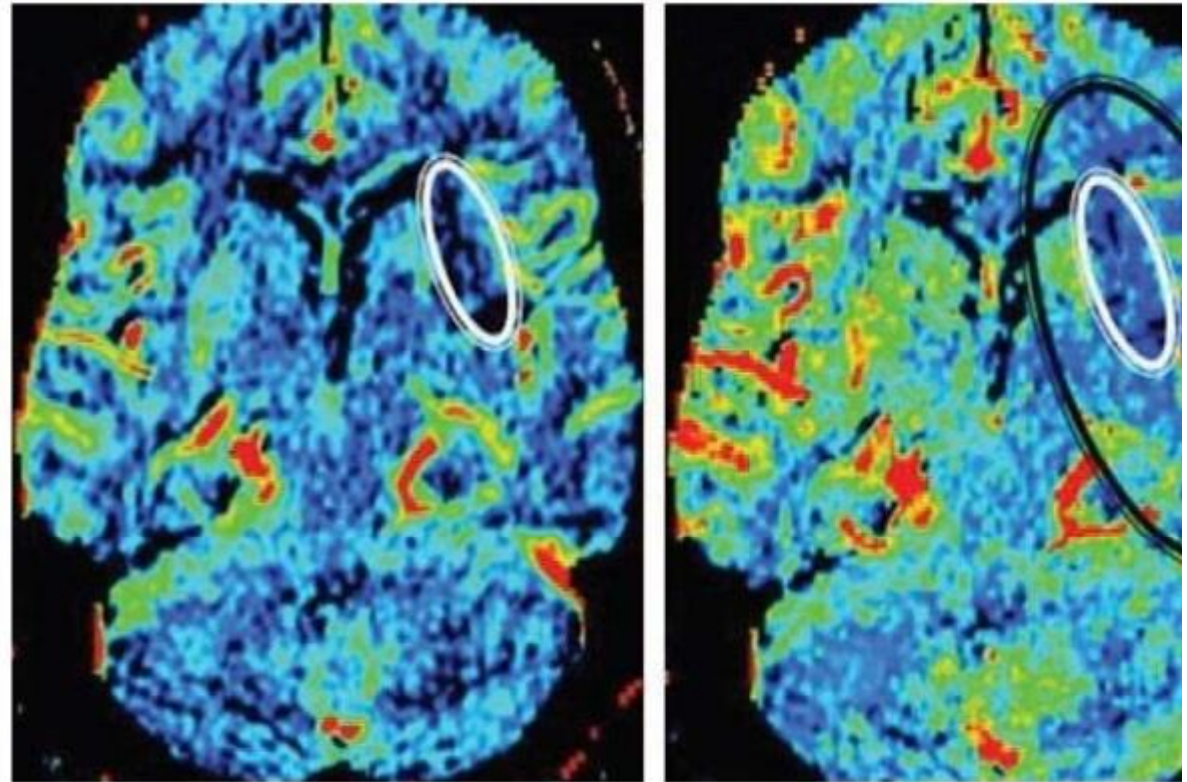


CT angiography typically involves a volumetric helical acquisition that extends from the aortic arch to the circle of Willis.

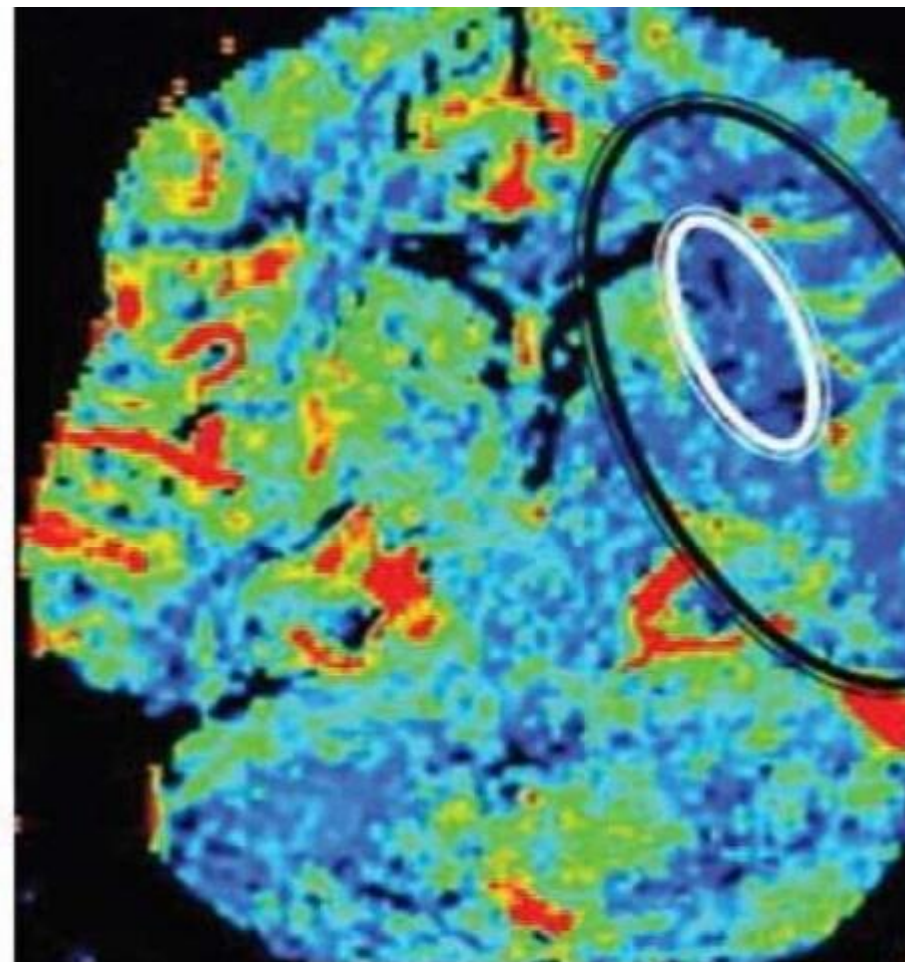
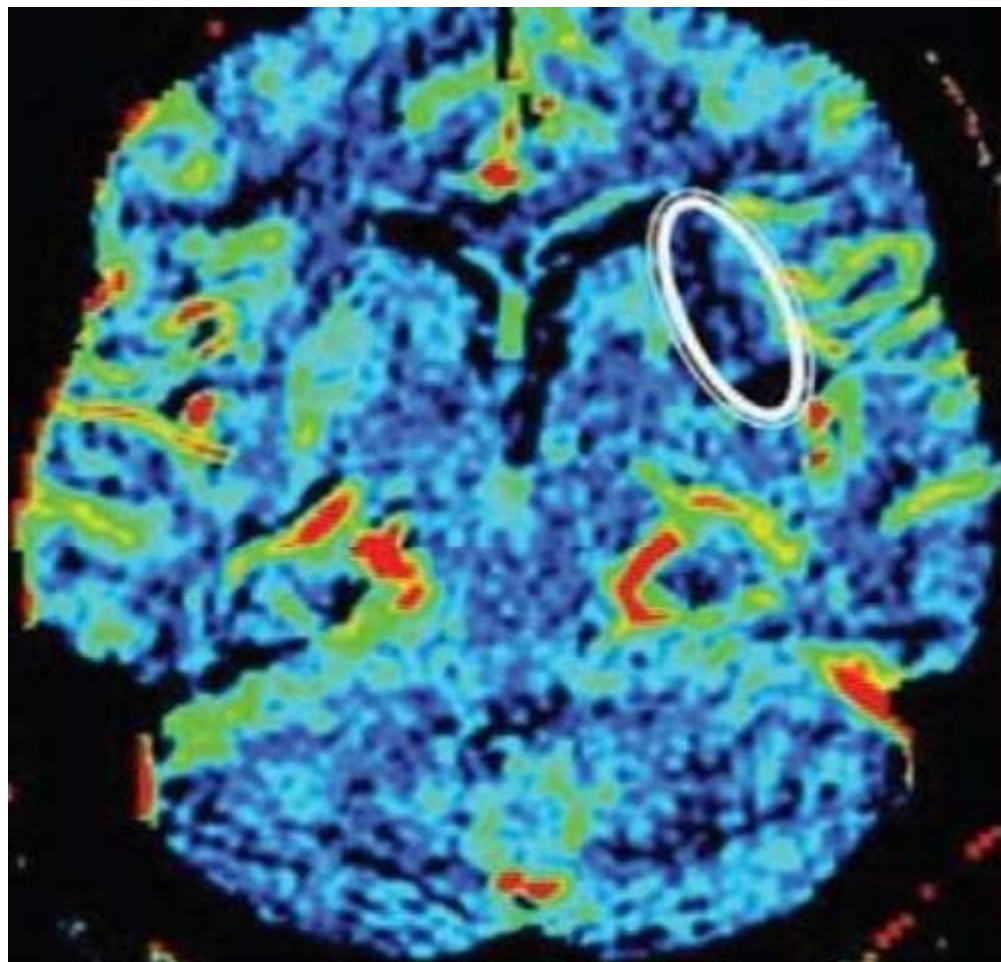
- The examination is performed by using a time-optimized bolus of contrast material for vessel enhancement.



## **CT PERFUSION (CTP):**



- **With CT and MR-diffusion we can get a good impression of the area that is infarcted.**
- **But, we cannot preclude a large ischemic penumbra (tissue**



**CT perfusion maps of cerebral blood volume (a) and cerebral blood flow (b) show, in the left hemisphere, a region of decrease**

# CT PERFUSION

- **PARAMETERS ASSESSED**
  - **CBV – VOLUME OF BLOOD PER UNIT OF BRAIN TISSUE (N 4-5ML/100GM)**
  - **CBF – VOLUME OF BLOOD FLOW PER UNIT OF BRAIN TISSUE PER MINUTE (N 50-60ML/100GM/MINUTE)**
  - **MTT – TIME DIFFERENCE BETWEEN THE ARTERIAL INFLOW AND VENOUS OUTFLOW**

# INTERPRETATION OF PCT

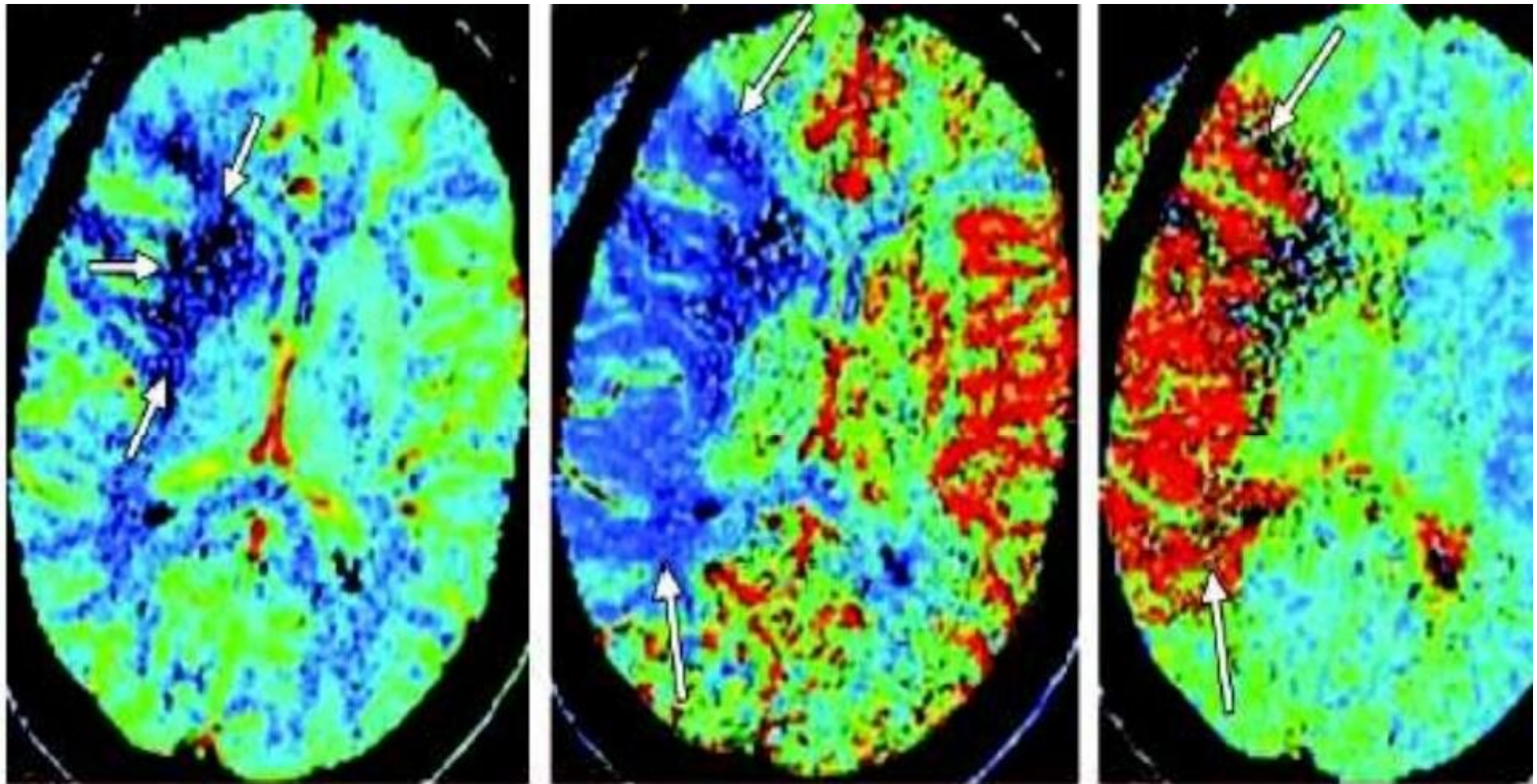
- **INFARCTED AREA**

- SEVERELY DECREASED CBF (<30%) AND CBV (<40%)
- PROLONGED MTT

- **PENUMBRA**

- INCREASED MTT
- MODERATELY DECREASED CBF (>60%)
- INCREASED CBV (80-100% OR HIGHER)

**OR**



Acute stroke in a 65-year-old man with left hemiparesis. CT perfusion

# **CURRENT MR IMAGING STRATEGIES**

- Conventional MRI
- Diffusion Imaging
- Perfusion Imaging

# CONVENTIONAL MRI

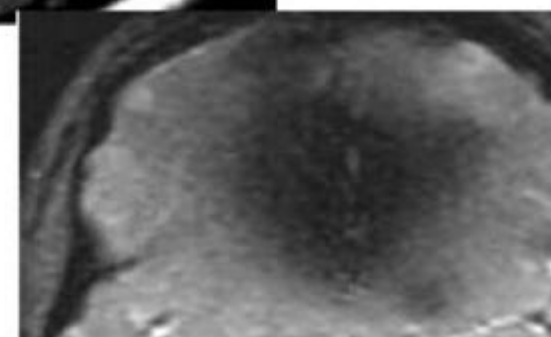
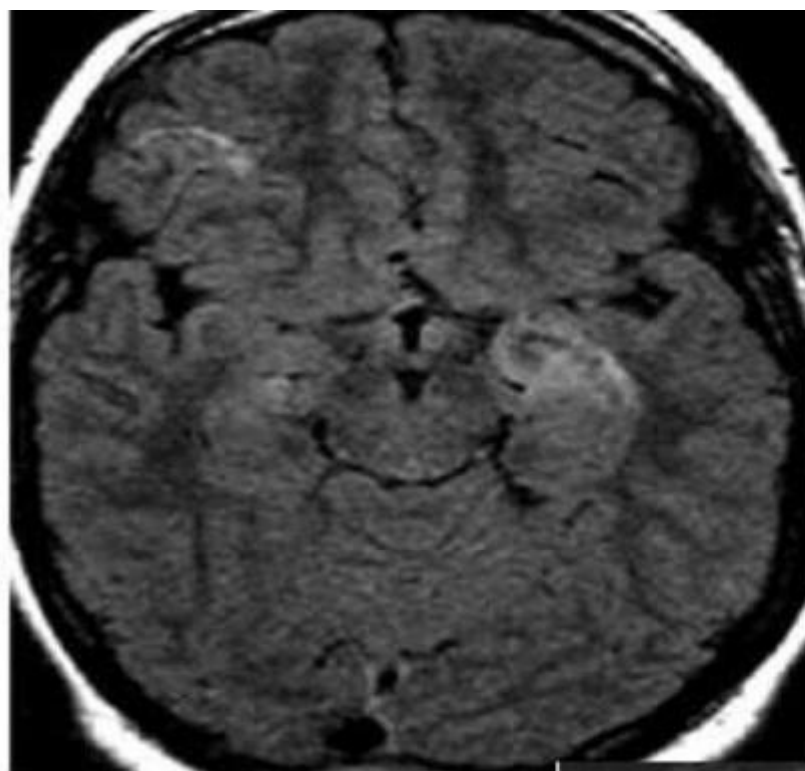
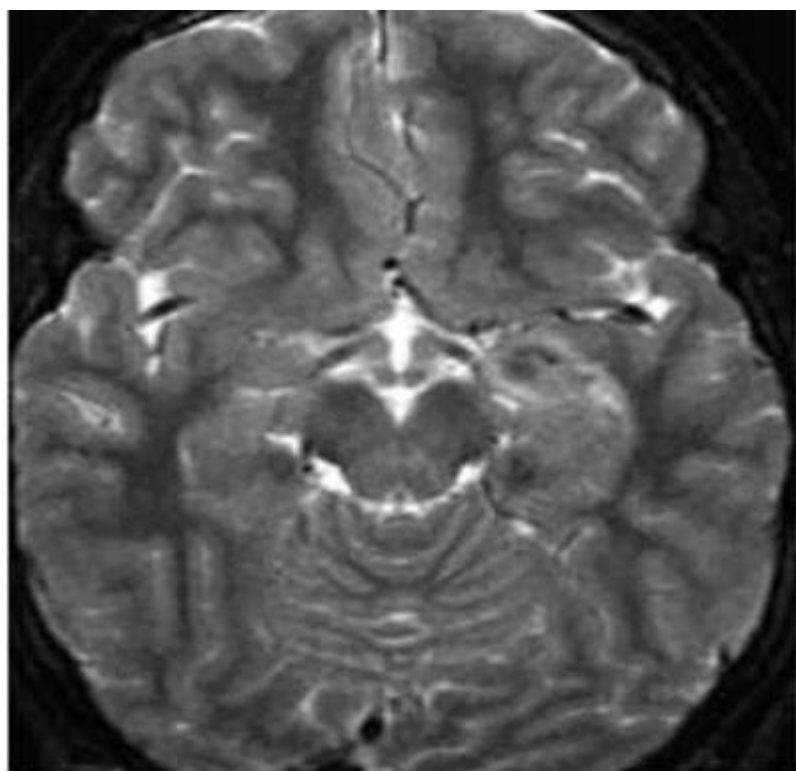
- **SPIN ECHO IMAGES MORE SENSITIVE AND SPECIFIC THAN CT IN ACUTE CVA**
- **SEQUENCES**
  - T1
  - T2
  - FLAIR
  - GRE

# **ACUTE CVA**

- **HYPER ON T2 AND FLAIR**
- **LOSS OF GRAY WHITE MATTER DIFFERENTIATION**
- **SULCAL EFFACEMENT**
- **MASS EFFECT**
- **LOSS OF FLOW VOID IN T2WI IN VESSEL**
- **BLOOMING IN GRE IF HRGE**

# **ROLE OF MRI IN STROKE**

- **To rule out cerebellar or brainstem infarct**
- **To detect hyperacute infarct and to differentiate acute vs. old infarct.**
- **To guide thrombolytic therapy**
- **In Pediatric stroke**



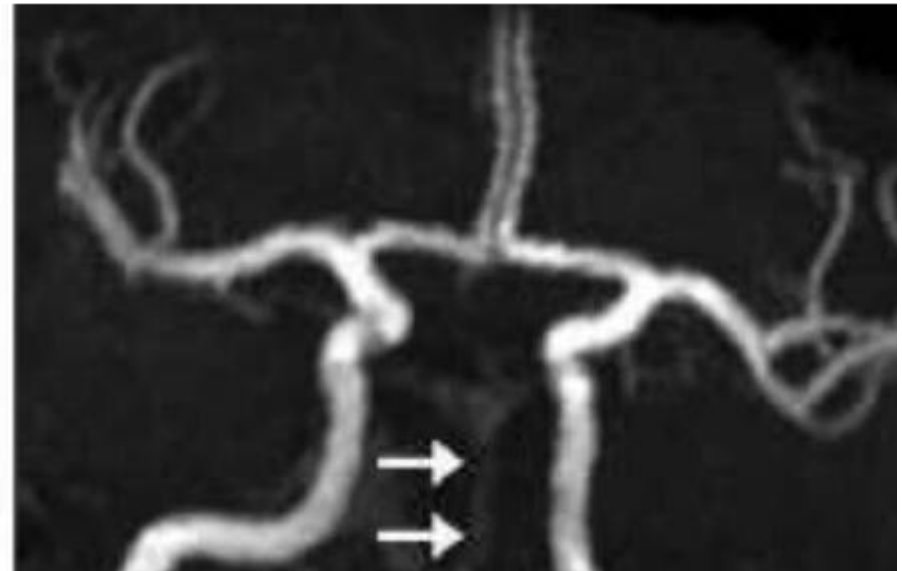
**Acute stroke in the left medial temporal lobe in a 44-year-old man.  
(a) Axial T2-weighted and (b) fluid attenuated**

# MR ANGIOGRAPHY

LEFT ICA THROMBUS

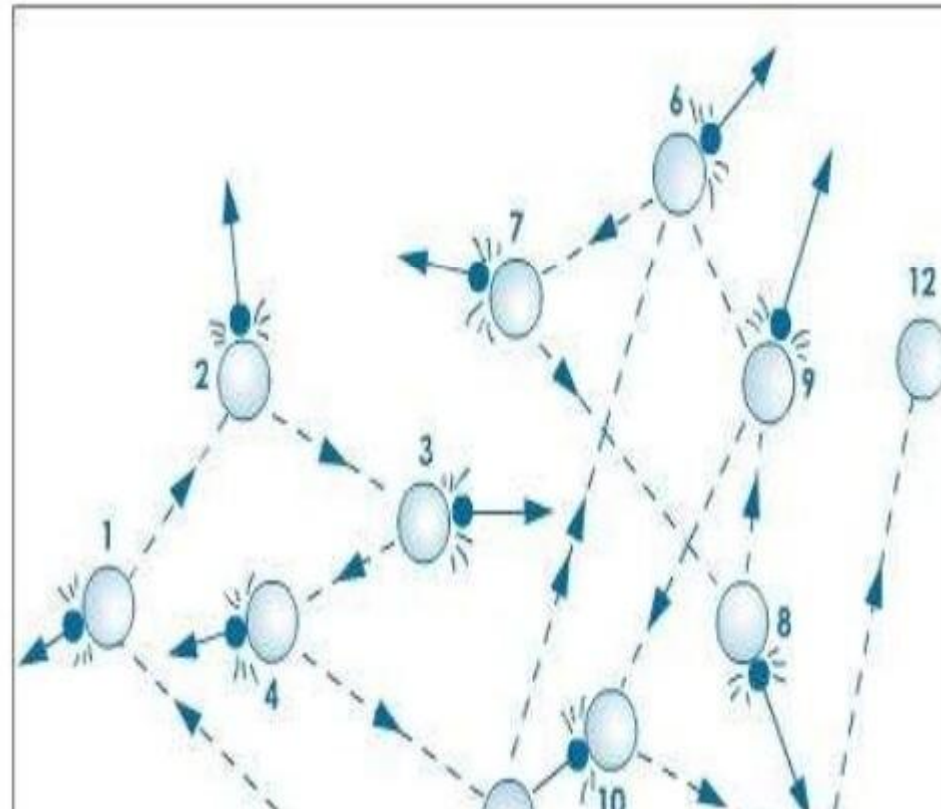


BASILAR ARTERY THROMBUS



# Diffusion-Weighted Imaging

- Brownian motion
- The normal motion of water molecules within living tissues is random.
- Acute stroke causes excess intracellular water accumulation or “cytotoxic edema”, with an overall decreased rate of water molecular



# **CLINICAL APP OF DWI**

- **Changes in DWI occur within 30min of onset of ischemia with corresponding reduction in ADC and seen up to 5 days**
- **Mild hyperintense DWI with pseudonormal ADC from 1 -4wks**
- **After several wks DWI signal varies (T2 effect) with increased ADC**

# OVERVIEW OF THEORY

- **Restricted diffusion hyperintense and conspicuous on DWI.**
- **On the ADC map, the area of restricted diffusion is dark.**
- **Use ADC map to exclude T2 shine**

# Diffusion-weighted MR

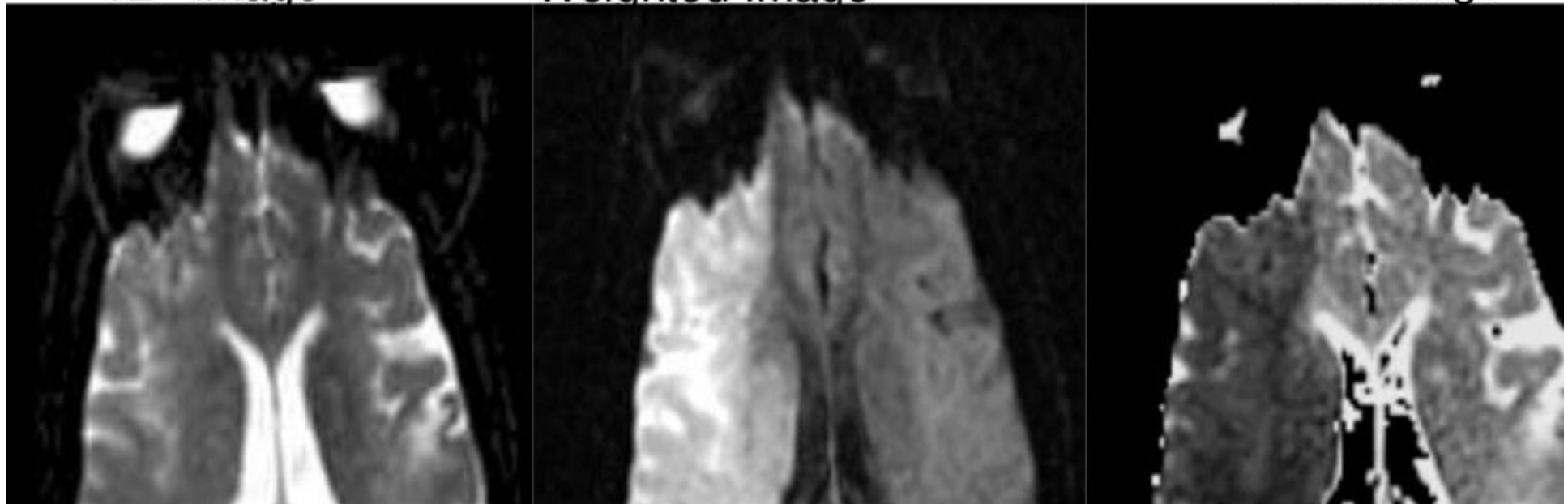
- **More sensitive for detection of hyperacute ischemia**
- **Becomes abnormal within 30 minutes**
- **Distinguish between old and new stroke**
  - **New stroke: Bright on DWI**
  - **Old stroke: Low on DWI**

# APPARENT DIFFUSION COEFFICIENT (ADC)

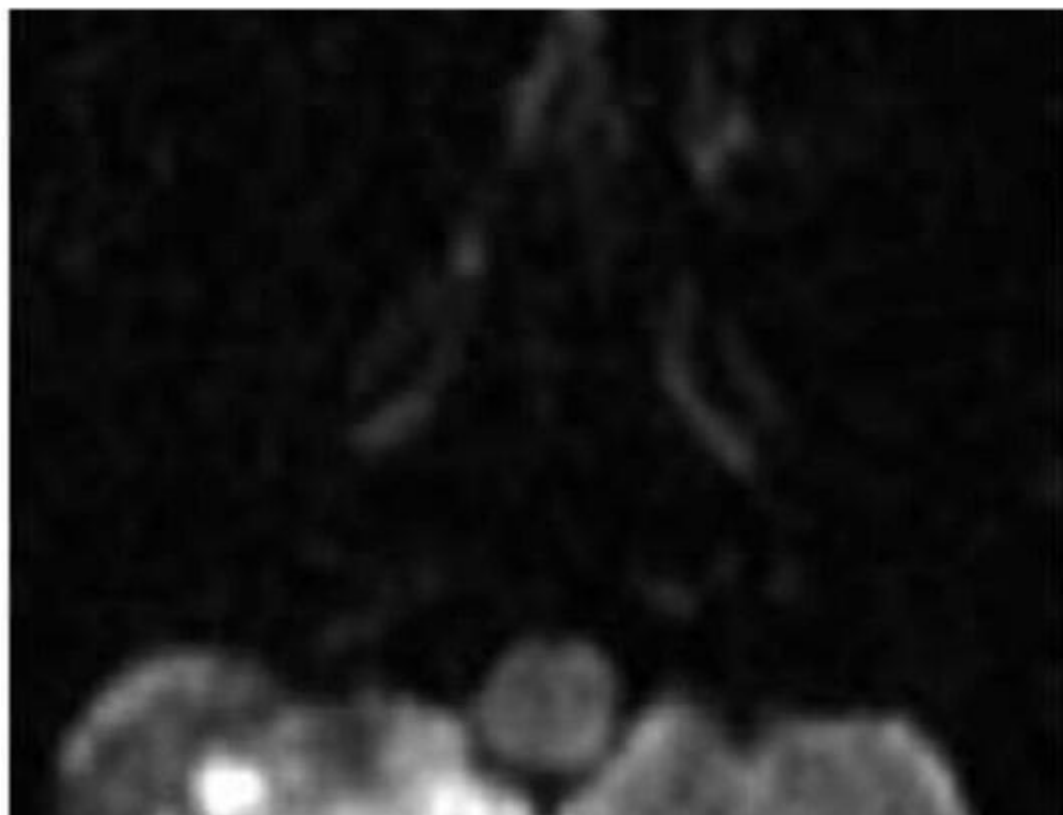
T2 - image

Isotropic Diffusion-  
Weighted Image

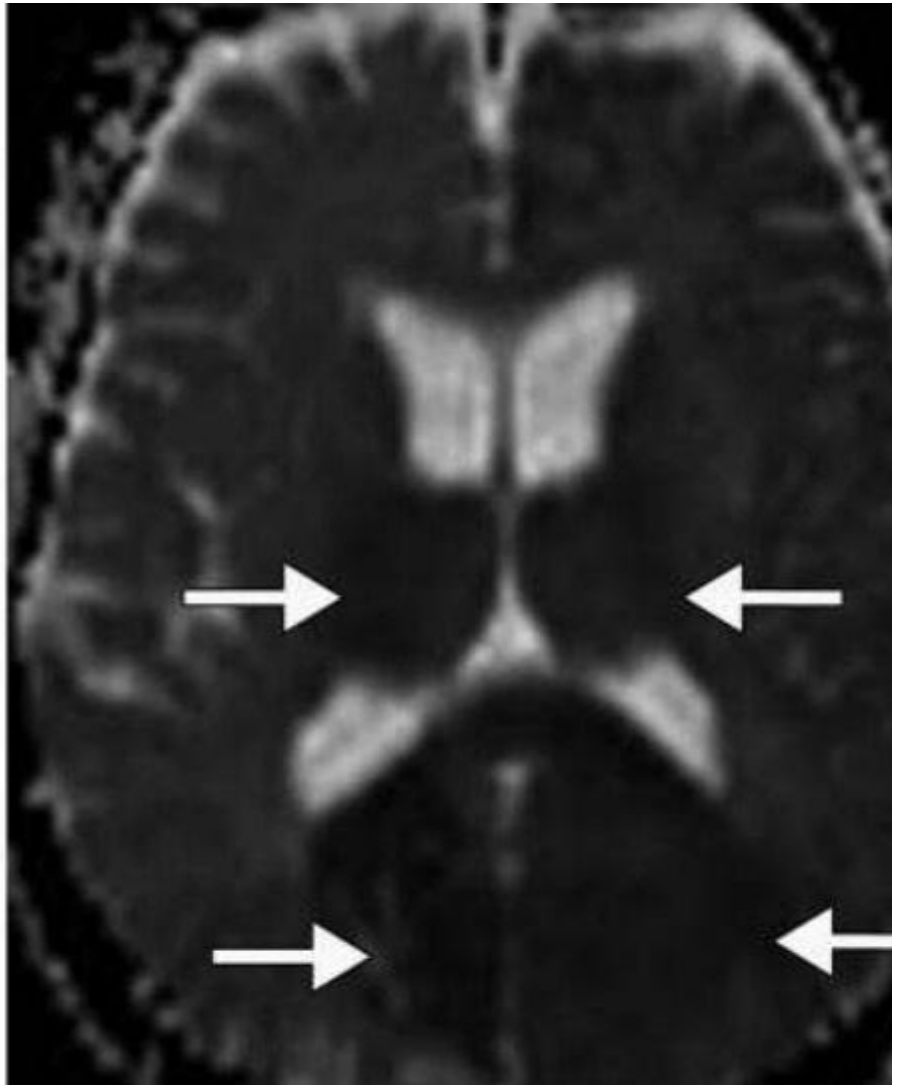
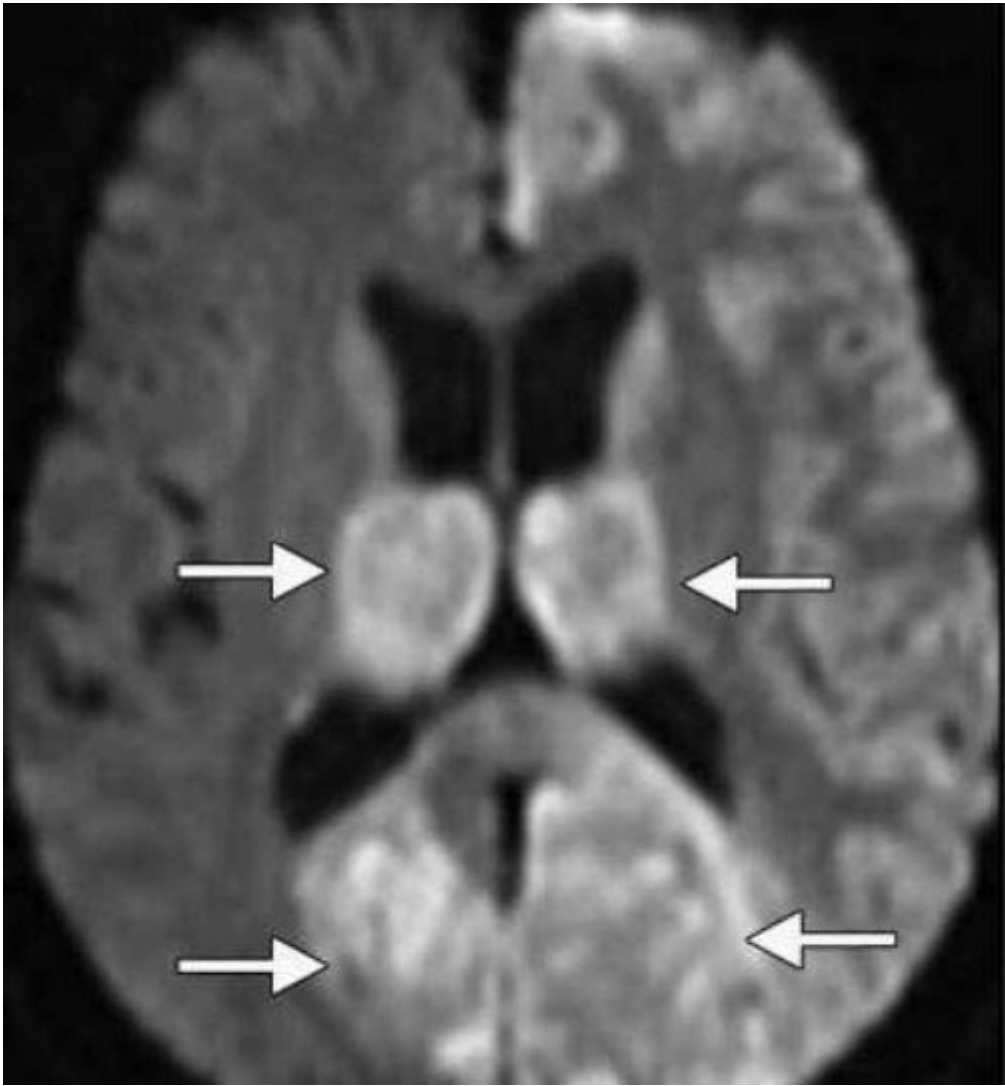
ADC image



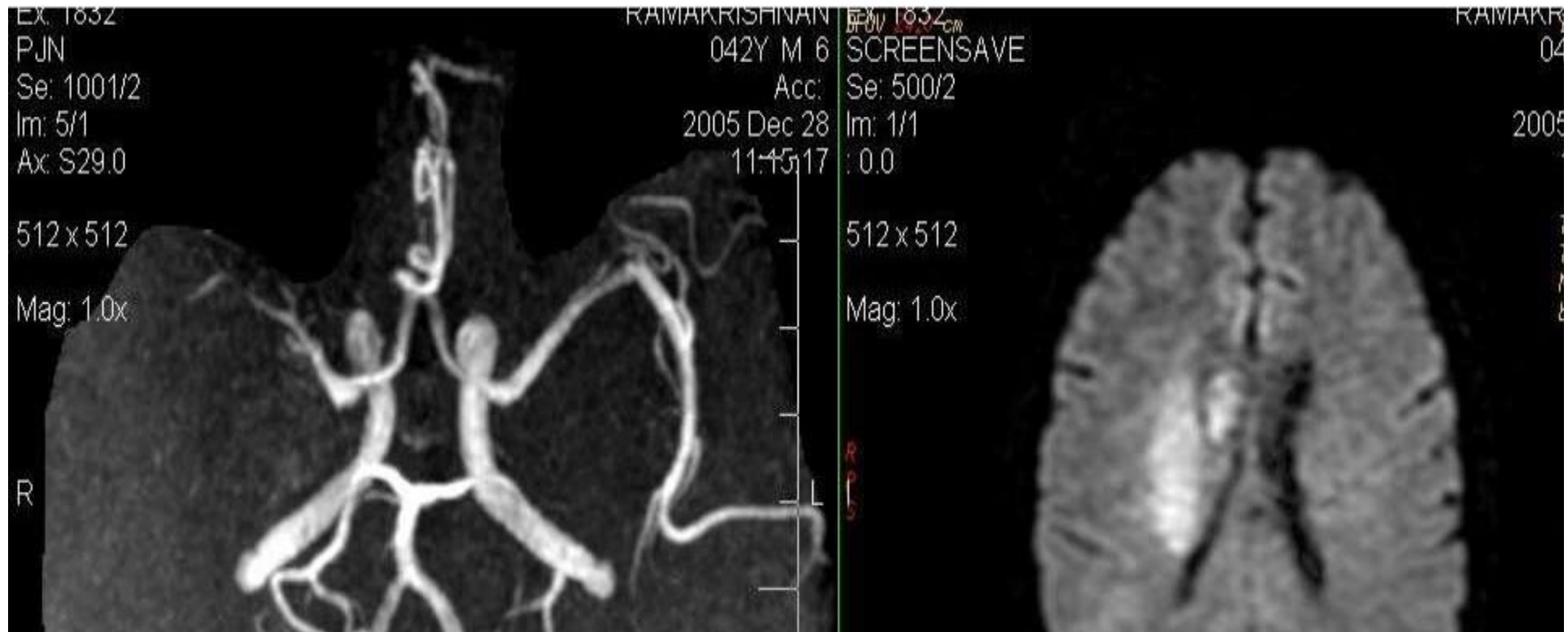
## DWI ACUTE CVA



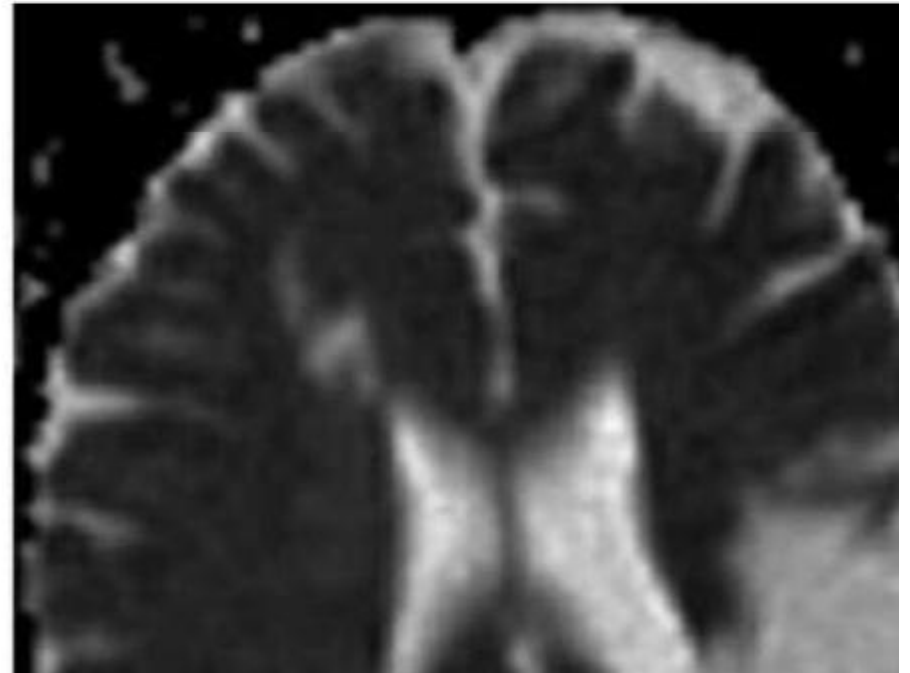
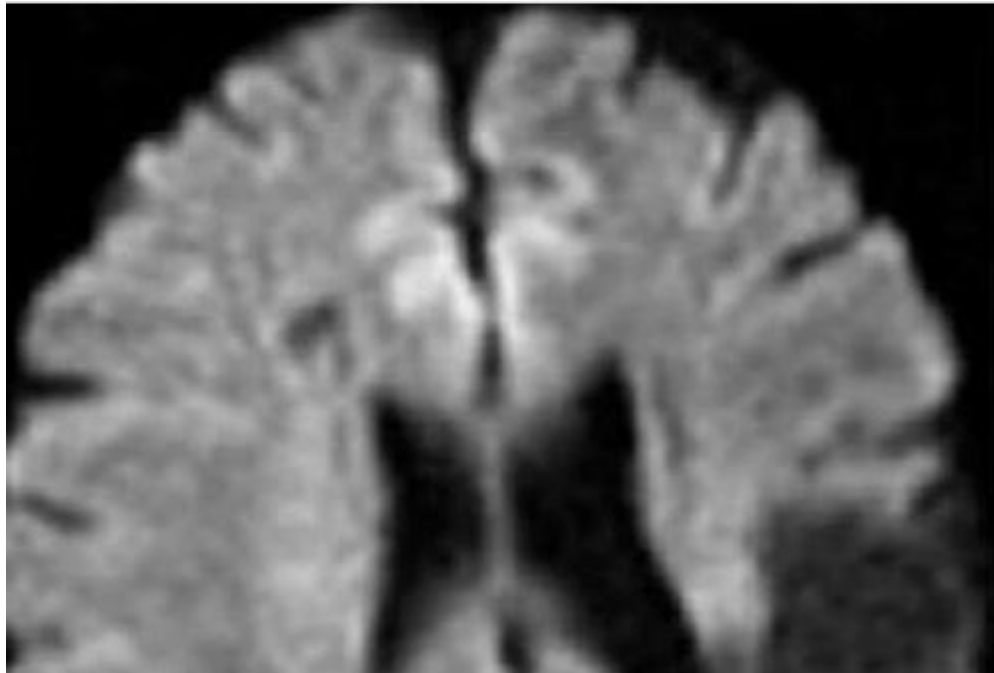
**Acute stroke–induced cytotoxic edema in the right cerebellar hemisphere. Diffusion-weighted MR image shows areas of signal intensity increase due to the restricted mobility of**



# ACUTE INFARCT



# CHRONIC INFARCT



# ACCURACY

- **CT/ CONVENTIONAL MRI**
  - SENSITIVITY AND SPECIFICITY < 50%
- **DWI**
  - SENSITIVITY 88-100%
  - SPECIFICITY 86-100%
- **FALSE -VE DWI**
  - LACUNAR INFARCTS OF BRAIN STEM
  - SMALL DEEP GREY MATTER INFARCTS

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# PERFUSION-WEIGHTED IMAGING

- **Allows the measurement of capillary perfusion of the brain**
- **Uses a MR contrast agent**
- **The contrast bolus passage causes a nonlinear signal decrease in proportion to the perfusion cerebral blood volume**
- **It can identify areas of hypoperfusion, the**

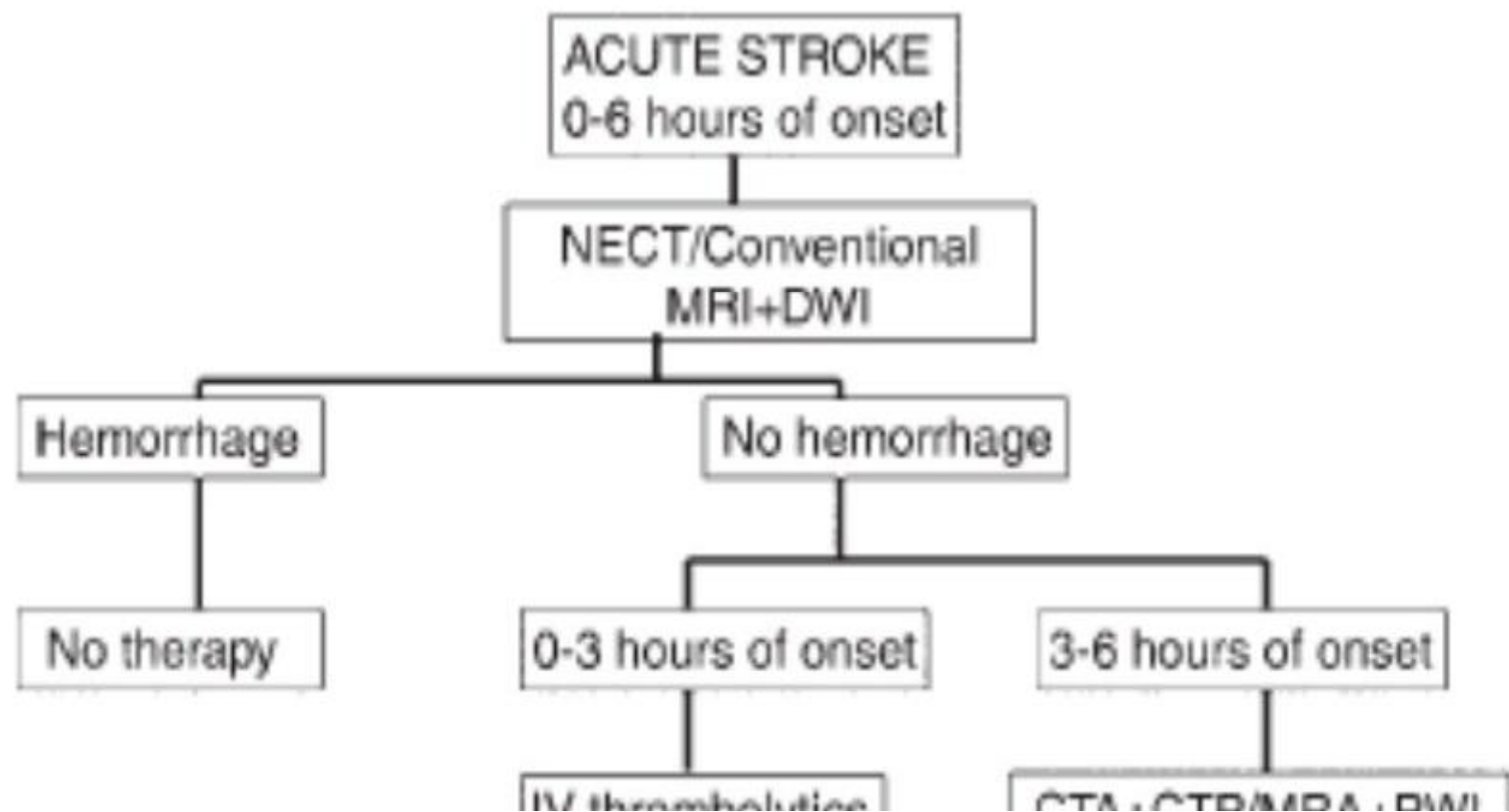
# **COMPARISON OF PWI AND DWI**

- **DWI → Depicts irreversibly damaged infarct**
- **PWI → Reflects the complete area of hypoperfusion**
- **The volume difference between these two, the PWI/DWI mismatch would be the PENUMBRA!**

## **SIGNIFICANCE OF PWI/DWI MISMATCH**

- **IV thrombolytic treatment is not typically administered to patients with acute stroke beyond 3-hrs period**
  - Risk of hemorrhage
- **However, recent studies have shown that IV thrombolytic therapy may benefit patients who are carefully selected according to**

# ACUTE CVA IMAGING PROTOCOL



# CLINICAL APPLICATION

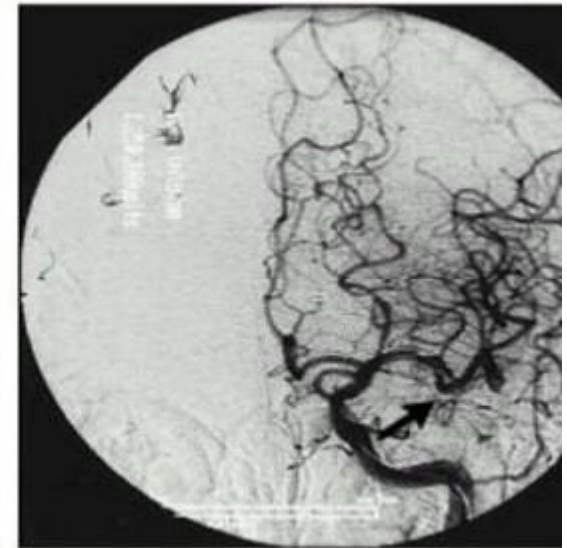
- **Unenhanced CT: rule out hemorrhage**
  - Not very good to detect ischemia
- **T1 or T2 weighted MRI**
  - Good for detecting ischemia
  - Cannot differentiate between acute versus chronic ischemia
- **DWI/PWI**

# INTRAARTERIAL TPA

2 Hours, 20 Minutes

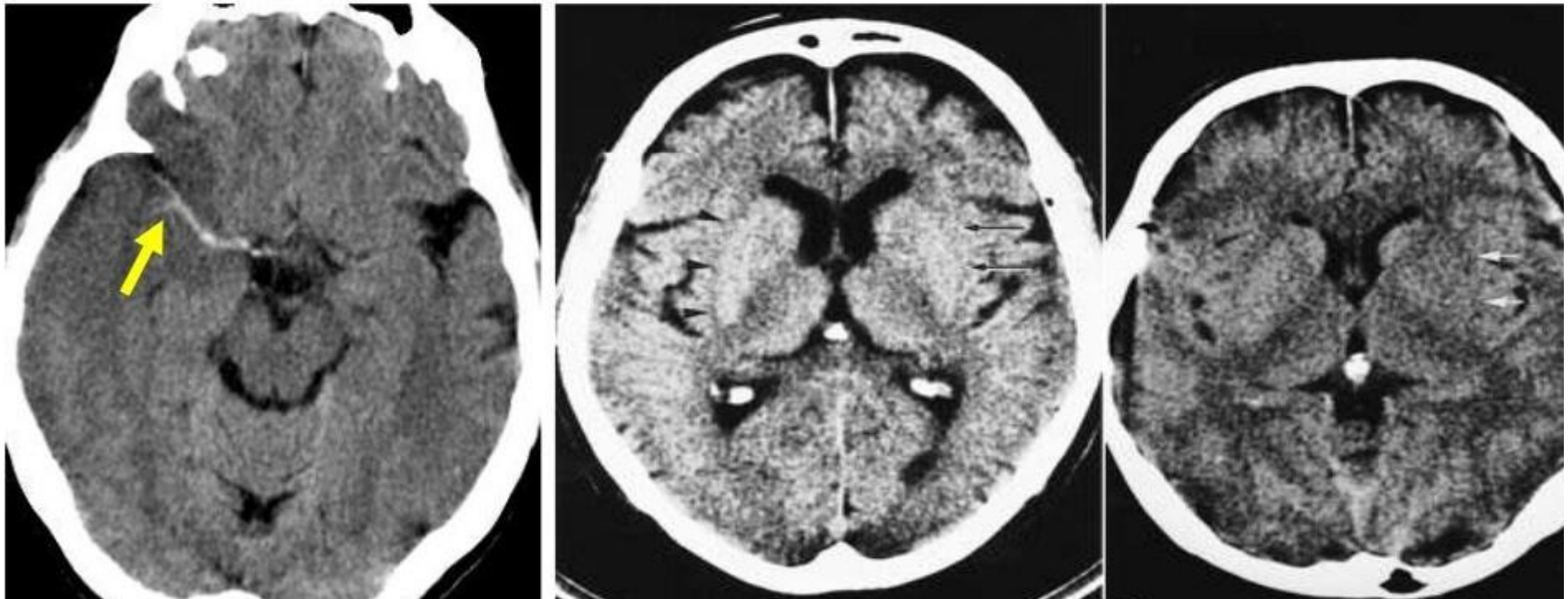


4 Hours, 20 Minutes

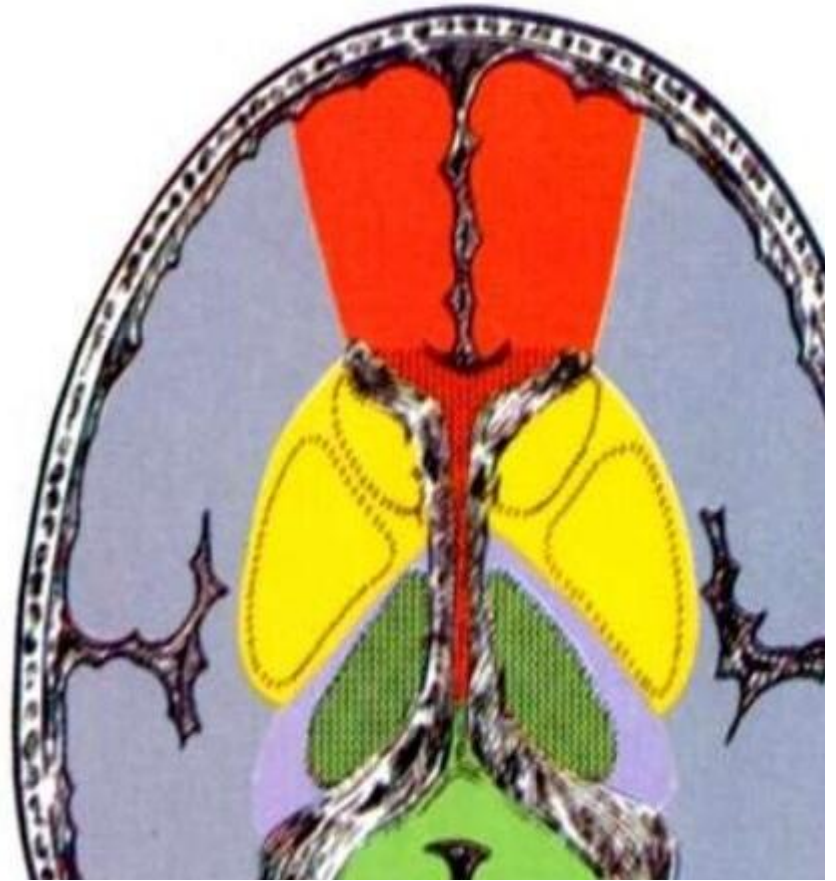
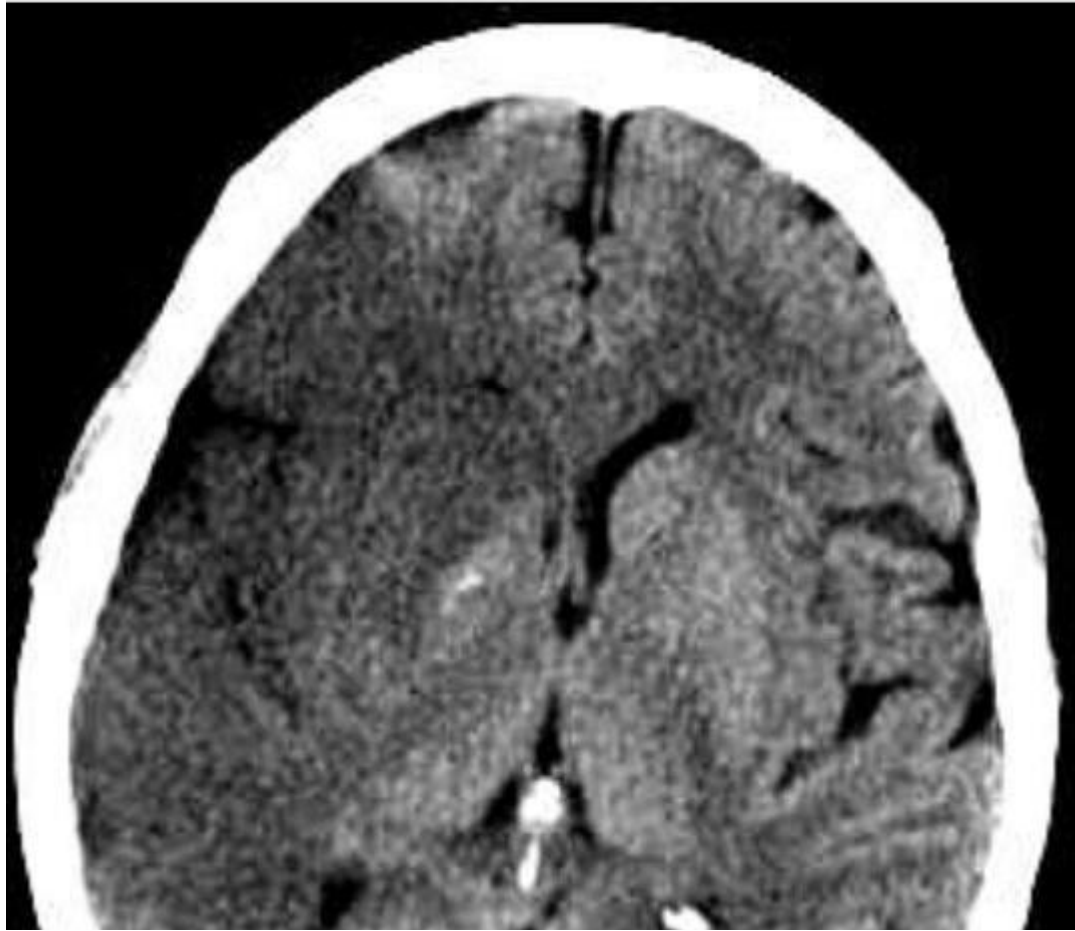


- IA-TPA in selected pts. In < 6 hours due to MCA & BA occlusion

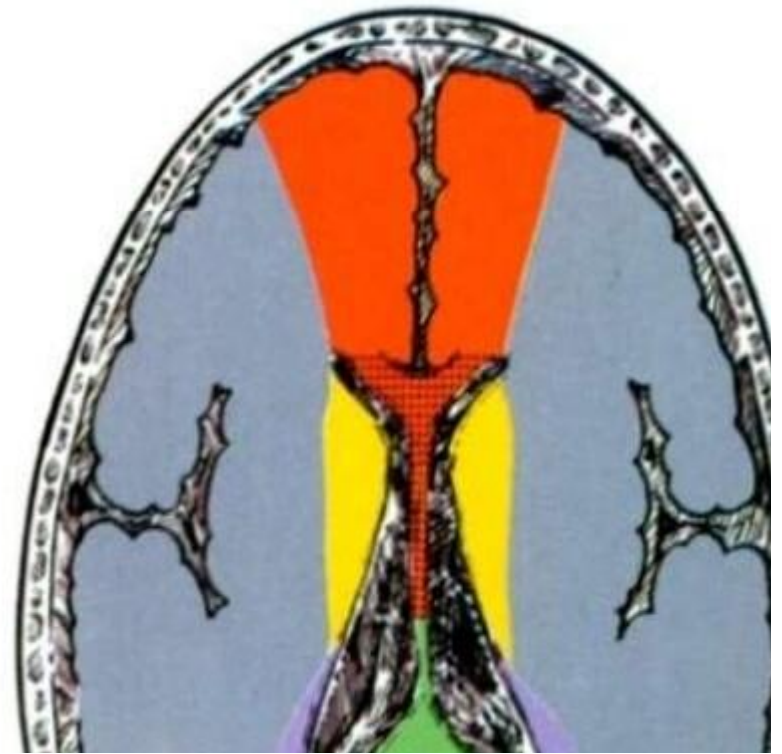
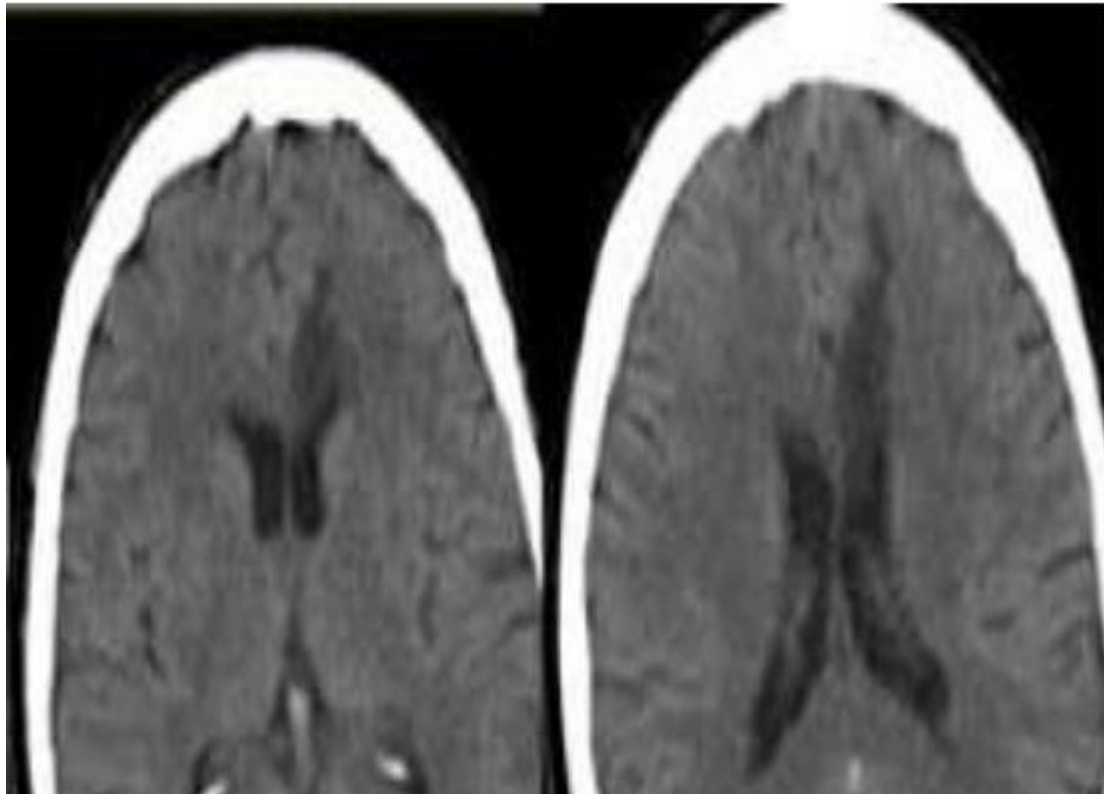
# CT SIGNS IN EARLY MCA ISCHEMIA



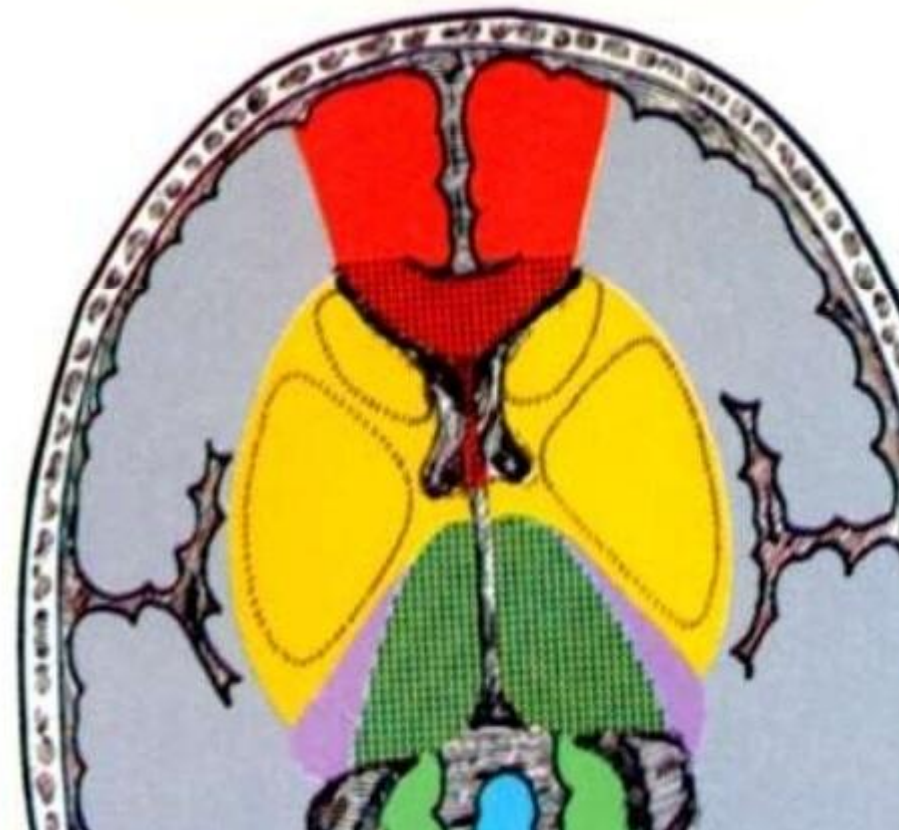
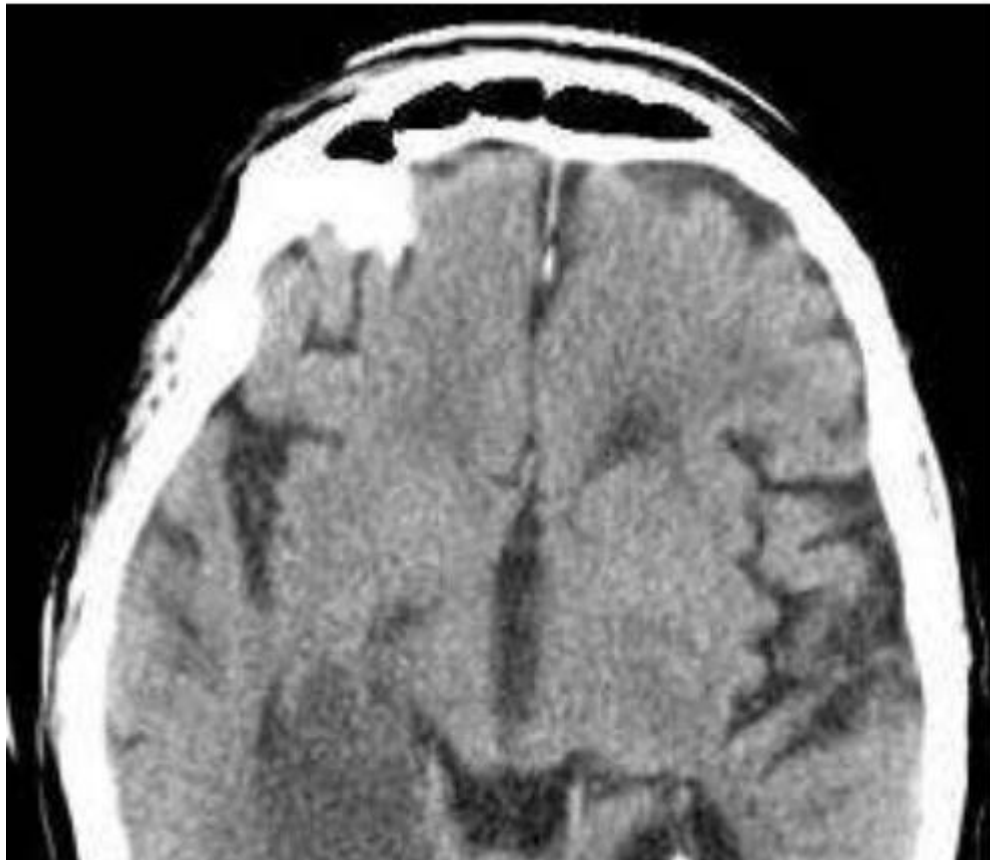
# MCA INFARCT



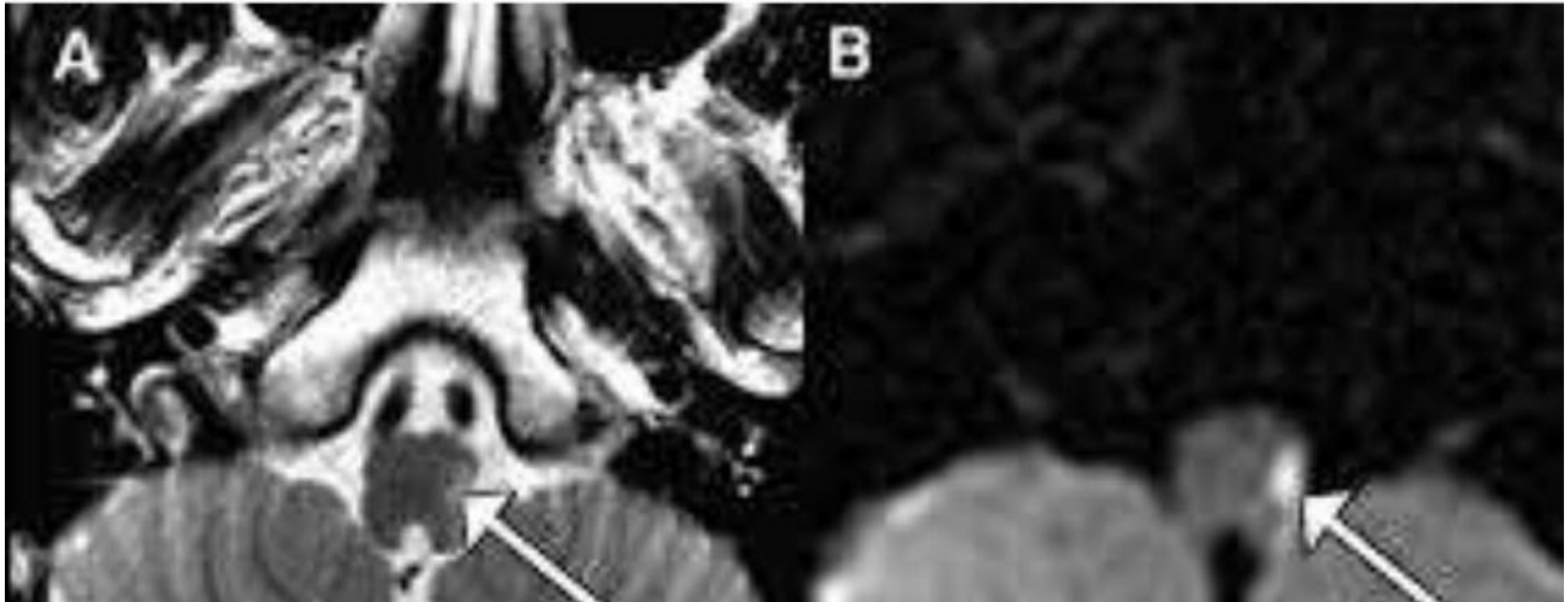
# ACA INFARCT



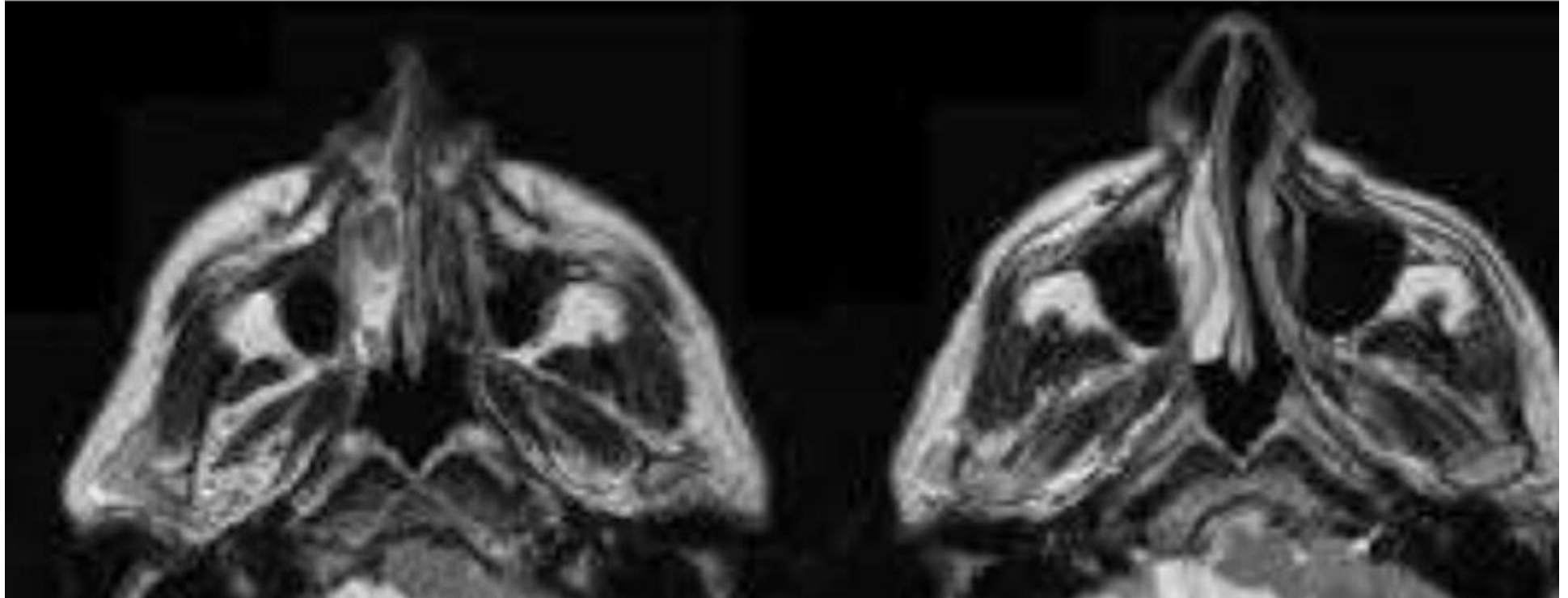
# PCA INFARCT



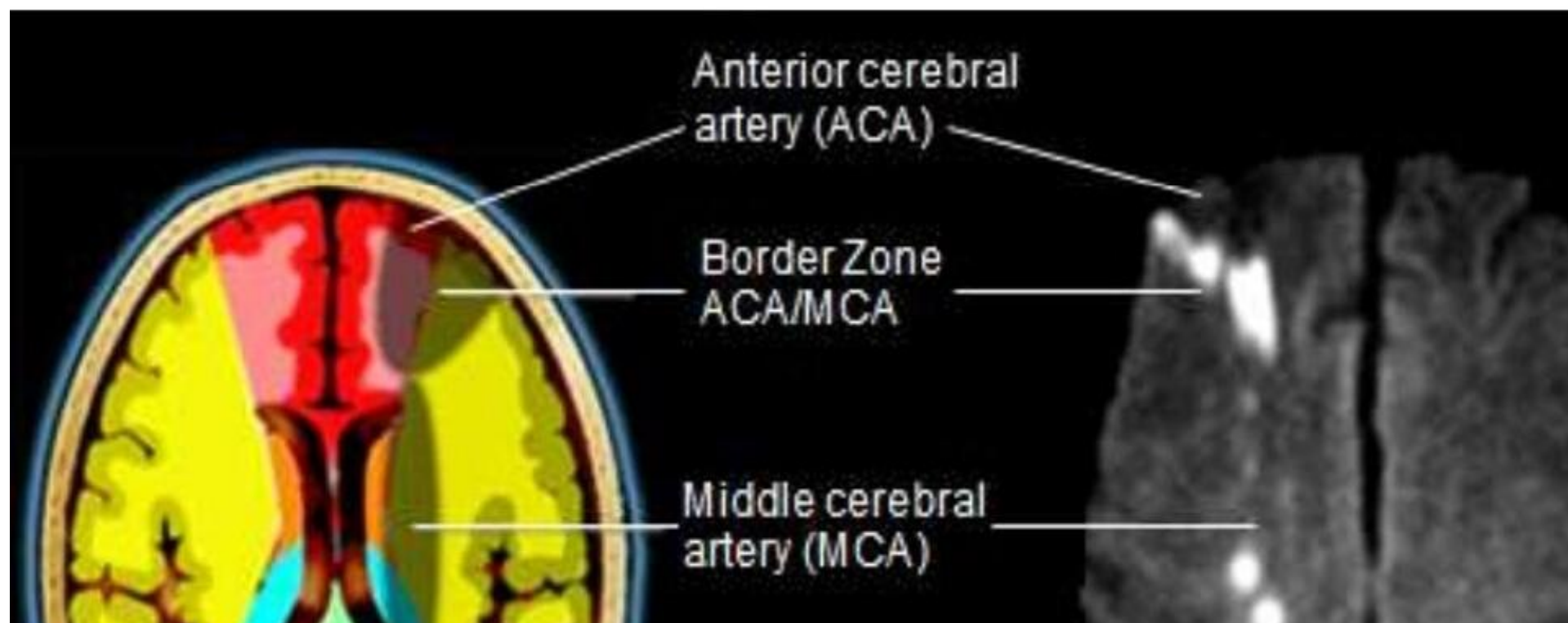
# MEDULLARY INFARCT



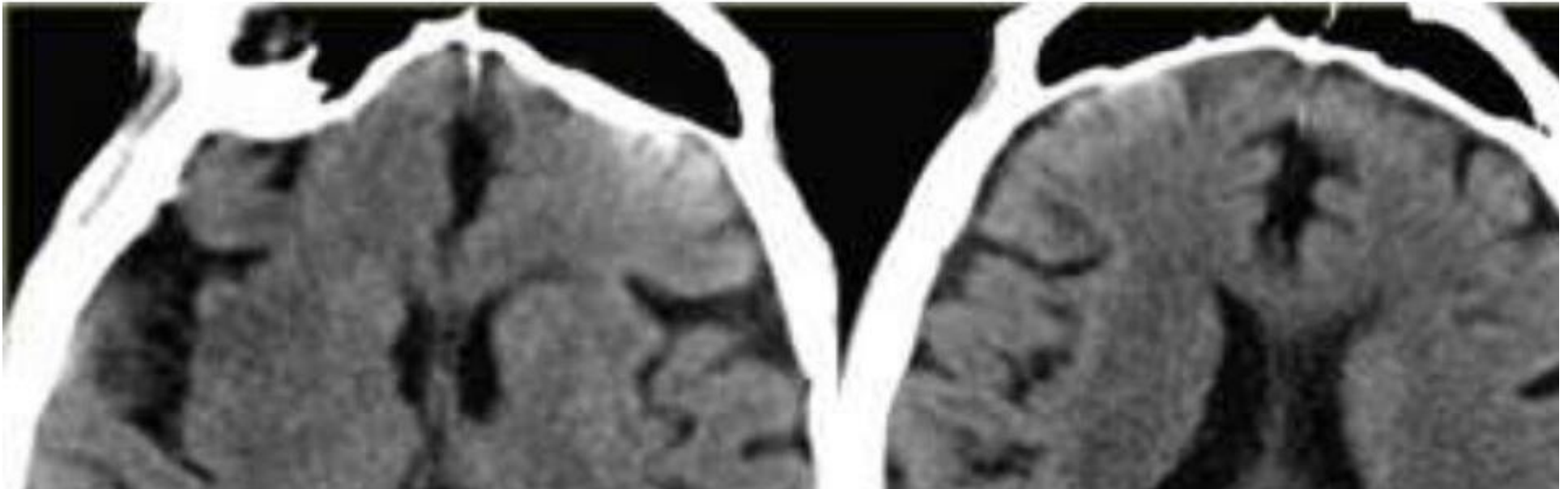
# CEREBELLAR INFARCT



# WATER SHED INFARCT



What do you see here? What do you expect on CTA?



# FILLING DEFECT DUE TO THROMBUS



## **TAKE HOME MESSAGE**

- **Appropriate usage of imaging modalities as per clinical scenario and availability.**
- **Both CT and MR imaging are useful for the comprehensive evaluation of acute stroke.**

F 1/1/1968  
Run 9B - Frame 9/22  
Lateral

ZOO

