VELAMMAL MEDICAL COLLEGE
HOSPITAL AND RESEARCH INSTITUTE
MADURAI - 625009

1.2.2

Add-on Certificates Details with Report

Index

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

VELAMMAL MEDICAL COLLEGE
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MADURAI - 625009

Department of Anatomy

Report of Certificate Course

Topic- Cavity Embalming

Date – 18th October 2018

Time – 8am – 4pm

Duration- 8 Hours

Venue- Department Of Anatomy

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

Program Coordinator- Dr. S Raja Sankar

Target Audience- 1st MBBS Students

Total Registered Participants- 150

Total Students Participated- 150

Speaker-

Dr. S Raja Sankar

Mr. David Ebenezer

Mode of Teaching - Classroom

1.	Introduction	8-9am	Dr. S Raja Sankar
2.	History and types of embalming	9-10am	Mr David Ebenezer
3.	Development of modern embalming	10.15-11.15am	Dr. S Raja Sankar
4.	Process of cavity embalming and fluid used	11.15-1pm	Dr. S Raja Sankar
5.	Purpose of cavity embalming	2-3 pm	Mr David Ebenezer
6.	Preference of cavity embalming in comparison to other types	3-4pm	Dr. S Raja Sankar

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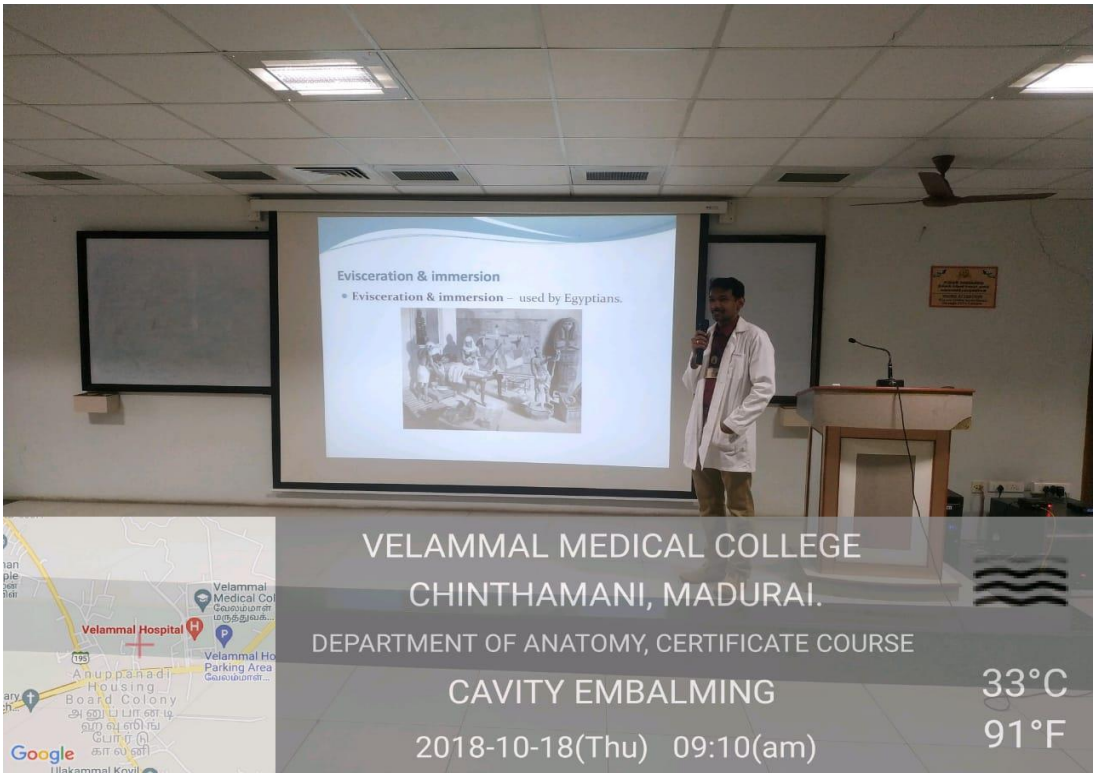

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MADURAI - 625009

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 "**Cavity Embalming**" conducted on 18th October 2018 by Department of Anatomy

Mr. M Ram Kumar
Coordinator

Dr. Sumana R
Head of Department

VELAMMAL MEDICAL COLLEGE
HOSPITAL AND RESEARCH INSTITUTE
MADURAI - 625009

Department of Anatomy

Report of Certificate Course

Topic- Special Stains

Date – 15th April 2019

Time – 8am – 4pm

Duration- 8 Hours

Venue- Department Of Anatomy

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

Program Coordinator- Mr. David Ebenezer

Target Audience- 1st MBBS Students

Total Registered Participants- 150

Total Students Participated- 149

Speaker-

Dr. Sumana R

Dr. Parineeta Suman

Mode of Teaching - Classroom

1.	Staining procedure	8-9am	Dr. Parineeta Suman
2.	Types of Special staining	9-10am	Dr. Sumana R
3.	Choosing the tissue for special stain	10.15-11.15am	Dr. Parineeta Suman
4.	Preparing tissue for special stain	11.15-1pm	Dr. Parineeta Suman
5.	Steps to improve quality of special stain	2-3 pm	Dr. Sumana R
6.	Staining stability and storage of reagents	3-4pm	Dr. Parineeta Suman

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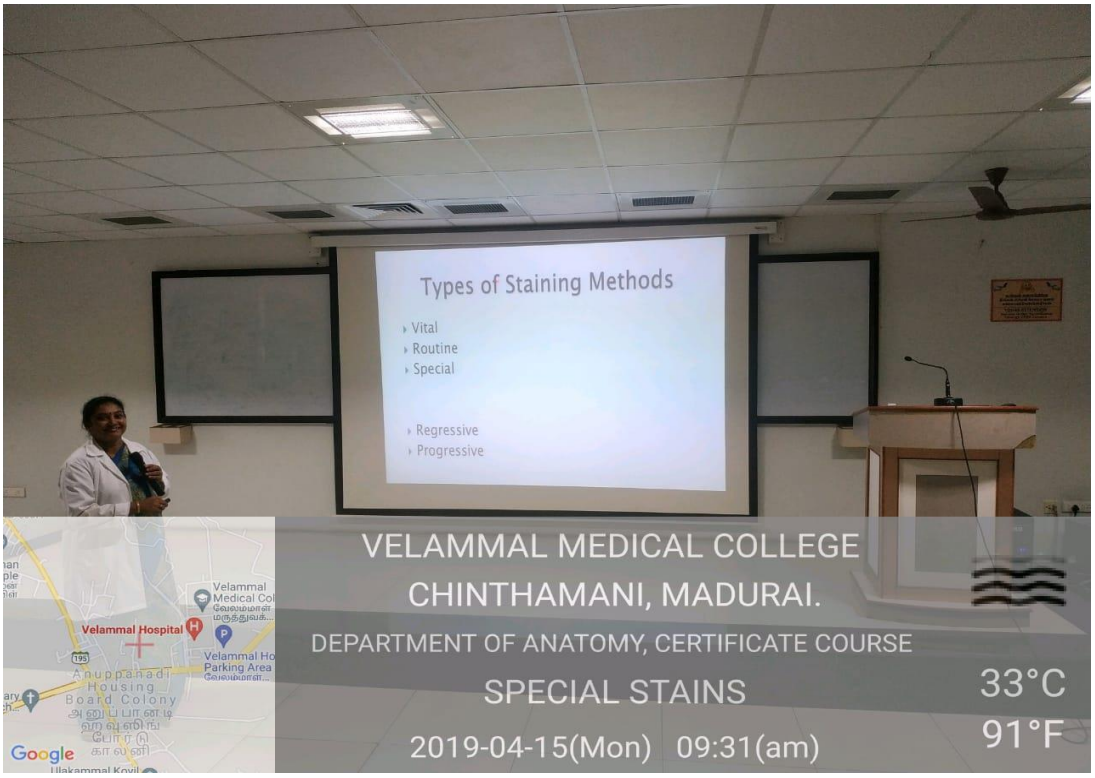

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VELAMMAL MEDICAL COLLEGE
HOSPITAL AND RESEARCH INSTITUTE
MADURAI - 625009

Certificate Sample-

Velammal Medical College Hospital and Research Institute, Madurai, TN

CERTIFICATE

OF PARTICIPATION

PROUDLY PRESENTED TO

NAME

For participating in the certificate course on "**Special stains**" conducted on 15th April 2019 by Department of Anatomy

Dr. Parineeta Suman
Coordinator

Dr. Sumana R
Head of Department

Department of Physiology

Velammal Medical College Hospital and Research Institute

Madurai

VMCH/Physio/CC/2018/02

CIRCULAR

To First MBBS students of 2018-19 batch

2.11.2018

There will be a certificate course on **ElectroEncephaloGram (EEG)** for first MBBS students from 9am to 4pm on Friday, 9-11-2018. All the students are expected to attend the course without fail.

Date: Friday, 9th November 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

ELECTROENCEPHALOGRAM

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

9 November 2018 • 9:00 am onwards

For First MBBS Students of 2018-19 batch

Venue

Lecture Hall 1, Velammal Medical College

Course Objective

At the conclusion of this activity, the learner (first MBBS student) will be better able to:

- Identify the indications of the electroencephalogram.
- Describe the technique of electroencephalogram.
- Outline the clinical significance of the electroencephalogram.

AGENDA

9.00 am	Inroduction
	By Dr.S.Anu, Professor and Head, Physiology
9.45 am	Basis of Action potential and recording
	By Dr.K.Rekha, Assistant Professor
10.30 am	Break
10.45 am	Functional anatomy of brain
	By Dr.M.Shanthi, Professor
11.45 noon	History and Physiology of brain potentials
	By Dr.Keba, Assistant Professor
1 pm	Lunch break
2 pm	Waves of brain – Wakefulness and sleep
	By Dr.A.S.Kanietha Priya, Assistant Professor
3 pm	Recording, Interpretation and Applied
	By Dr.M.Saravanan, Associate Professor
3.45 pm	Vote of thanks
	By Dr.John Rajpathy, Professor
4 pm	Conclusion

Electroencephalogram

record of electrical potentials of the brain

instrument is called **Electroencephalograph**

procedure is called **Electroencephalography**

- German psychiatrist Hans Berger recorded with scalp electrodes
- **ElectroCorticoGram (ECoG)** - record obtained with electrodes on the pial surface of the cortex

Sources of EEG

Influence of nerve terminals on dendrite tree and cell body produce local potentials (EPSP or IPSP) wave like potential fluctuation

Basic Pattern of EEG Influenced by

- Thalamus
- Reticular formation
- Recording...
- EEG records may be **bipolar** or **unipolar**
- Bipolar records show fluctuations in potential between two cortical electrodes
- Unipolar records show potential differences between a cortical electrode and a theoretically indifferent electrode on some part of the body distant from the cortex

Waves of EEG...

- **Alpha rhythm**
- **Beta rhythm**
- **Theta rhythm**
- **Delta rhythm**

Alpha Rhythm

- In adult humans who are awake but at rest with the mind wandering and the eyes closed

- Frequency of 8–12 Hz
- Amplitude of 50–100v when recorded from the scalp
- most marked in the parieto-occipital area, though it is sometimes observed in other locations
- On arousal, replacement of alpha rhythm with fast, high frequency, irregular wave from : **alpha block** or **desynchronization of EEG**

frequency of the alpha rhythm is decreased by

- Low blood glucose level
- Low body temperature
- Low level of glucocorticoid hormones
- High arterial partial pressure of CO_2 ($PaCO_2$)

Beta Rhythm

- Generally seen in the frontal region
- Normal awake pattern
- frequency 14-30 Hz

Theta Rhythm

- It is found over the parietal and occipital areas
- Normal in children and early sleep
- In adults during emotional stress and brain disorders
- Frequency 4-7 Hz

Delta Rhythm

- Usually appear during sleep
- Evidence of organic brain diseases when seen in awake state
- Frequency 1-4 Hz

Beta rhythm: in alert state

Alpha rhythm: awake but eyes closed

Theta rhythm: seen in children; adult with initiation of motor activity

Delta rhythm: NREM sleep

Uses of EEG

1. EEG is used to differentiate the type of epilepsy like Grandmal epilepsy and Petit mal epilepsy

2. EEG is used to localize areas in the brain which produce epilepsy

3. Delta wave in awake adult indicates intra cranial tumor

4. Theta wave in awake adult indicates depression

5. Low voltage waves EEG waves indicate clot (hematoma) under the skull

6. It helps to confirm the death (amplitude of EEG waves in a dead person is greatly reduced)

7. Prognostic factor in head injuries

8. To differentiate between organic and functional disorders of brain

Factors Influencing EEG waves

- Adrenaline, Noradrenaline, Amphetamine produce β rhythm (action on RF)
- Anaesthetic agents induce α or δ rhythm
- Infants show β rhythm → α rhythm by adolescence
- Decreased glucoarticoid, hyperglycaemia, hypothermia, hypercapnia – induce decrease in α rhythm frequency

Desyrichronisation of EEG

- Replacement of α rhythm – β rhythm ARAS is responsible for desynchronisation following sensory stimulation.

Synchronising mechanisms :

β rhythm →α rhythm

a) synchronising effect of adjacent parallel fibers.

b) Rhythmic discharge of impulses from thalamus.

c) Rhythmic discharge of stimulation at low frequency of some sub cortical centers.

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

Topic:	Certificate course in EEG
Date:	9-11-2018
Venue:	Lecture Hall 1, Velammal Medical College
Target Audience:	First MBBS students 2018-19 batch
Number of participants:	137

Report:

A certificate course on EEG was organised by Department of Physiology to first MBBS students on 9.11.2018. 137 first MBBS students participated in the course. The program began by 9am with introduction to the topic followed by scientific lectures on Basis of Action potential and recording, Functional anatomy of brain, History and Physiology of brain potentials, Waves of brain – Wakefulness and sleep, Recording, Interpretation and Applied by faculty of Department of Physiology. The program concluded with vote of thanks.

Outcome:

Students learnt about the concepts of EEG, its clinical importance, theoretical aspects of placements of electrodes and interpretation. The coverage of the topic stimulated their interest in the concepts of biopotentials and wayed a platform for their understanding of action potentials and evoked potentials.

Photos:

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

Department of Physiology

Velammal Medical College Hospital and Research Institute

Madurai

VMCH/Physio/CC/2019/01

CIRCULAR

To First MBBS students of 2018-19 batch

25.1.2019

There will be a certificate course on **Aviation and Space Physiology** for first MBBS students from 9am to 4pm on Friday, 1-2-2019. All the students are expected to attend the course without fail.

Date: Friday, 1st February 2019

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

AVIATION AND SPACE PHYSIOLOGY

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

1 February 2019 • 9:00 am onwards

For First MBBS Students of 2018-19 batch

Venue

Lecture Hall 1, Velammal Medical College

Course Objective

At the conclusion of this activity, the learner (first MBBS student) will be better able to:

- Identifying the circumstance in which humans undergo in proposed aerospace operations
- Identify the physiologic effects of flight and spaceflight on human physiology
- Analyse relationships among systems that influence the health of humans in the flight and spaceflight environments
- Understand the design of biomedical equipment to promote flight safety.

AGENDA

9.00 am	Inroduction
	By Dr.S.Anu, Professor and Head, Physiology
9.45 am	Space flight physiology
	By Dr.M.Shanthi, Professor
10.30 am	Break
10.45 am	Human capabilities in space & system adaptation
	By Dr.K.Rekha, Assistant Professor
11.45 noon	Psychological Considerations and training
	By Dr.A.S.Kanietha Priya, Assistant Professor
1 pm	Lunch break
2 pm	Biomedical equipment of safety & consideration
	By Dr.Keba, Assistant Professor
3 pm	Recent advances in aerospace physiology
	By Dr.M.Saravanan, Associate Professor
3.45 pm	Vote of thanks
	By Dr.John Rajpathy, Professor
4 pm	Conclusion

AVIATION AND SPACE PHYSIOLOGY

Barometric Pressures at Different Altitudes.

At sea level, the barometric pressure is 760 mm Hg; at 10,000 feet, only 523 mm Hg; and at 50,000 feet, 87 mm Hg.

This decrease in barometric pressure is the basic cause of all the hypoxia problems in high-altitude physiology because, as the barometric pressure decreases, the atmospheric oxygen partial pressure decreases proportionately, remaining at all times slightly less than 21 per cent of the total barometric pressure—Po2 at sea level about 159 mm Hg, but at 50,000 feet only 18 mm Hg.

Alveolar PO2 at Different Elevations

The approximate Po2s in the alveoli at different altitudes when one is breathing air for both the unacclimatized and the acclimatized person.

At sea level, the alveolar Po2 is 104 mm Hg; at 20,000 feet altitude, it falls to about 40 mm Hg in the unacclimatized person but only to 53 mm Hg in the acclimatized.

The difference between these two is that alveolar ventilation increases much more in the acclimatized person than in the unacclimatized person.

Carbon Dioxide and Water Vapor Decrease the Alveolar Oxygen.

At high altitudes carbon dioxide is continually excreted from the pulmonary blood into the alveoli.

Water vaporizes into the inspired air from the respiratory surfaces.

These two gases dilute the oxygen in the alveoli, thus reducing the oxygen concentration.

Water vapor pressure in the alveoli remains 47 mm Hg as long as the body temperature is normal, regardless of altitude.

In the case of carbon dioxide, during exposure to very high altitudes, the alveolar Pco2 falls from the sea-level value of 40 mm Hg to lower values. In the acclimatized person, who increases his or her ventilation about fivefold, the Pco2 falls to about 7 mm Hg because of increased respiration.

Effect of Breathing Pure Oxygen on Alveolar PO2 at Different Altitudes

If a person breathes pure oxygen instead of air, most of the space in the alveoli formerly occupied by nitrogen becomes occupied by oxygen.

At 30,000 feet, an aviator could have an alveolar Po2 as high as 139 mmHg instead of the 18 mm Hg when breathing air.

The red curve of arterial blood hemoglobin oxygen saturation at different altitudes when one is breathing pure oxygen. Note that the saturation remains above 90 per cent until the aviator ascends to about 39,000 feet; then it falls rapidly to about 50 per cent at about 47,000 feet.

Acclimatization to Low PO2

The principal means by which acclimatization comes about are

(1) a great increase in pulmonary ventilation,

(2) increased numbers of red blood cells,

(3) increased diffusing capacity of the lungs,

(4) Increased vascularity of the peripheral tissues, and

(5) Increased ability of the tissue cells to use oxygen despite low Po2.

(6) Cellular Acclimatization. In animals native to altitudes of 13,000 to 17,000 feet, cell mitochondria and cellular oxidative enzyme systems are slightly more plentiful than in sea-level inhabitants.

Natural Acclimatization of Native Human Beings Living at High Altitudes

Many native human beings in the Andes and in the Himalayas live at altitudes above 13,000 feet—one group in the Peruvian Andes lives at an altitude of 17,500 feet and works a mine at an altitude of 19,000 feet.

Many of these natives are born at these altitudes and live there all their lives. In all aspects of acclimatization, the natives are superior to even the best acclimatized lowlanders, even though the lowlanders might also have lived at high altitudes for 10 or more years.

Acclimatization of the natives begins in infancy. The chest size, especially, is greatly increased, whereas the body size is somewhat decreased, giving a high ratio of ventilatory capacity to body mass.

In addition, their hearts, which from birth onward pump extra amounts of cardiac output, are considerably larger than the hearts of lowlanders.

Reduced Work Capacity at High Altitudes and Positive Effect of Acclimatization

Acute Mountain Sickness and High-Altitude Pulmonary Edema

Acute cerebral edema

Acute pulmonary edema

Chronic Mountain Sickness

(1) the red cell mass and hematocrit become exceptionally high

(2) the pulmonary arterial pressure becomes elevated even more than the normal elevation that occurs during acclimatization,

(3) the right side of the heart becomes greatly enlarged,

(4) the peripheral arterial pressure begins to fall

(5) congestive heart failure ensues, and

(6) death often follows unless the person is removed to a lower altitude.

Rapid changes in velocity and direction of motion in airplanes or spacecraft, several types of acceleratory forces affect the body during flight.

Acceleratory Force—"G." : +G and –G

Effects of Centrifugal Acceleratory Force on the Body— (Positive G)

The most important effect of centrifugal acceleration is on the circulatory system, because blood is mobile and can be translocated by centrifugal forces. When an aviator is subjected to positive G, blood is centrifuged toward the lowermost part of the body.

Thus, if the centrifugal acceleratory force is +5 G and the person is in an immobilized standing position, the pressure in the veins of the feet becomes greatly increased (to about 450 mm Hg). Both systolic and diastolic pressure varies due to increase in G force.

Acceleration greater than 4 to 6 G causes "blackout" of vision within a few seconds and unconsciousness shortly thereafter. If this great degree of acceleration is continued, the person will die.

Effects on the Vertebrae.

Extremely high acceleratory forces for even a fraction of a second can fracture the vertebrae. The degree of positive acceleration that the average person can withstand in the sitting position before vertebral fracture occurs is about 20 G.

Negative G.

The effects of negative G on the body are less dramatic acutely but possibly more damaging permanently than the effects of positive G.

Protection of the Body Against Centrifugal Acceleratory Forces.

Specific procedures and apparatus

Tightening the abdomen muscles and bend forward can delay "black out"

Special anti G suit to overcome this problem

Effects of Linear Acceleratory Forces on the Body

Blast-off acceleration and landing deceleration can be tremendous; both of these are types of linear acceleration, one positive and the other negative.

Parachuting and deceleration

parachuting aviator leaves the airplane, the velocity of fall is at first exactly zero feet per second.

Because of the acceleratory force of gravity, within 1 second his velocity of fall is 32 feet per second (if there is no air resistance); in 2 seconds it is 64 feet per second; and so on. As the velocity of fall increases, the air resistance tending to slow the fall also increases.

After falling for about 12 seconds, the person will be falling at a "terminal velocity" of 109 to 119 miles per hour (175 feet per second).

an "opening shock load" of up to 1200 pounds can occur on the parachute shrouds.

Artificial Climate in the Sealed Spacecraft

Because of the absence of oxygen in the outer atmosphere artificial atmosphere and climate must be used in sealed spacecrafts.

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Dean
Velammal Medical College Hospital
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Madurai-Tuticorin Ring Road
Anuppanadi, Madurai, T.N.-625 009

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

Topic: Certificate course in Aviation and Space Physiology

Date: 1-2-2019

Venue: Lecture Hall 1, Velammal Medical College

Target Audience: First MBBS students 2018-19 batch

Number of participants: 150

Report:

A certificate course on Aviation and Space Physiology was organised by Department of Physiology to first MBBS students on 1.2.2019. 150 first MBBS students participated in the course. The program began by 9am with introduction to the topic followed by scientific lectures on Space flight physiology, Human capabilities in space & system adaptation, Psychological Considerations and training, Biomedical equipment of safety & consideration and Recent advances in aerospace physiology by faculty of Department of Physiology. The program concluded with vote of thanks.

Outcome:

Students learnt about the concepts of Aviation and Space physiology, barometric pressure and its influence on human physiology, changes in 'g', Alveolar PO2 at Different Elevations, Acclimatization, complications, Specific procedures and apparatus and Artificial Climate in the Sealed Spacecraft.

Photos:

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

Velammal Medical College Hospital and Research Institute

Ref. No: VMCHRI/BIOCHEM/CC-3 Date: 01.02.2019

CIRCULAR

To

All Doctors

Certificate Course on Analysis and Interpretation of Renal Function test

Department of Biochemistry is organizing a certificate course on Analysis and Interpretation of Renal Function test on 19.02.19 (Tuesday) between at 9.00 AM to 3.00 PM.

All Faculties are invited.

VICE PRINCIPAL

Dr. P.K. MOHANTY
Vice Principal
Velammal Medical College Hospital
and Research Institute
Madurai-625 009

Copy submitted to:

The Hon. Chairman

Copy to:

The Dean

Medical Superintendent

Chief Administration Officer

HOD, Biochemistry

All Clinical and Non-Clinical HODs

Velammal Medical College Hospital & Research Institute

Anuppanadi, Madurai - 625009

Department of Biochemistry

Certificate course on

Analysis and Interpretation of Renal Function test

Venue: Biochemistry

Demonstration Room

Date: 19.02.2019

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.K.Suganthy
Course coordinator
Biochemistry

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

Agenda

Time	Topic	Speaker
09.00-09.15 AM	Welcome address	DR.P.K.Mohanty
09.15 – 09.45 AM	Pre test	DR.K.Suganthy
09.45 – 11.00 AM	Renal Function	DR.K.Suganthy
11.00 AM – 1.00 PM	Renal Function Test	DR.K.Suganthy
1.00 – 2.00 PM	LUNCH	
2.00 2.30 PM	Post test	DR.K.Suganthy
2.30 – 3.00 PM	Feed back and Valediction	DR.P.K.Mohanty

RENAL FUNCTION TEST

-DR.K.Suganthy M.D., (Biochemistry)

Renal System

I. Functions of kidney.

II. Renal disorders.

III. Renal function test.

IV. Interpretation of renal function test.

V. Exercise questions.

II. Renal function test

Excretion

Metabolic waste

Urea
Creatinine
Uric acid

Hypertension

Potassium

Cardiac arrest

H^+ ion

Acid base disorder

II. Renal function test

Production.

- Vitamin D deficiency
- Anaemia

Renal disorders

- Polyuria, oliguria and anuria.
- Cardiac problems.
- Acid base disorders.
- Vitamin D deficiency.
- Anemia.
- Pedal oedema.
- Puffiness of face.
- Hypertension.
- Breathlessness.
- Retinopathy.

Renal disorders

I. Acute kidney injury:
   1. Prerenal AKI.
   2. Renal AKI:
      i. Vascular.
      ii. Glomerular.
      iii. Tubular.
   3. Postrenal AKI.

II. Chronic kidney diseases.

II. Renal Function test

I. To screen for kidney disease:

1. USG abdomen.
2. Complete urine analysis.
3. Serum urea and creatinine.

II. To assess renal function:

1. Glomerular function.
2. Tubular function.

III. Test to screen for complications.

II. Renal Function test

I. To screen for kidney disease:

1. USG abdomen.
2. Complete urine analysis.
3. Serum urea and creatinine.

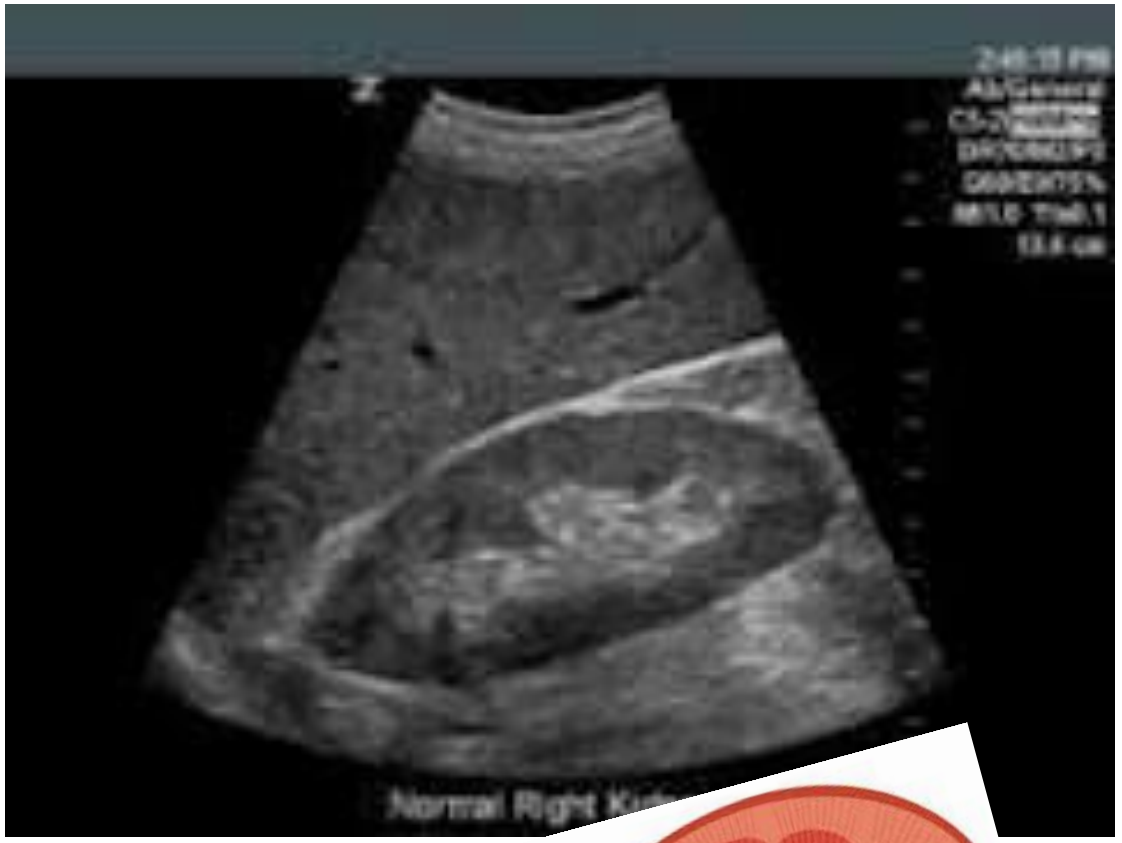
II. To assess renal function:

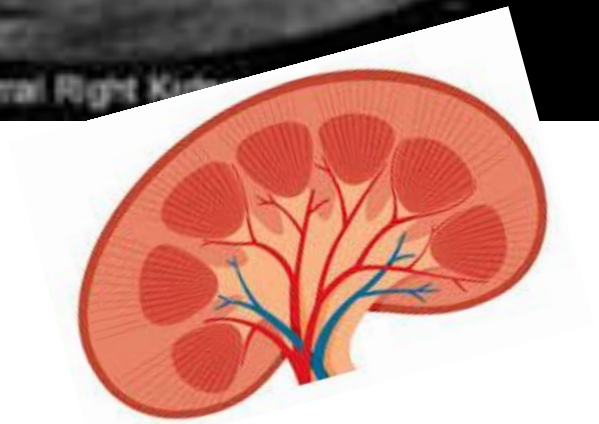
1. Glomerular function.
2. Tubular function.

III. Test to screen for complications.

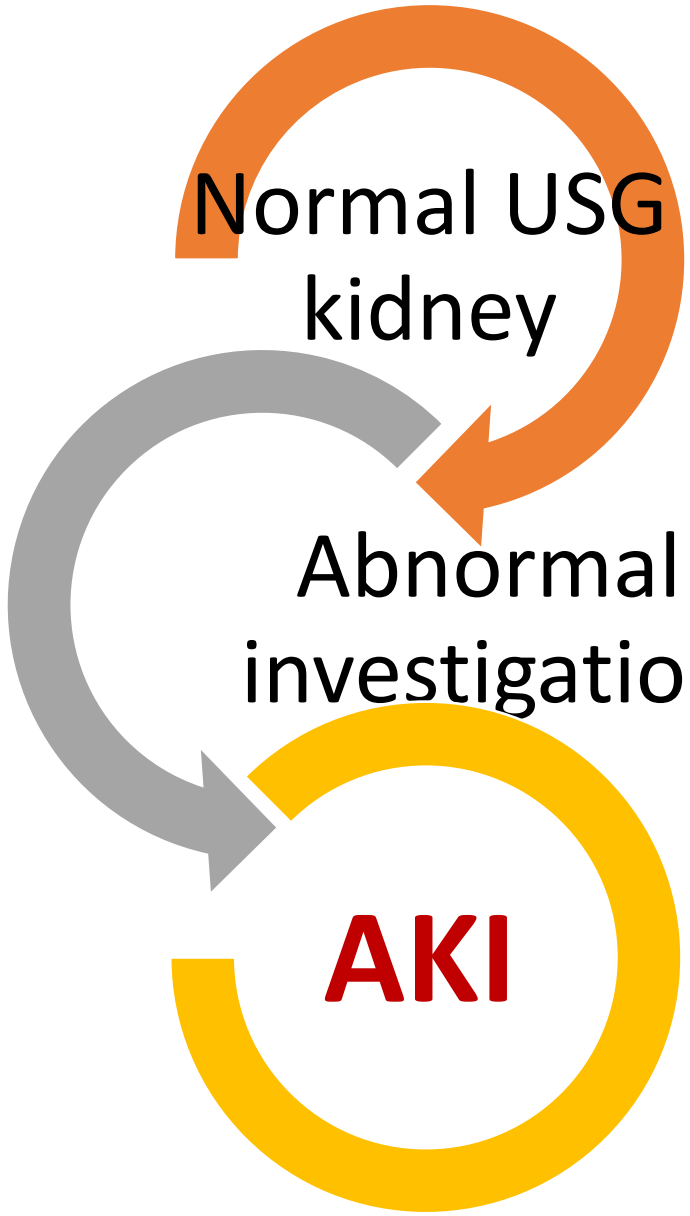
1. USG abdomen

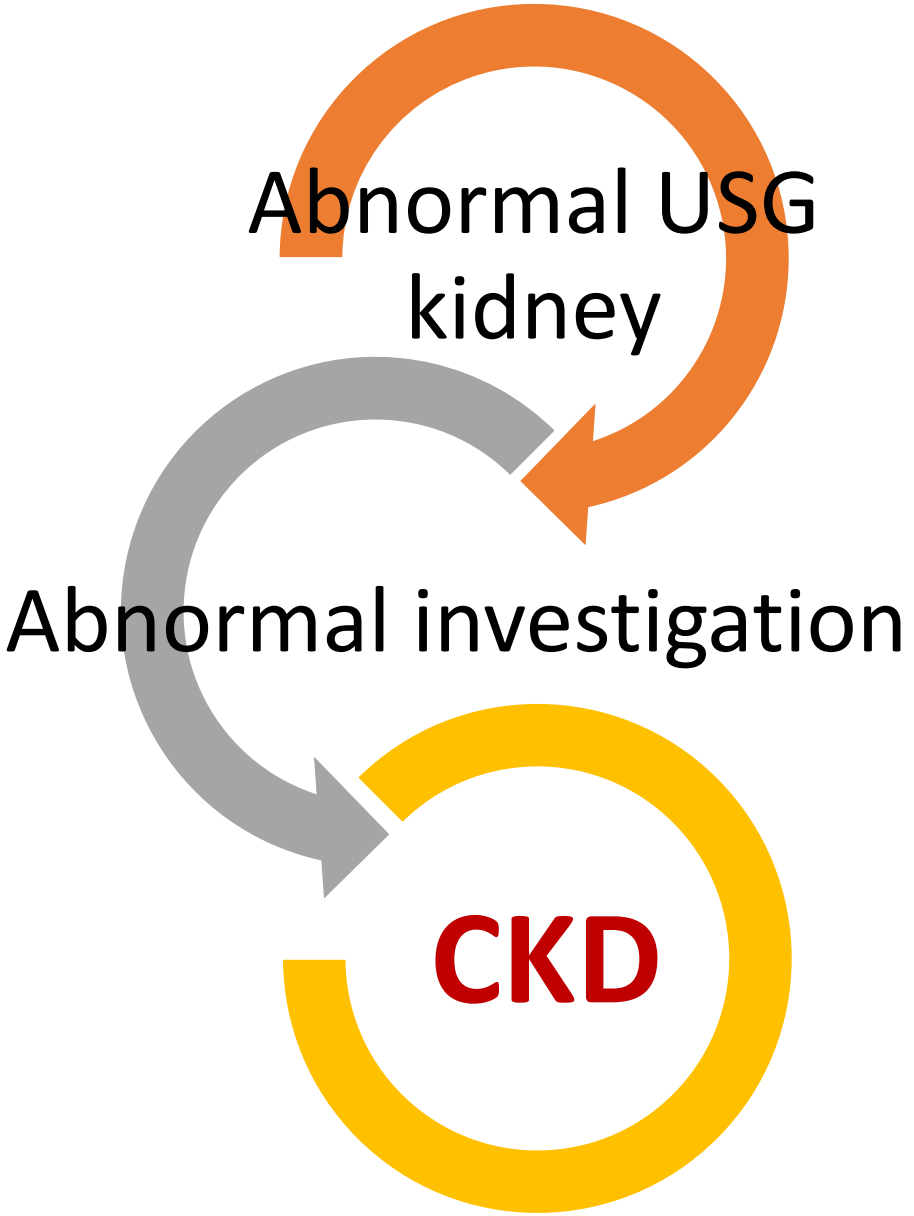
- Normal kidney size > 10 cm.
- If kidney size is < 8cm → contracted kidney → CKD.
- Corticomedullary differentiation.





1. USG abdomen





II. Renal Function test

I. To screen for kidney disease:

1. USG abdomen.
2. Complete urine analysis.
3. Serum urea and creatinine.

II. To assess renal function:

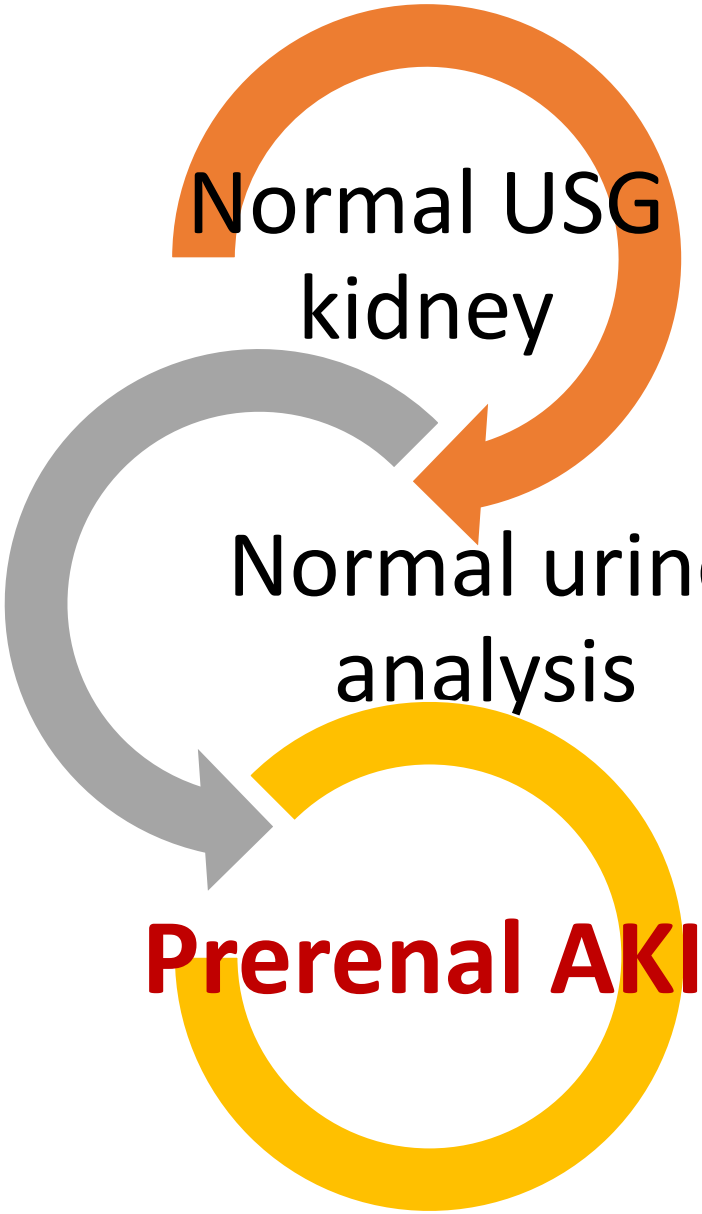
1. Glomerular function.
2. Tubular function.

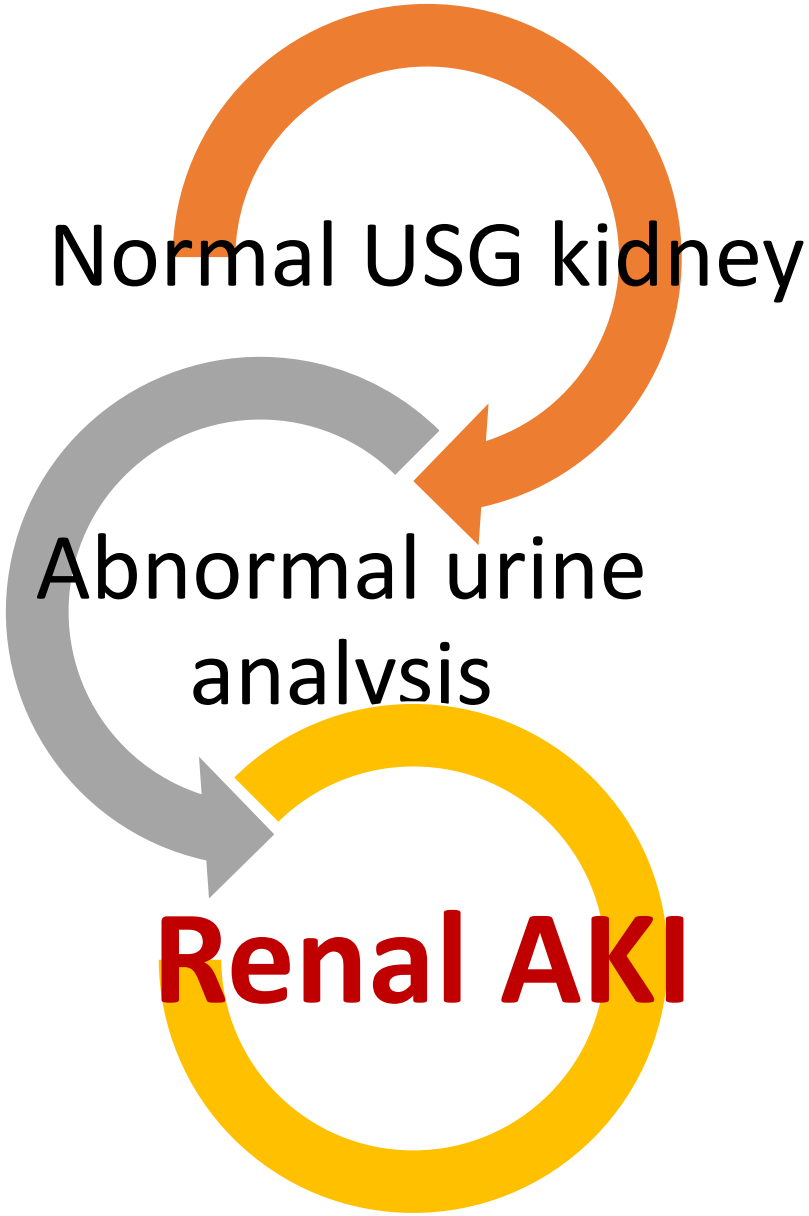
III. Test to screen for complications.

2. Complete urine analysis

- If the urine analysis is normal with elevated serum urea and creatinine→ prerenal disease.
- If the urine analysis is abnormal. Depending upon the abnormality it can be,
  - ➢Vascular disease.
  - ➢Glomerular disease.
  - ➢Tubular disease.

2. Complete urine analysis





2. Complete urine analysis

- Second morning mid stream sample has to be collected.
- Analysis with in 3hrs.
- Storage: 4^{*}- 8^{*} C → analysis within 12hrs.

2. Complete urine analysis

I. Physical characteristics:

i. Volume.

ii. Appearance.

iii. Color.

iv. Odor.

v. Specific gravity.

II. Chemical characteristics:

i. pH.

ii. Protein.

iii. Blood.

iv. Reducing sugar.

v. Ketone bodies.

vi. Urobilinogen, bile salt and pigments.

2. Complete urine analysis

1. Volume:

- Normal 24hrs urine volume : 800-2000 ml/day.
- **Polyuria**: 24hrs urine volume more than 2500 ml /day.
- **Oliguria**: 24hrs urine volume less than 500 ml /day.
- **Anuria**:24hrs urine volume less than 100 ml /day.

2. Complete urine analysis

1. Volume:

- **Polyuria**: DM, diabetes insipidous, late stage of glomerulonephritis etc.
- **Oliguria**: dehydration.
- **Anuria**: tubular necrosis, shock, bilateral renal stone.

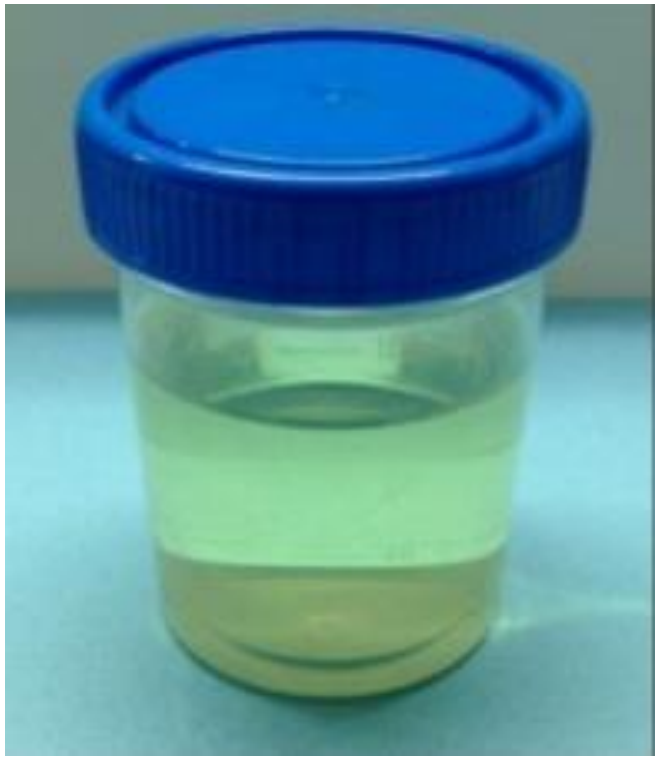
2. Complete urine analysis

2. Appearance :

- Normal fresh urine will we clear.
- If the fresh urine is turbid, it may be due to
  - Pus cell → Infection.

2. Complete urine analysis - color

Normal urine



Diluted urine

2. Complete urine analysis - color

Jaundice

Chyluria

2. Complete urine analysis - color

Hematuria

Hemoglobinuria

alkaptonuria/ imipenem

2. Complete urine analysis - color

Rifampicin

Deferoxamine

2. Complete urine analysis

4. Odor:

- Foul smell – bacterial infection.
- Fruity odour – Diabetic ketoacidosis.
- Mousy odour – Phenylketonuria.

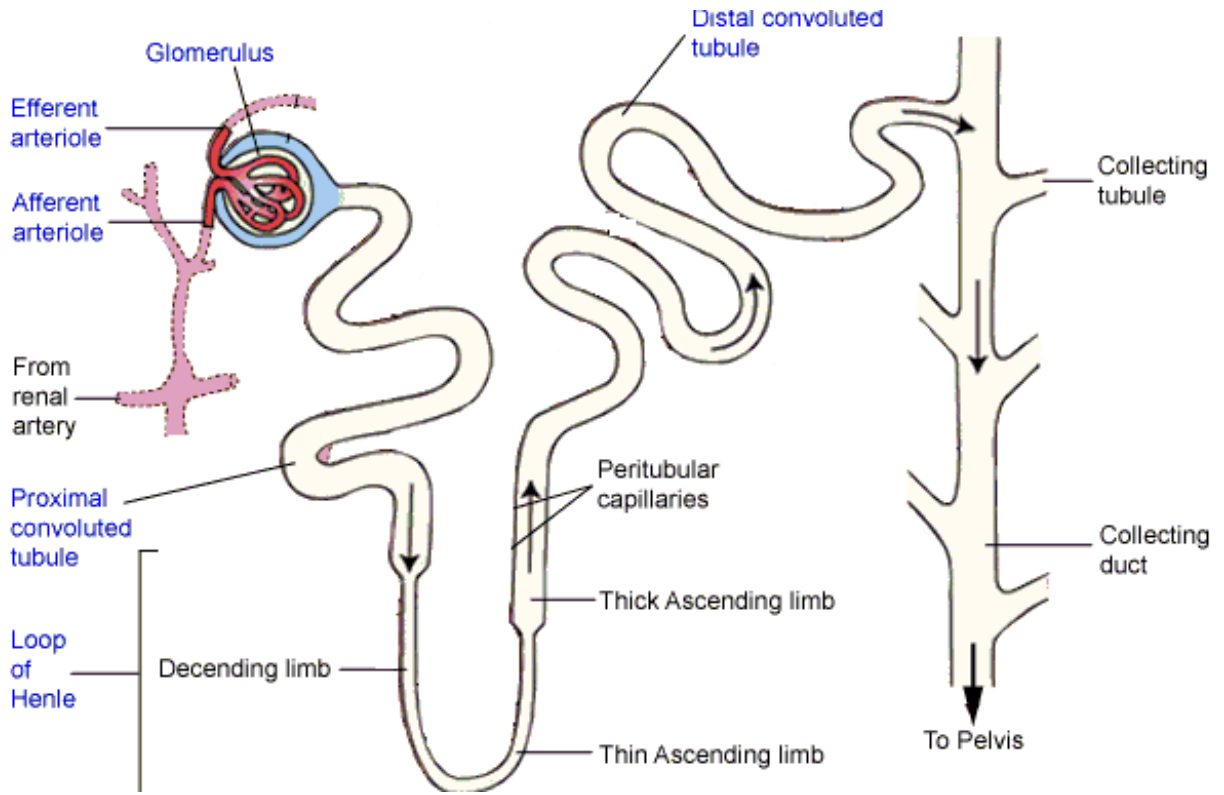
2. Complete urine analysis

5. Specific gravity :

- Blood specific gravity is 1.010.
- Urine specific gravity is 1.015 – 1.025.
- Isosthenuria.
- Chronic tubulointerstitial disease.

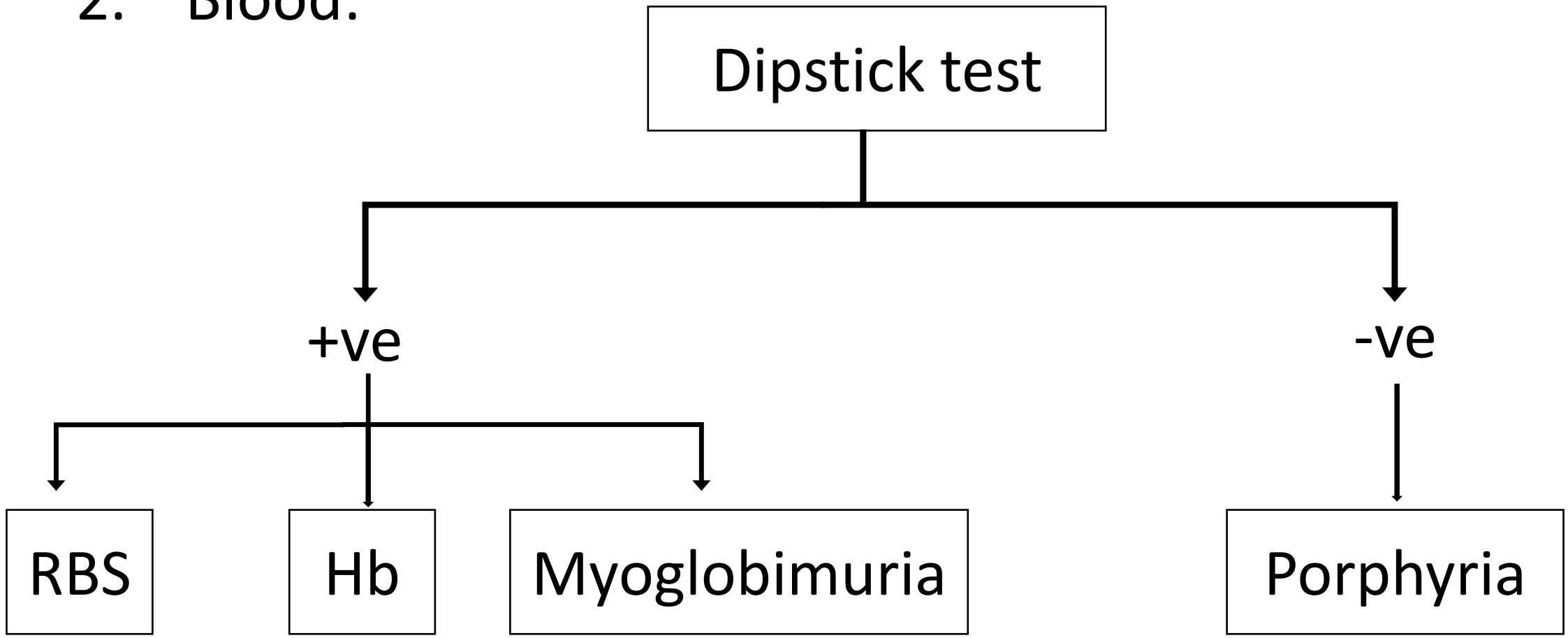
2. Complete urine analysis

1. pH:
   - Urine pH: 5.5 – 7.5.
   - More towards acidic side.
   - In PCT → HCO_3.
   - In PCT & DCT → H^+ is secreted.
   - Drug: Sodium bicarbonate and potassium citrate.



2. Complete urine analysis

2. Blood:



2. Complete urine analysis

2. Blood:

- Centrifuge the urine at 3000 rpm for 5 min → clear supernatant seen → RBC.
- If the urine is still high color →Hb or myoglobulin.

2. Complete urine analysis

2. Blood:

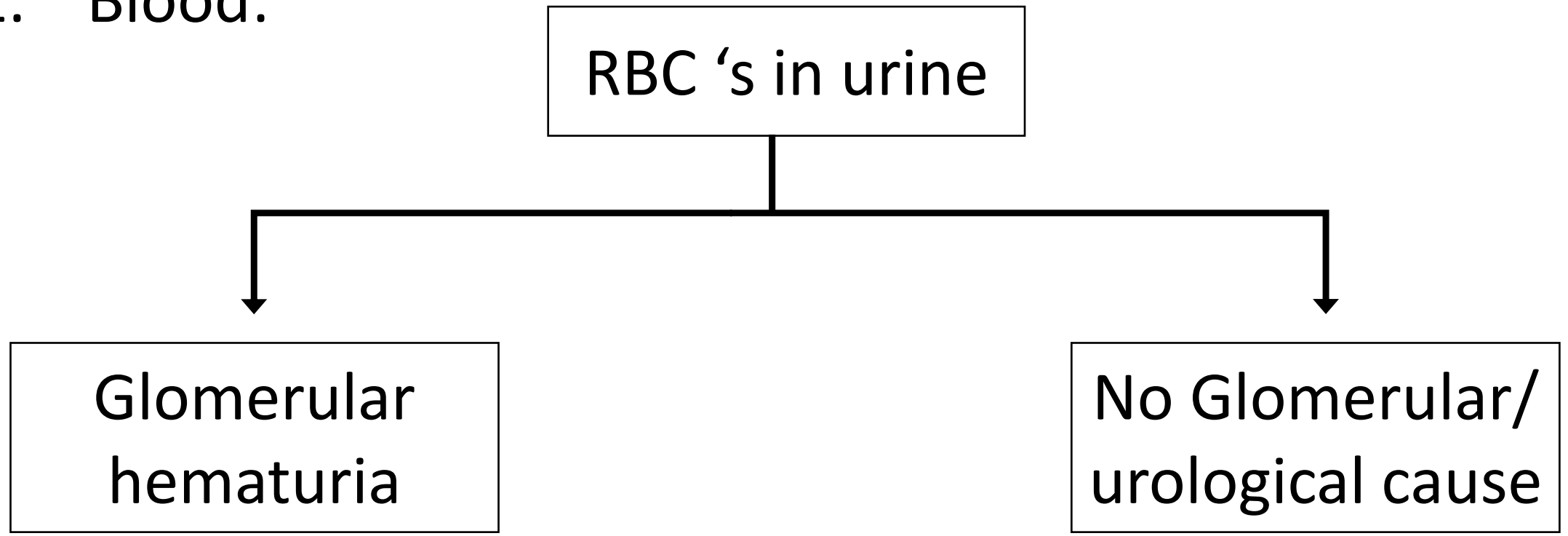
   i. Urological cause. (90%)

   ii. Nephrological cause. (9%)

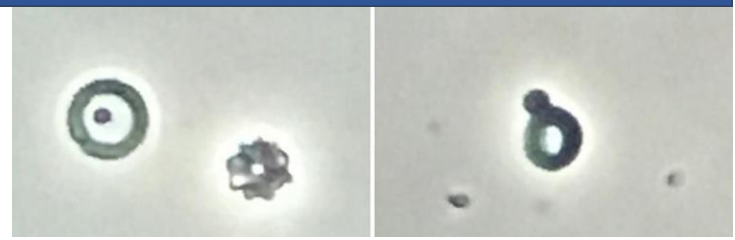
   iii. Hemolytic cause. (1%)

2. Complete urine analysis

2. Blood:

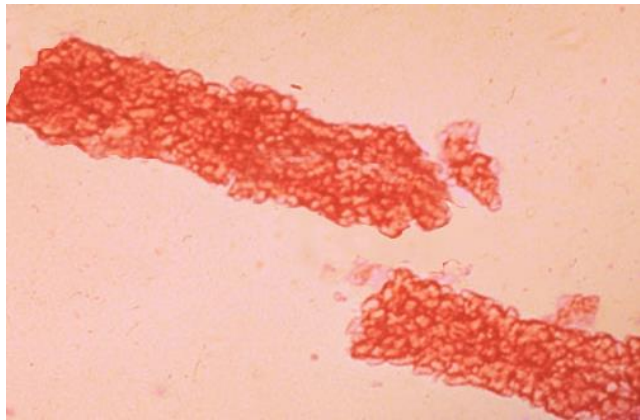


2. Complete urine analysis



2. Blood:

- Glomerular cause of hematuria: 40/5/1 rule.
  - 40% or more dysmorphic RBC.
  - 5% or more acanthocytes.
  - 1 RBC cast.



2. Complete urine analysis

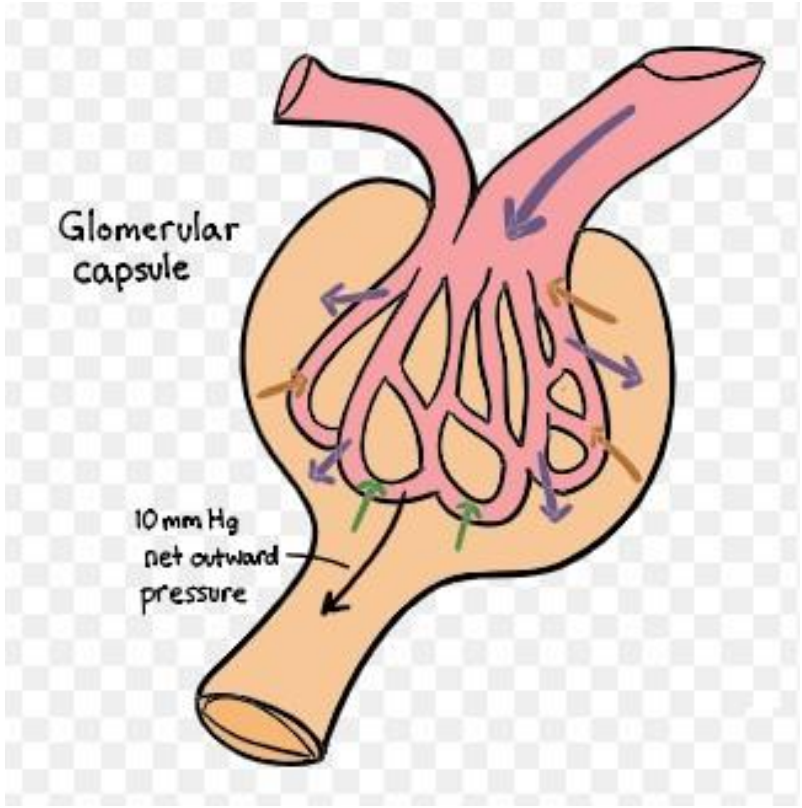
3. Protein:

- Glomerular proteinuria.
- Tubular proteinuria.
- Urogenic proteinuria.
- Overflow proteinuria.

2. Complete urine analysis

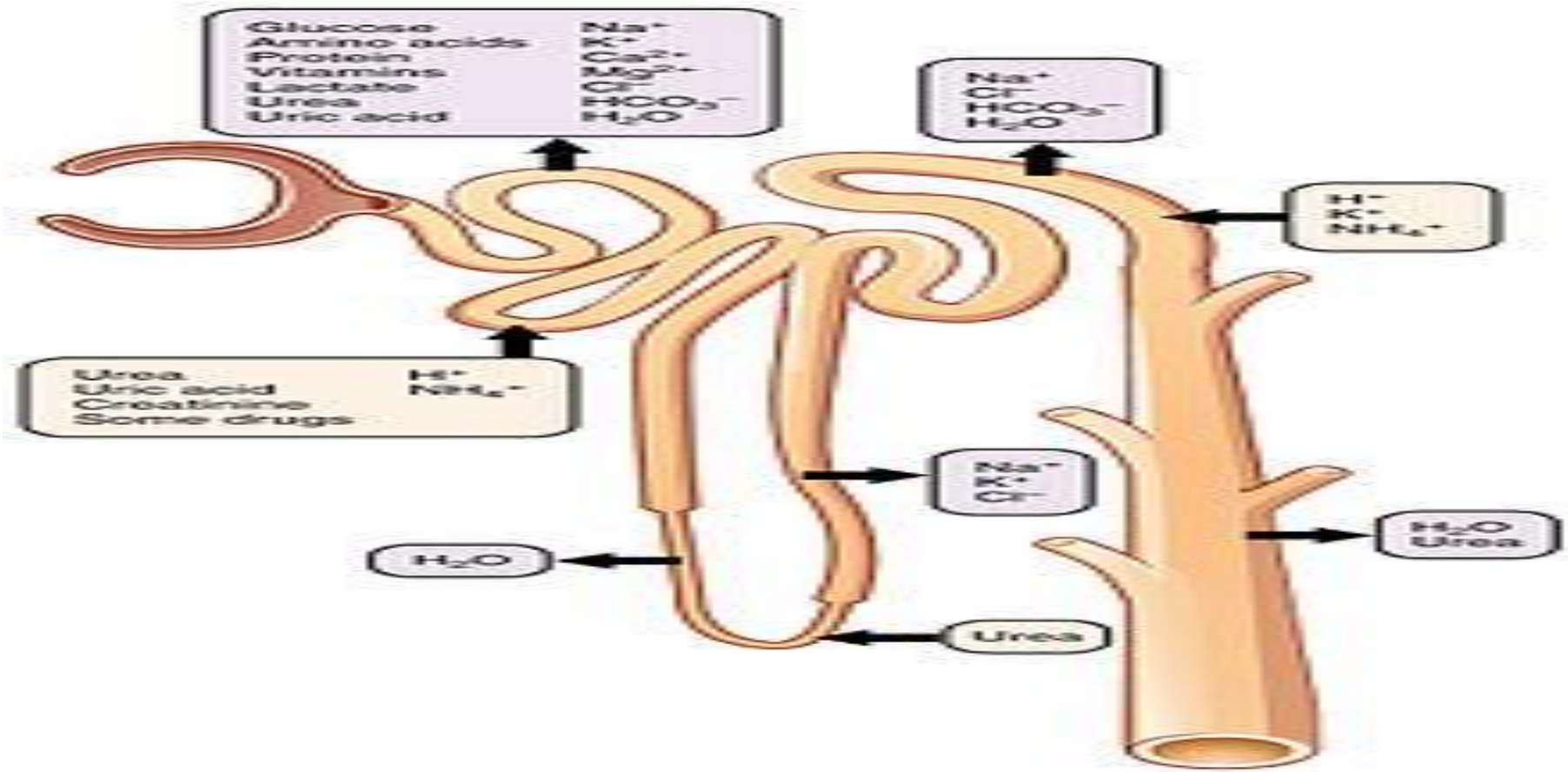
3. Protein:

- Glomerular proteinuria:



2. Complete urine analysis

3. Protein:
   - Tubular proteinuria: RBP and α-1 macroglobulin.



2. Complete urine analysis

3. Protein:

- Urogenic proteinuria: UTI.
- Overflow proteinuria: Multiple myloma.

2. Complete urine analysis

3. Protein:

- Dip stick test.
- 24hrs urine protein.
- Protein creatinine ratio.
- Albumin creatinine ratio.

2. Complete urine analysis

3. Protein: Dip stick test

Dip stick test	
Trace	15 mg/dL
+	30 mg/dL
++	100 mg/dL
+++	300 mg/dL
++++	1000 mg/dL

2. Complete urine analysis

3. Protein:

- 24hrs urine protein:
  - Normal: < 150 mg/day.
  - Functional proteinuria → 150 – 500 mg/day.
  - Nephrotic proteinuria → > 3.5 g/day.
  - Massive proteinuria → > 4g/day.

2. Complete urine analysis

3. Protein:

- Protein creatinine ratio :
  - Normal: < 0.1 mg/mg.

2. Complete urine analysis

3. Protein:

- Albumin creatinine ratio:
  - Normal → < 30 mg/g.
  - Microalbuminuria → 30 – 300 mg/g.
  - Albuminuria → > 300 mg/g.
  - Nephrotic albuminuria → > 2200 mg/g.

2. Complete urine analysis

- Reducing substance in urine:
  - Detected by benedict test.
  - It’s a semiquantitative test.
  - Renal threshold for glucose = 180 mg/dL.
  - Benedicts test is positive for glucose, fructose, lactose, galactose, Ascorbic acid and homogentisic acid.

2. Complete urine analysis

- Ketone bodies.
- Urobilinogen.
- Bile salt.
- Bile pigments.

2. Complete urine analysis

Vascular (renal AKI)	Glomerular (renal AKI)	Tubular (renal AKI)
Urine albumin strip → trace/+	Urine albumin strip → 2 + or 3 + Hematuria	Urine albumin strip → trace/+
Hypertension	-	No hypertension

II. Renal Function test

I. **To screen for kidney disease:**

1. USG abdomen.
2. Complete urine analysis.
3. Serum urea and creatinine.

II. **To assess renal function:**

1. Glomerular function.
2. Tubular function.

III. **Test to screen for complications.**

3. Serum urea and creatinine

1. Urea:

- End product of protein catabolism.
- Urea is synthesized by liver and excreted by kidney.
- Reference value:
  - Serum urea = 15-40 mg/dL
  - Blood urea nitrogen = 6-20 mg/dL

3. Serum urea and creatinine

1. Urea:

- 24hrs Urinary urea = 15-30 g/day
- Urea is estimated by glutamate dehydrogenase method.
- Estimation of urea is used to differentiate prerenal, renal or postrenal type of AKI.

3. Serum urea and creatinine

1. Creatinine:

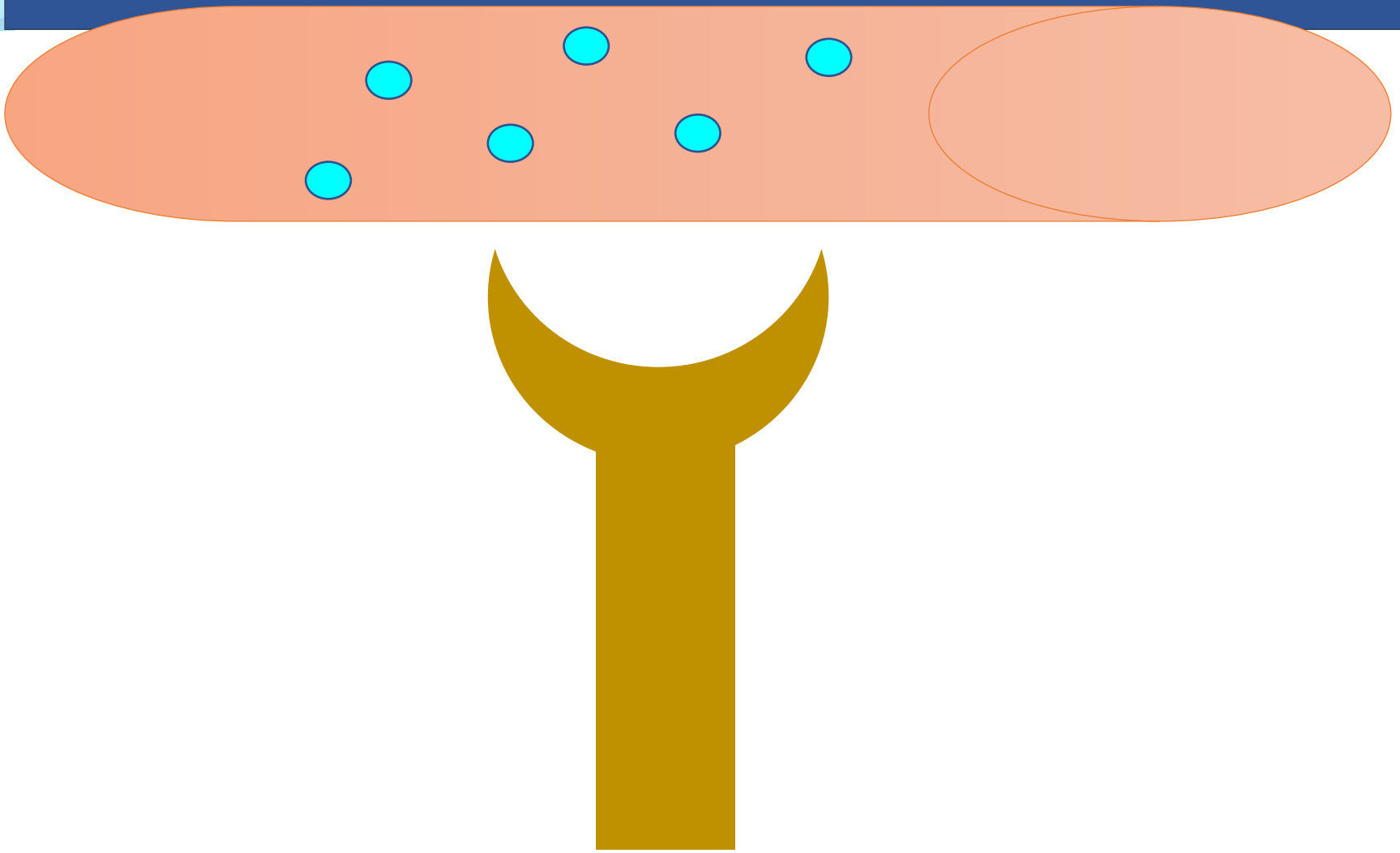
- Creatinine is produced from creatine which is the storage form of energy in muscle.
- Creatinine is the anhydride form of creatine.

3. Serum urea and creatinine

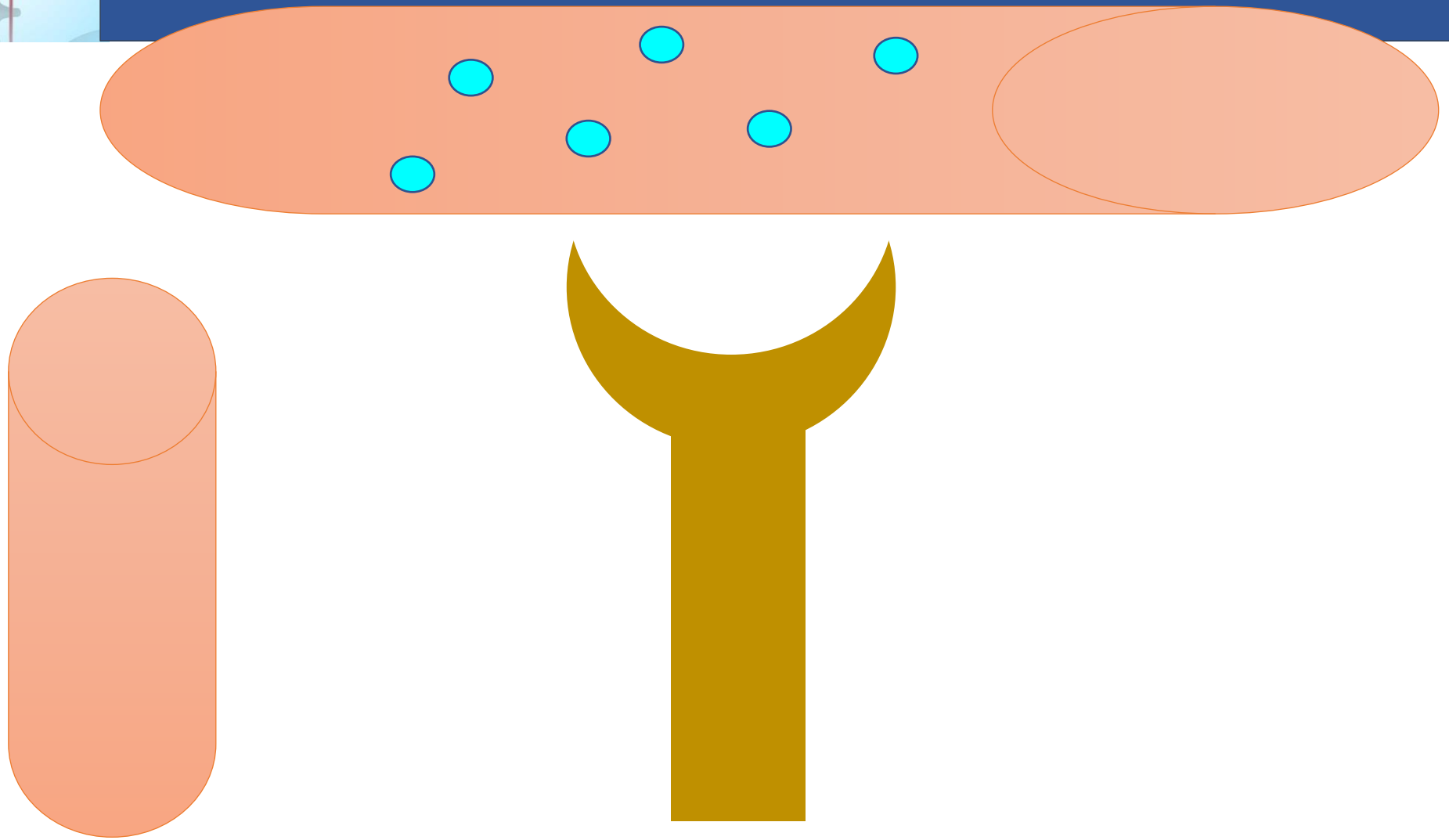
1. Creatinine:

   - Reference value:
   - Serum creatinine = 0.7-1.4 mg/dL.
   - 24hrs Urine creatinine = 1-2 g/day.

3. Serum urea and creatinine - Pre renal AKI



3. Serum urea and creatinine - Pre renal AKI



3. Serum urea and creatinine

	S. urea	BUN	S. creatinine	Urea: Creatinine ratio	BUN: Creatinine ratio
NORMAL	20 - 40	6 - 20	0.7 – 1.4	20 - 40	6 - 20
Prerenal	↑ ↑ ↑		N	> 40	> 20 :1
Renal	↑ ↑		↑ ↑ ↑	< 20	<10 :1
Postrenal	↑ ↑ ↑		↑ ↑ ↑	20 - 40	10-20 :1

II. Renal Function test

I. **To screen for kidney disease:**

1. USG abdomen.
2. Complete urine analysis.
3. Serum urea and creatinine.

II. **To assess renal function:**

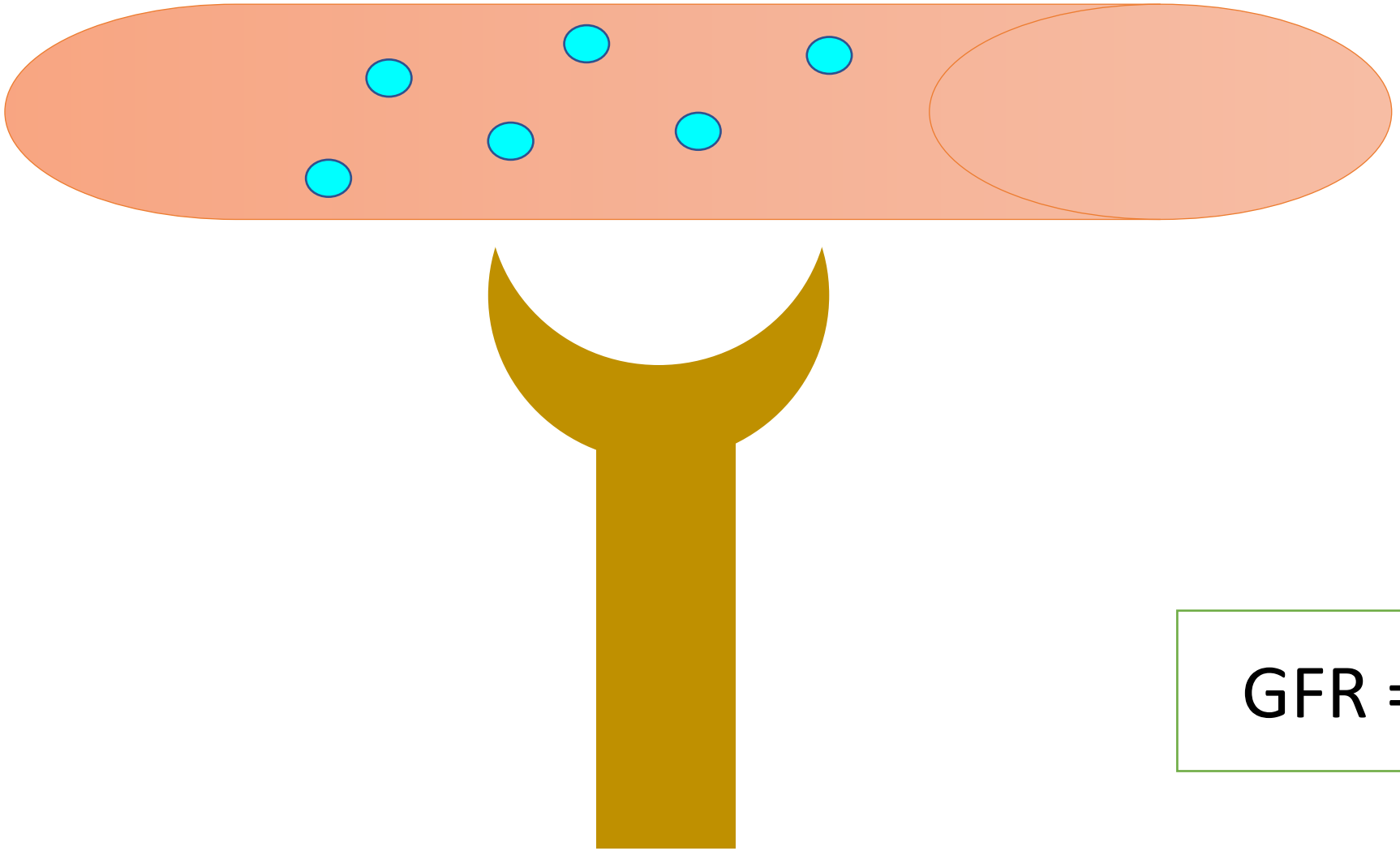
1. Glomerular function.
2. Tubular function.

III. **Test to screen for complications.**

1. GFR – Clearance test

1. **<u>Exogenous marker</u>:**
   - Inulin. (GOLD STANDARD)
2. **<u>Endogenous marker</u>:**
   - Urea.
   - Creatinine.
   - Cystatin C.

1. Glomerular function – Clearance test

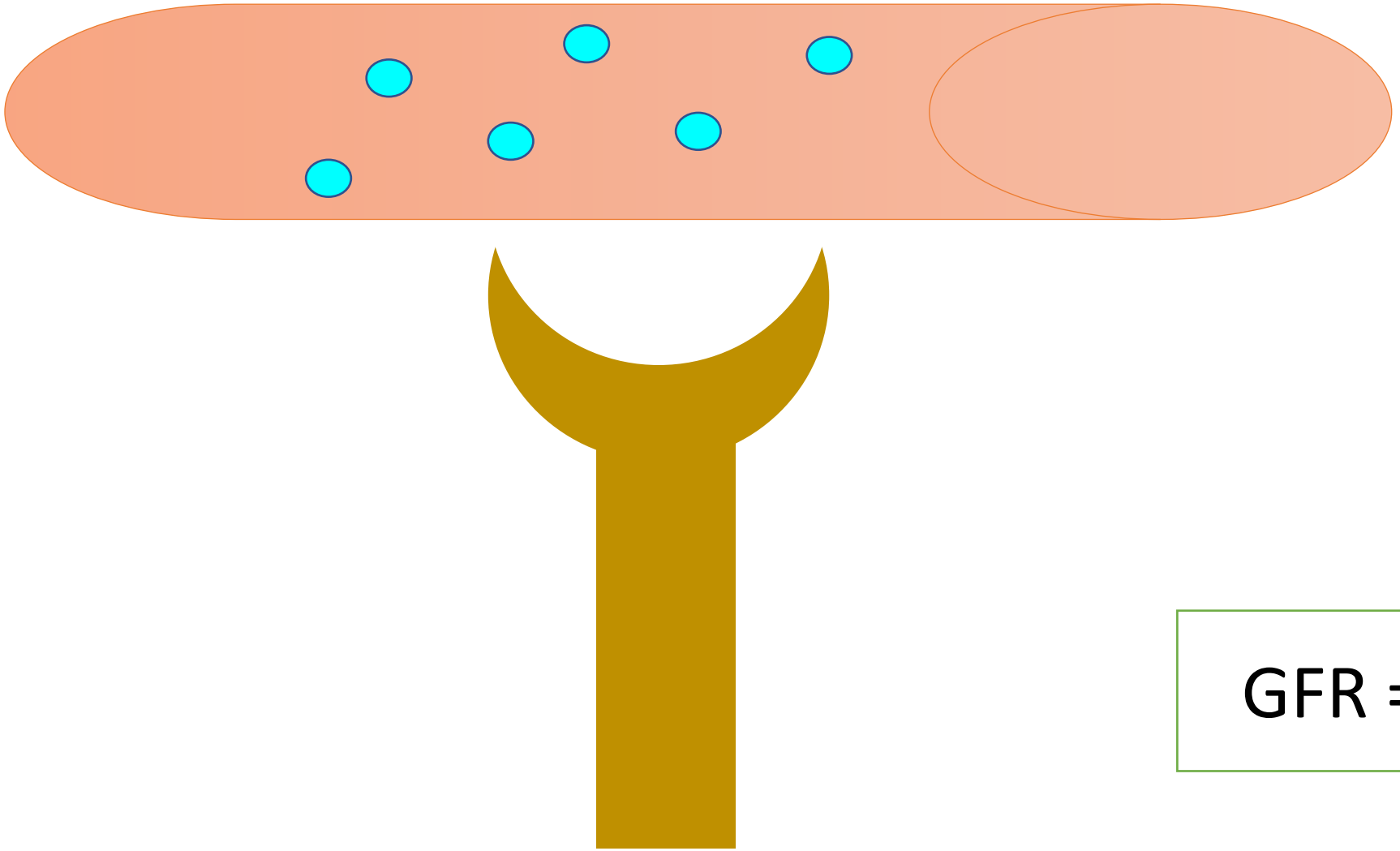


GFR = 4/2 = 2.

1. GFR – Clearance test

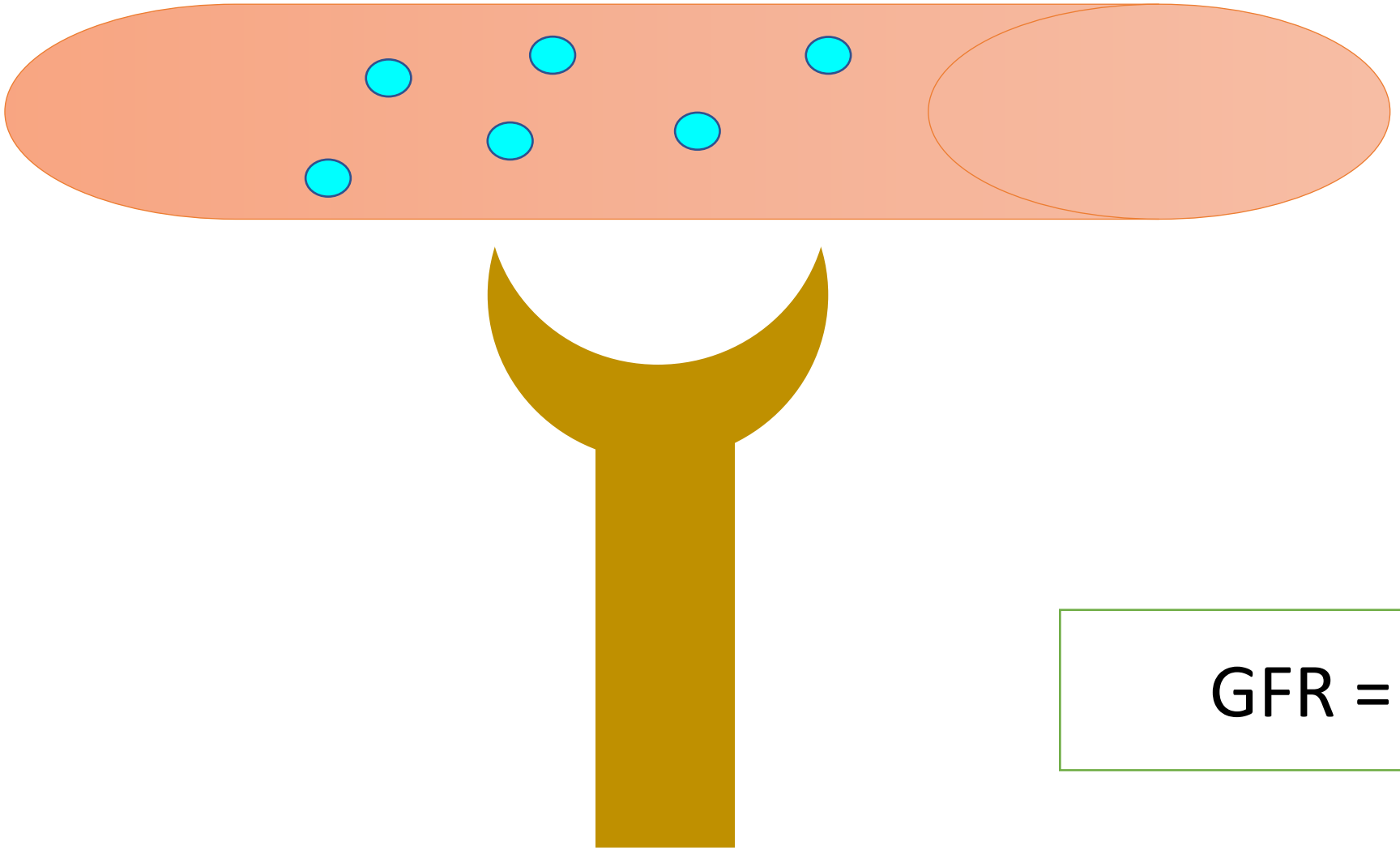
- **Clearance:** It is defined as the volume of blood or plasma completely cleared of a substance per unit time.

1. Glomerular function – Clearance test



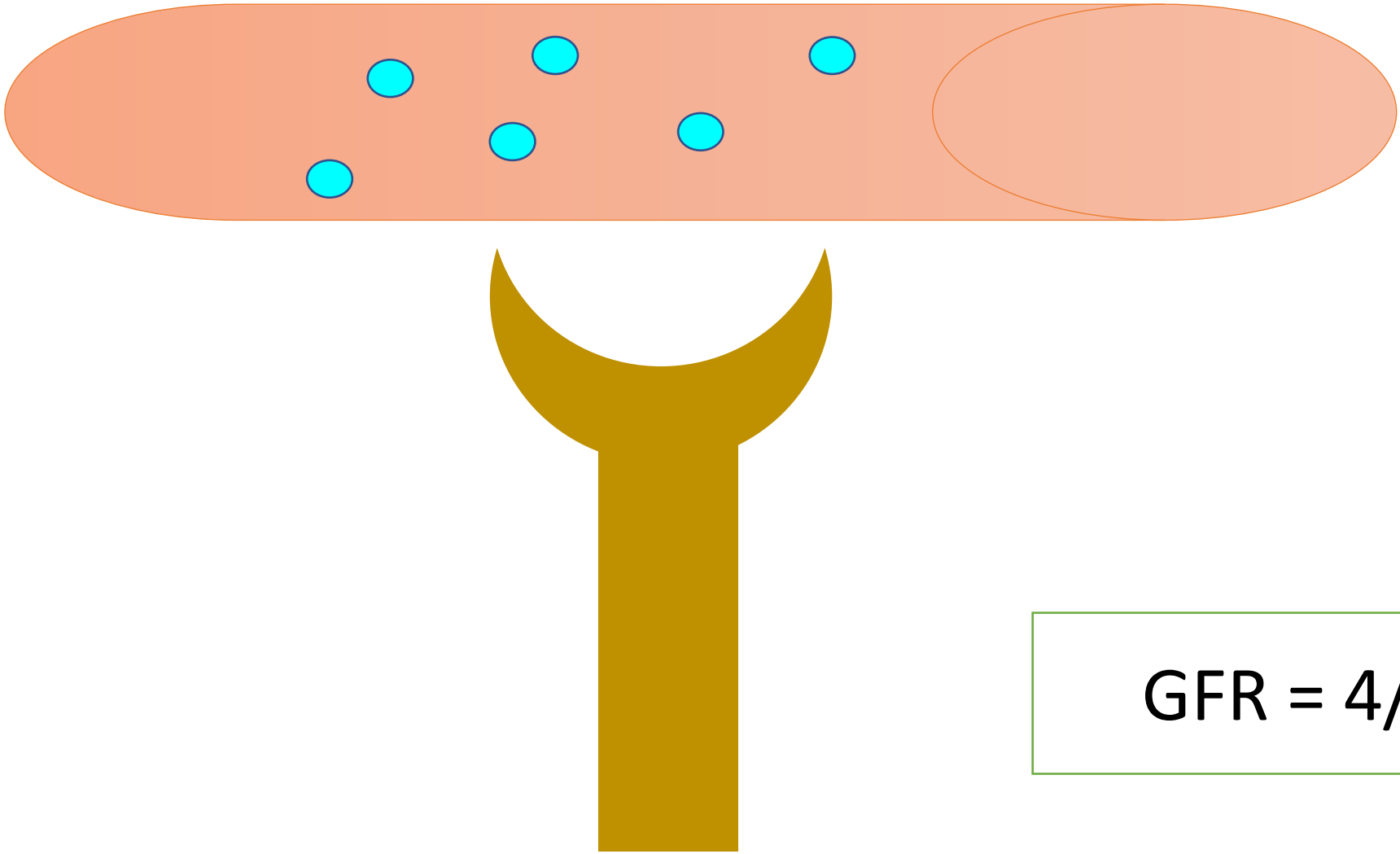
GFR = 4/2 = 2.

1. Glomerular function – Clearance test



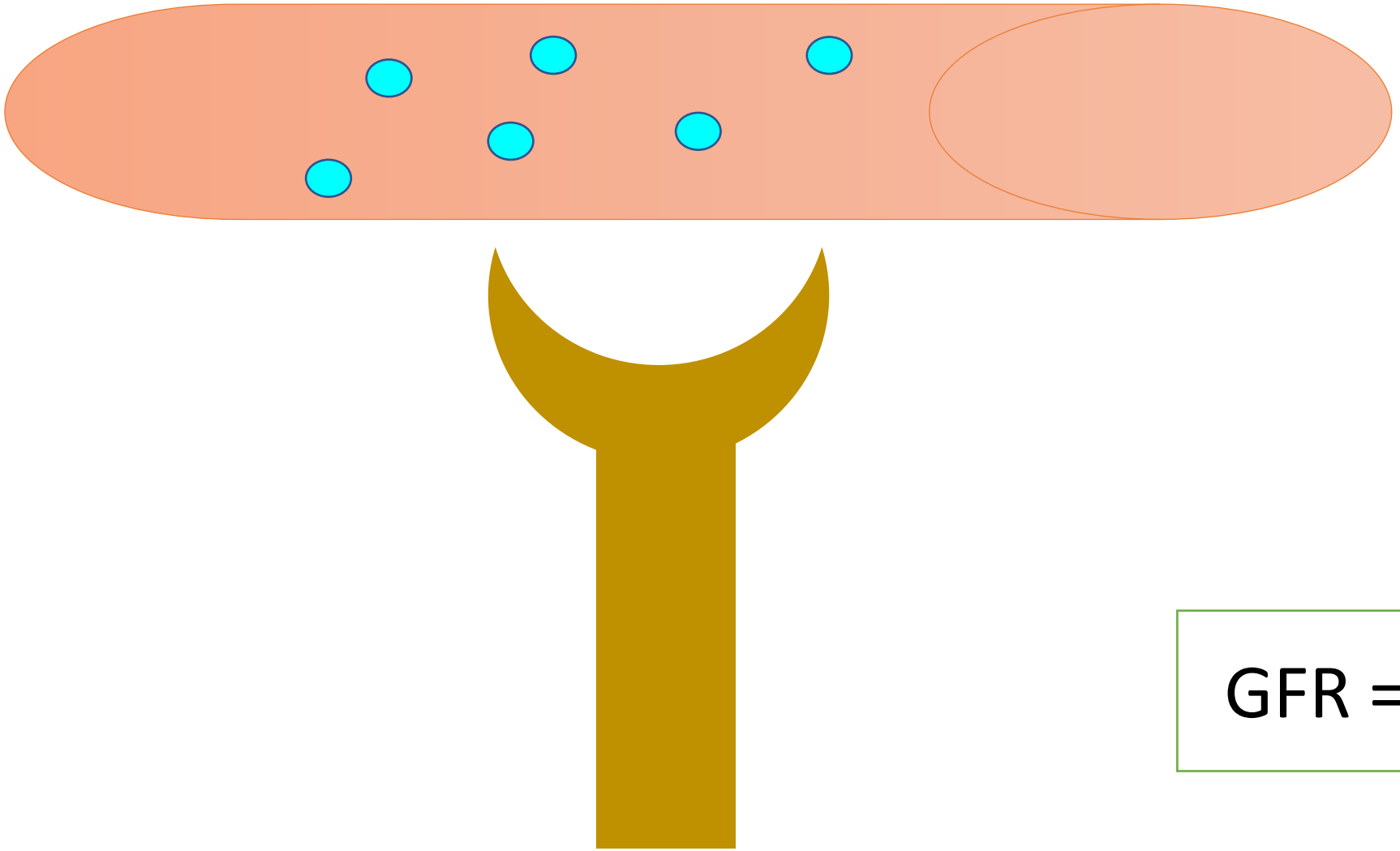
GFR = 3/3 = 1.

1. Glomerular function – Clearance test



GFR = 4/2 = 2.

1. Glomerular function – Clearance test



GFR = 2/4 = 0.5.

1. GFR – Clearance test

Creatinine Clearance :

i. Serum creatinine = 0.9 mg/dL.

(Reference value: serum creatinine = 0.7 – 1.4 mg/dL)

ii. Urine volume:

- Urine Volume in 1hr (60min)→ 84ml.
- Urine Volume in 1 min → 1.4 ml/min.

iii. Urine creatinine:

- Creatinine in 1hrs urine sample → 45 mg/dL.

1. GFR – Clearance test

Creatinine Clearance :

- Creatinine clearance = (U×V)/P.
  - U → Urinary creatinine.
  - V → Urinary flow in ml/min.
  - P → Plasma creatinine.

  Answer = (45 × 1.4) / 0.9 = 70 ml/min.

1. GFR – Clearance test

Creatinine Clearance :

- Corrected Creatinine clearance = (U × V × 1.73)/P.
  - U → Urinary creatinine.
  - V → Urinary flow in ml/min.
  - P → Plasma creatinine.

  Answer=(45×1.4×1.73)/0.9 = 121 ml/min/1.73 sq meter.

  GFR by creatinine reference value = 95 – 115 ml/min/1.73 sq meter.

1. GFR – Clearance test

1. **eGFR by creatinine:**
    1. CKD-EPI equation.
    2. MDRD formula.

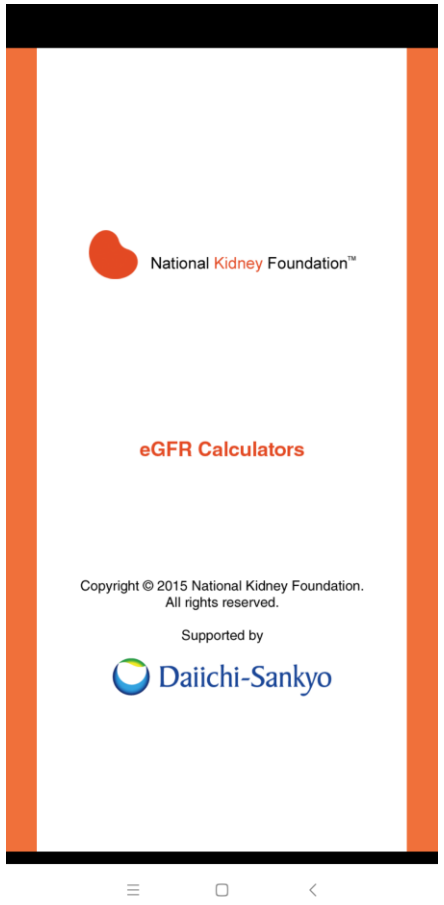
$$\text{eGFR (ml/min/1.73 m}^2\text{)} = 186 \times (\text{creatinine}/88.4)\text{-}1.154 \times (\text{Age}) - 0.203 \times 0.742 \text{ (if female)}$$

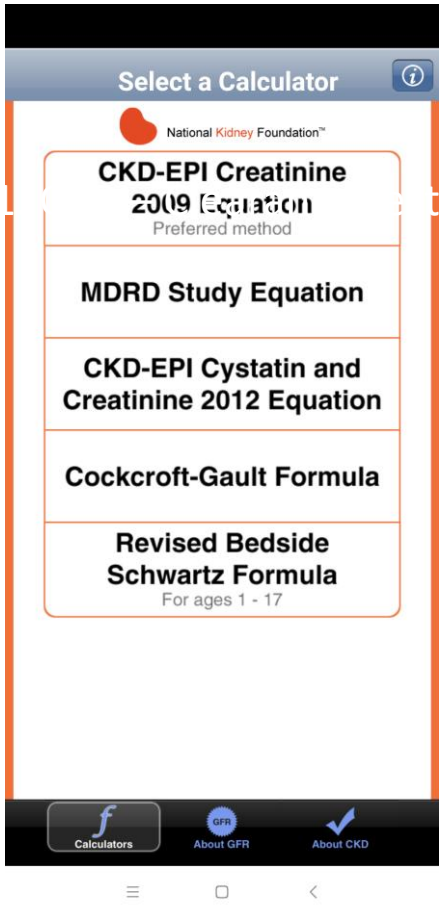
3. Cockcroft – Gault formula.

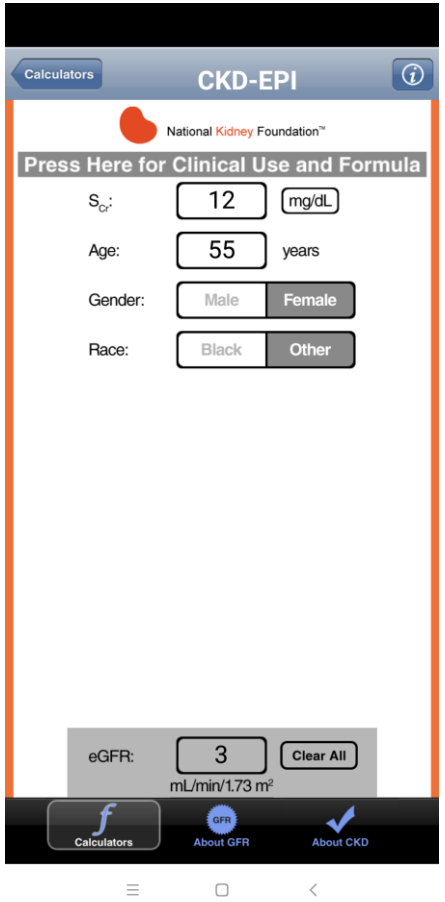
$$\text{Ccr} = (140\text{-age in years}) \times \text{weight in kg (0.85 in females)}/72 \times \text{plasma creatinine in mg/dL}$$

1. GFR – Clearance test

- eGFR:







1. GFR – Clearance test

- eGFR:

GFR categories (ml/min/1.73 m^2) description and range			
	G1	Normal or high	≥90
	G2	Mildly decreased	60–89
	G3a	Mildly to moderately decreased	45–59
	G3b	Moderately to severely decreased	30–44
	G4	Severely decreased	15–29
	G5	Kidney failure	<15

1. GFR – Clearance test

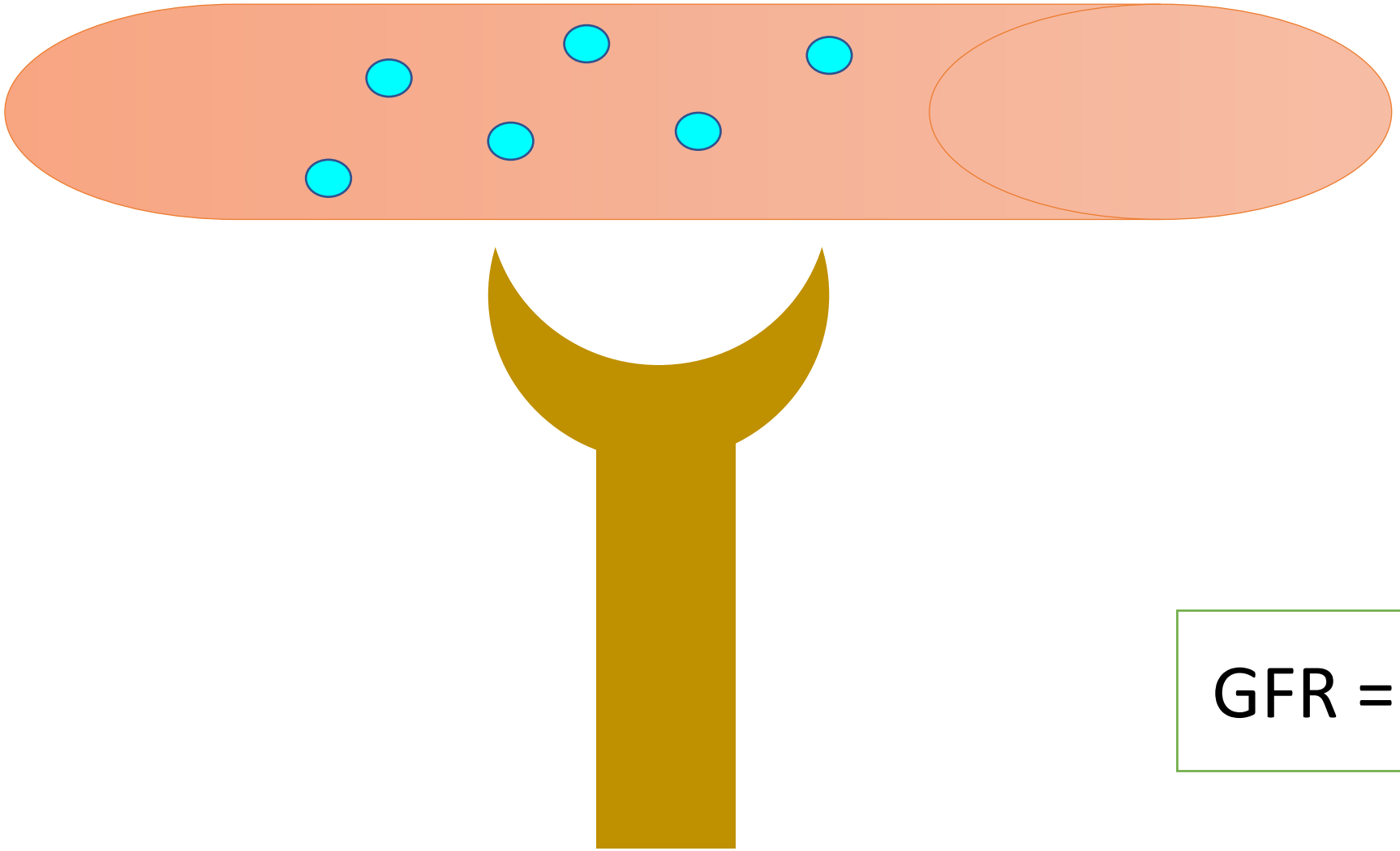
1. **Advantage of Creatinine Clearance:**

   - Diet will not interfere.
   - There is no diurnal variation.

2. **Disadvantage of Creatinine Clearance** :

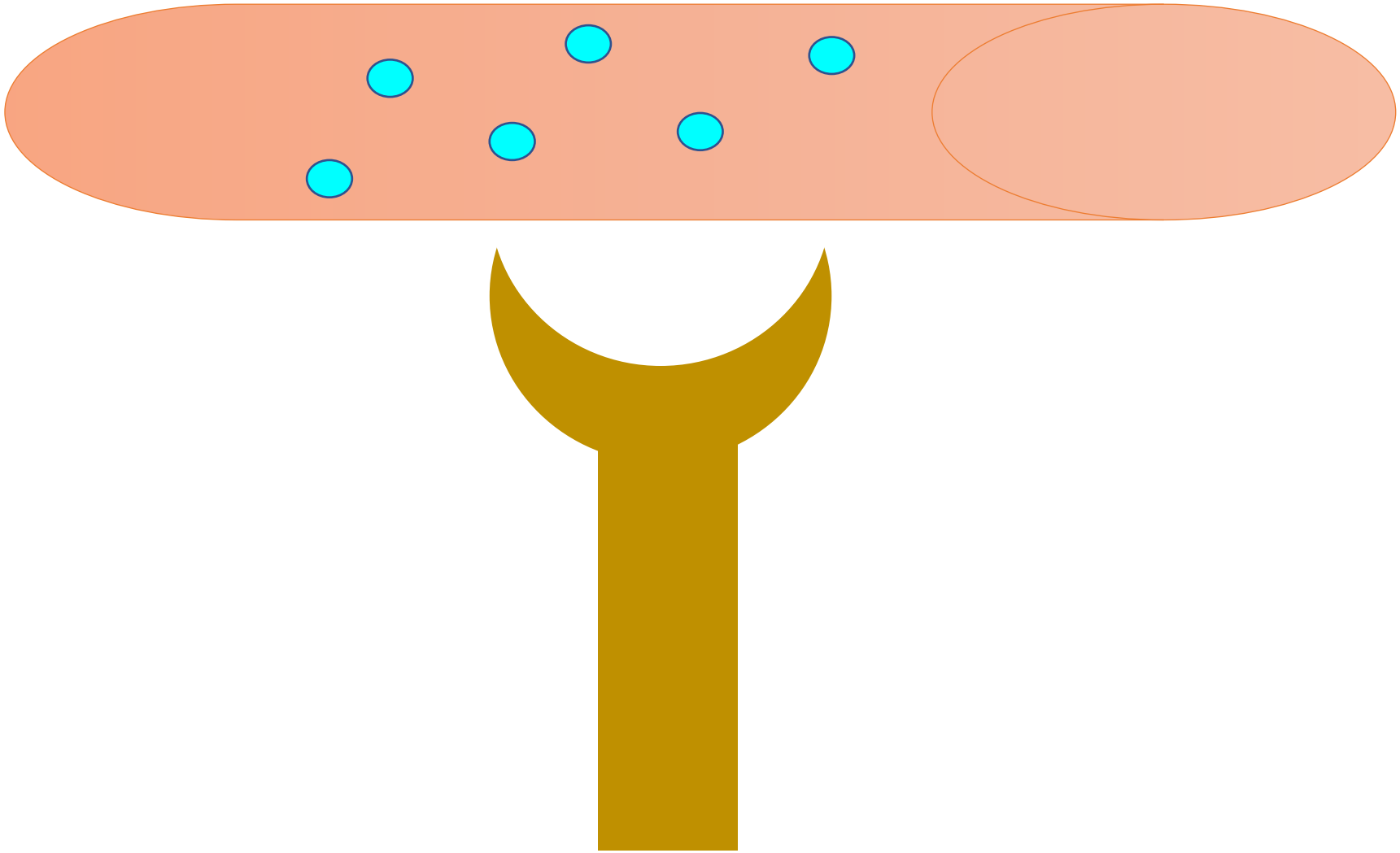
   - Early damage to kidney will not be detected.

1. Glomerular function – Clearance test

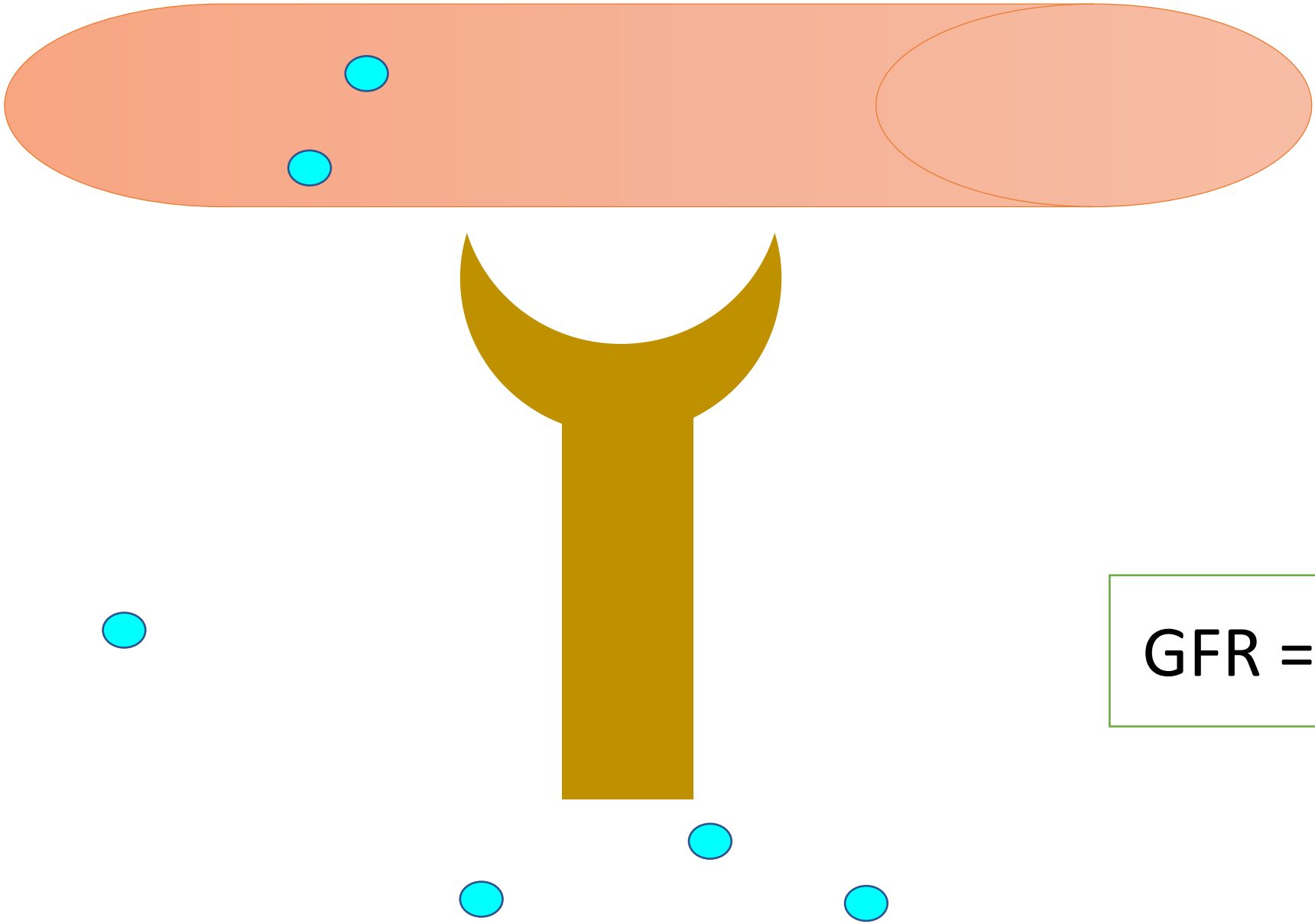


GFR = 4/2 = 2.

1. Glomerular function – Clearance test



1. Glomerular function – Clearance test



GFR = 3/3 = 1.

1. GFR – Urea

- Urea is reabsorbed by renal tubules and it is depended on renal flow rate.
- Maximum urea clearance = 75 ml/min.
- Standard urea clearance = 54 ml/min.

1. GFR – Clearance test

1. **<u>Ideal marker to measure GFR by clearance:</u>**

    1. Should be freely filtered across capillary wall.
    2. Should not be secreted or reabsorbed by renal tube.
    3. Should not be altered by diet.

1. GFR – Cystatin - C

1. Advantage of using cystatin C:
    1. No diurnal variation.
    2. Freely filtered by glomeruli.
    3. Not secreted by renal tubules.

2. Tubular function

1. Specific gravity of urine.
2. Concentration Test.
3. Dilution Tests.
4. Osmolality.
5. Urinary Acidification.

2. Tubular function

1. <u>Specific gravity of urine:</u>

   - Specific gravity depends on the concentration of solutes in the urine.
   - Normal urine specific gravity is 1.015-1.025.
   - In renal damage specific gravity will decrease.

2. Tubular function

2. Concentration Test:

- The patient is allowed no food or water after a meal at 6 pm.
- The next day at 7 AM, the bladder is emptied and specimen is discarded.
- A second specimen is collected at 8 am and the specific gravity is measured.

2. Tubular function

2. <u>Concentration Test:</u>

- In normal subjects Specific gravity will be increased > 1.025.
- As the disease progresses the urine specific gravity is fixed at and around 1.010. It is then called **isosthenuria**.

2. Tubular function

3. Dilution Tests:
   - The patient is not allowed to drink any water after midnight.
   - Bladder is emptied at 7 am and a water load is given (1200 ml over the next 30 minutes).
   - Hourly urine samples are collected for the next 4 hours separately.

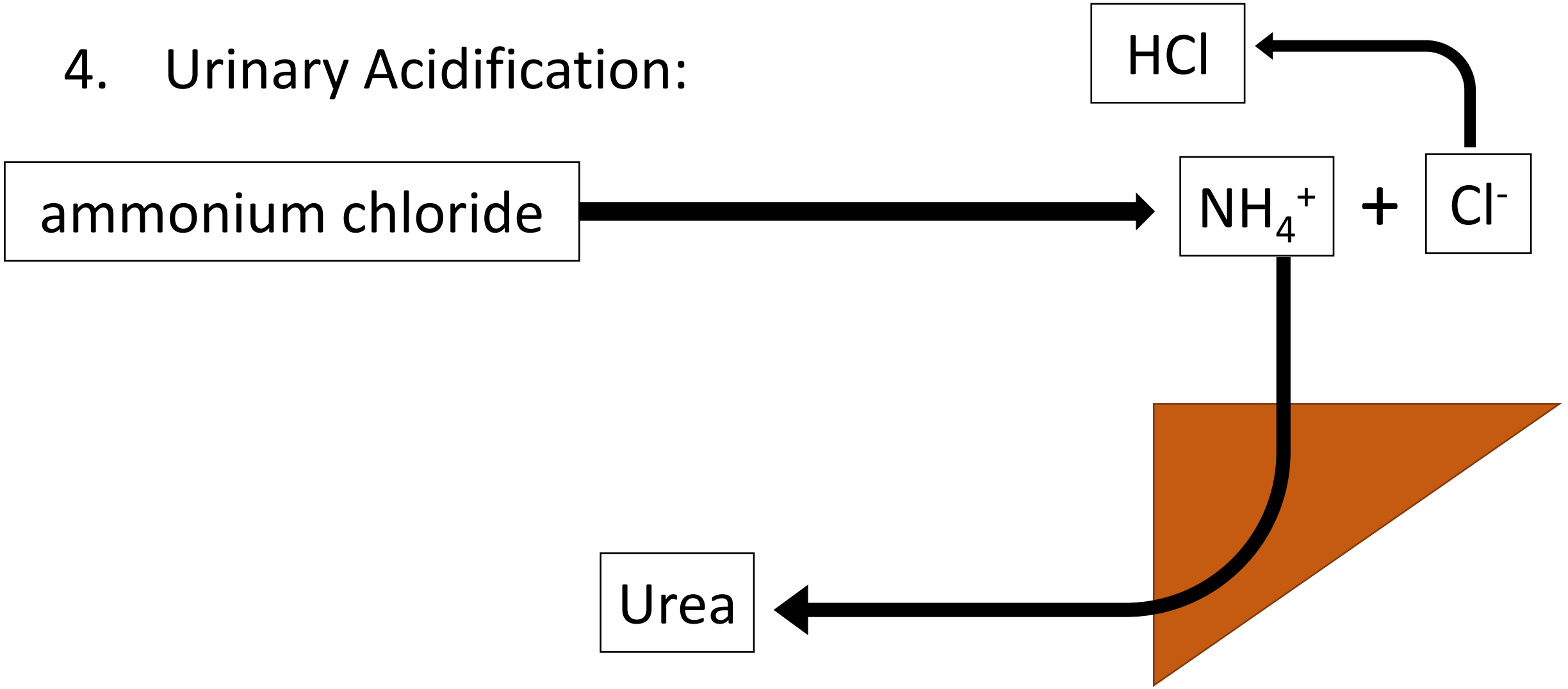
2. Tubular function

3. Dilution Tests:

- Volume, specific gravity and osmolality of each sample are measured.
- A normal person will excrete almost all the water load within 4 hours and the specific gravity of at least one sample should fall to 1.003 and osmolality to 50 milliosmol/kg.

2. Tubular function

4. Urinary Acidification:



2. Tubular function

5. Urine Osmolality:

- Urine Osmolality = 2 × (Urine Na + Urine K)+ Urine urea/6.
- Reference value:
  - Urine Osmolality = 60 - 1200 milliosmol/kg.
  - Urine Osmolality is decreased in diabetes insipidus.

II. Renal Function test

I. **To screen for kidney disease:**
   1. USG abdomen.
   2. Complete urine analysis.
   3. Serum urea and creatinine.

II. **To assess renal function:**
   1. Glomerular function.
   2. Tubular function.

III. Test to screen for complications.

II. Renal function test

Excretion

Metabolic waste

Urea
Creatinine
Uric acid

Hypertension

Potassium

Cardiac arrest

H^+ ion

Acid base disorder

II. Renal function test

Production.

- Vitamin D deficiency
- Anaemia

III. Interpretation of renal function test

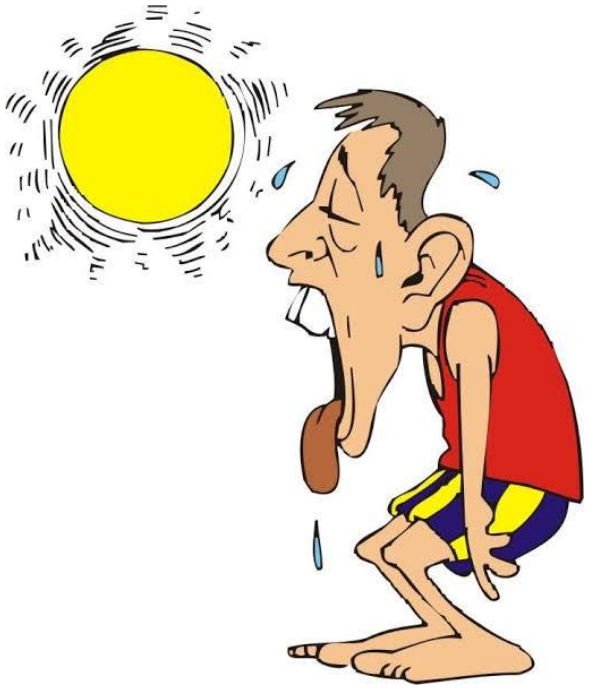
- Step – 1: When to do renal profile.
- Step – 2: Is there is a renal damage.
- Step – 3: Acute or chronic.
- Step – 4: Prerenal, renal or postrenal.

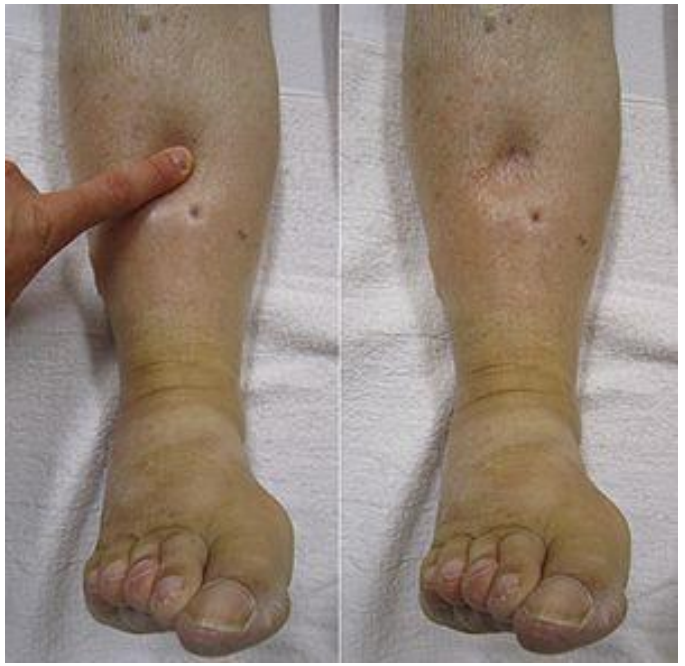
III. Interpretation of renal function test

- Step – 5: Magnitude of renal damage.
- Step – 6: Investigation related to complications.
- Step – 7: Dialysis need or not ?

III. Interpretation of renal function test

- Step – 1: When to do renal profile.
  - ➢ Symptomatic patients:





  - ➢ Asymptomatic patients:

III. Interpretation of renal function test

- Step – 1: When to do renal profile.
  - ➢ Symptomatic patients:
    - ✓ Dehydration, hematuria, polyuria, oliguria, anuria, swelling of legs and breathlessness.
  - ➢ Asymptomatic patients:
    - ✓ Before starting medications.
    - ✓ Before any interventional procured.

III. Interpretation of renal function test

Step – 2: Is there is a renal damage.

- Elevated urine protein and albumin.

Reference value:

- 24hrs Urine protein < 150 mg/day.
- Urine microalbumin < 30 mg/dL

III. Interpretation of renal function test

- Step – 3: Acute or chronic.
  - Based on the duration of the symptoms and rate of eGFT reduction.

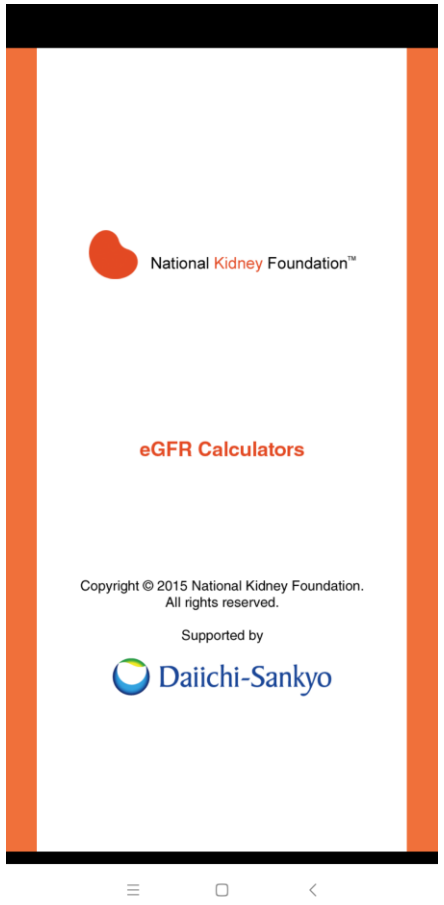
III. Interpretation of renal function test

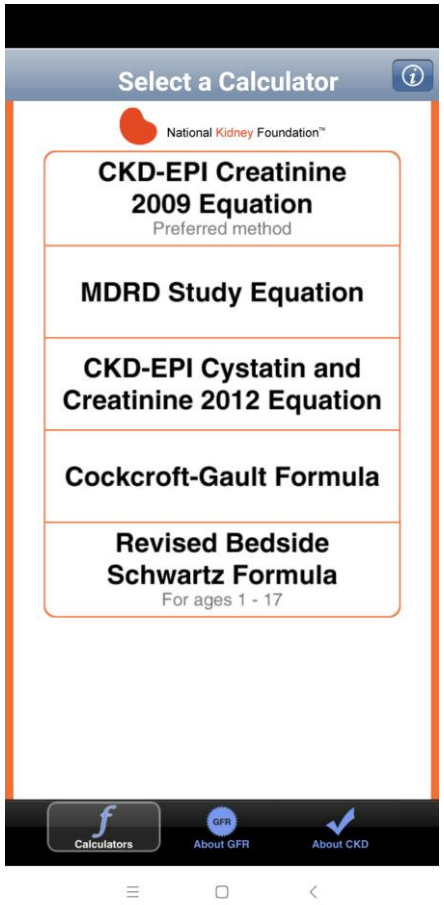
- Step – 4: Prerenal, renal or postrenal.

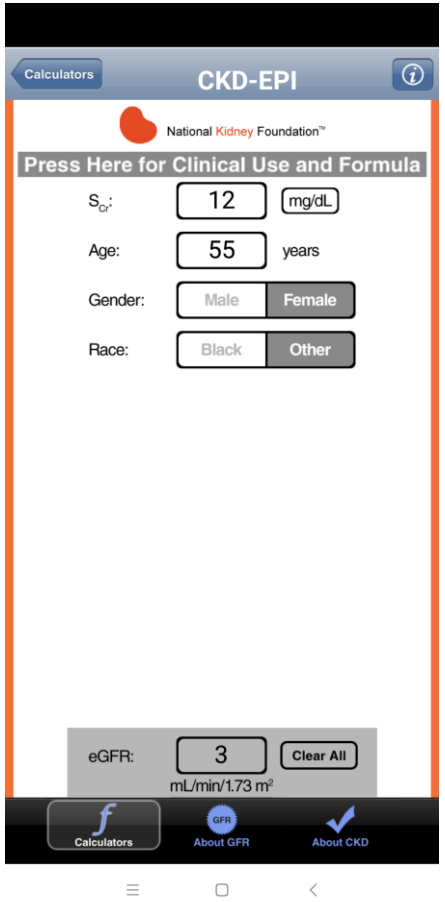
	S. urea	BUN	S. creatinine	Urea: Creatinine ratio	BUN: Creatinine ratio
NORMAL	20 - 40	6 - 20	0.7 – 1.4	20 - 40	6 - 20
Prerenal	↑ ↑ ↑		N	> 40	> 20 :1
Renal	↑ ↑		↑ ↑ ↑	< 20	<10 :1
Postrenal	↑ ↑ ↑		↑ ↑ ↑	20 - 40	10-20 :1

III. Interpretation of renal function test

- Step – 5: Magnitude of renal damage.
  - eGFR:







III. Interpretation of renal function test

- Step – 5: Magnitude of renal damage.
  - eGFR:

GFR categories (ml/min/1.73 m^2) description and range			
	G1	Normal or high	≥90
	G2	Mildly decreased	60–89
	G3a	Mildly to moderately decreased	45–59
	G3b	Moderately to severely decreased	30–44
	G4	Severely decreased	15–29
	G5	Kidney failure	<15

III. Interpretation of renal function test

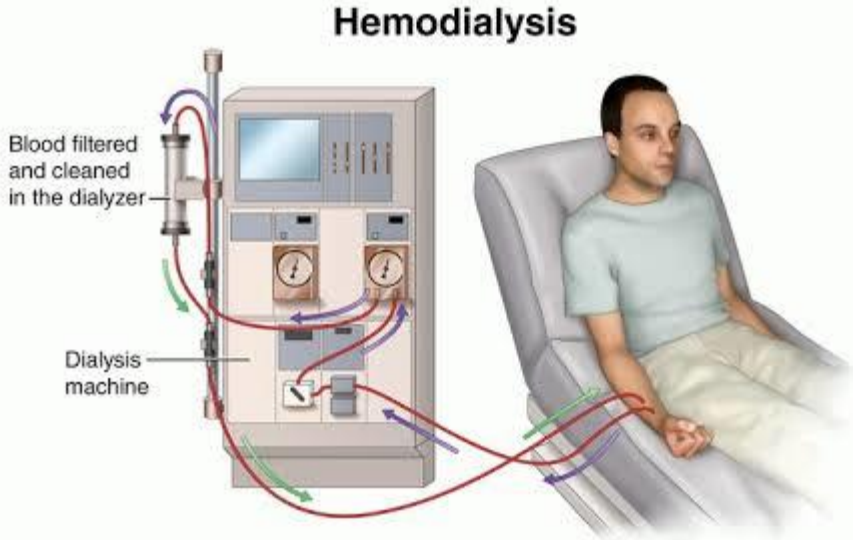
- Step – 6: Investigation related to complications:.
  - Serum uric acid.
  - Serum electrolyte.
  - Blood pressure.
  - ECG.
  - Arterial blood gas analysis.
  - Serum vitamin D concentration.
  - Serum calcium.
  - Serum phosphorus.

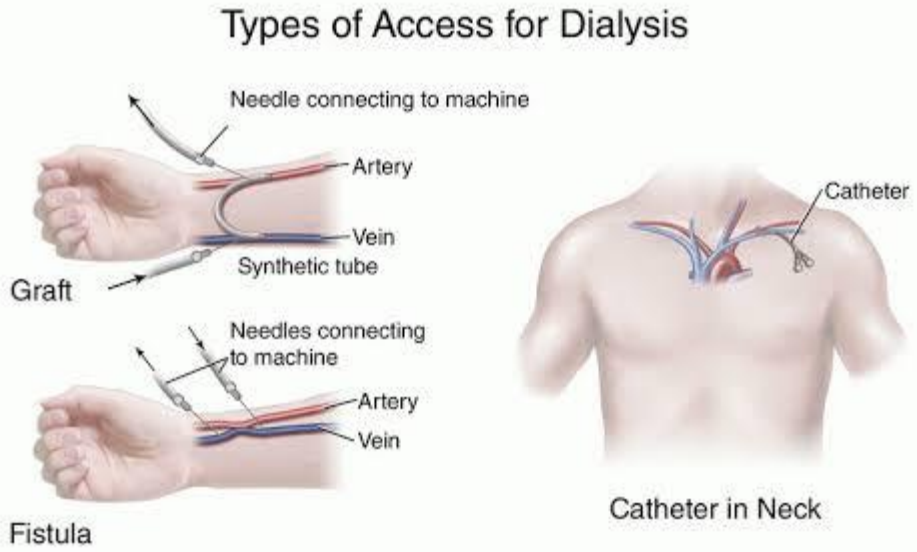
III. Interpretation of renal function test

- Step – 6: Investigation related to complications:
  - Complete blood count.
  - Total protein & albumin.

III. Interpretation of renal function test

- Step – 7: Dialysis need or not.
  1. Volume overload .
  2. Hyperkalemia.
  3. Acidosis.
  4. Severe complications of uremia.





VELAMMAL MEDICAL COLLEGE
HOSPITAL AND RESEARCH INSTITUTE
MADURAI - 625009

Department of Biochemistry

Report on Certificate course on

Analysis and Interpretation of Renal Function test

Topic: Analysis and Interpretation of Renal Function test

Date: 19.02.2019

Venue: Biochemistry Demonstration Room

Target audience: Faculties and M.B.B.S., students

Number of participants:

Event Report: The event started with the welcome address by Dr.P.K.Mohanty. Following that DR. A. Hariharan started the lecture on Renal function. He explained the list of test in renal function test and how to interpret the renal function test.

Outcome: Participants should be able to interpret the Renal function test.

Prof. T. THIRUNAVUKKARASU, M.D.,D.A.
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MADURAI - 625009

Velammal Medical College Hospital and Research Institute

Ref. No: VMCHRI/BIOCHEM/CC-4 Date: 01.03.2019

CIRCULAR

To

All Doctors

Certificate Course on Arterial Blood Gas analysis

Department of Biochemistry is organizing a certificate course on Arterial Blood Gas analysis on 22.03.2019 (Friday) between at 9.00 AM to 3.00 PM.

All Faculties are invited.

VICE PRINCIPAL

Dr. P.K. MOHANTY
Vice Principal
Velammal Medical College Hospital
and Research Institute
Madurai-625 009

Copy submitted to:

The Hon. Chairman

Copy to:

The Dean

Medical Superintendent

Chief Administration Officer

HOD, Biochemistry

All Clinical and Non-Clinical HODs

Velammal Medical College Hospital & Research Institute

Anuppanadi, Madurai - 625009

Department of Biochemistry

Certificate course on

Arterial Blood Gas analysis

Venue: Biochemistry Demonstration Room

Date: 22.03.2019

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

Agenda

Time	Topic	Speaker
9.00-9.15 AM	Welcome address	DR.P.K.Mohanty
9.15 – 9.45 AM	Pre test	DR.A.Hariharan
9.45 – 11.00 AM	Basics of acid base balance	DR.A.Hariharan
11.00 AM – 1.00 PM	Interpretation of Arterial Blood Gas Analysis	DR.A.Hariharan
1.00 PM – 2.00 PM	LUNCH	
2.00 – 2.30 PM	Post test	DR.A.Hariharan
2.30 – 3.00 PM	Feed back and Valediction	DR.P.K.Mohanty

Interpretation of Acid Base disorders

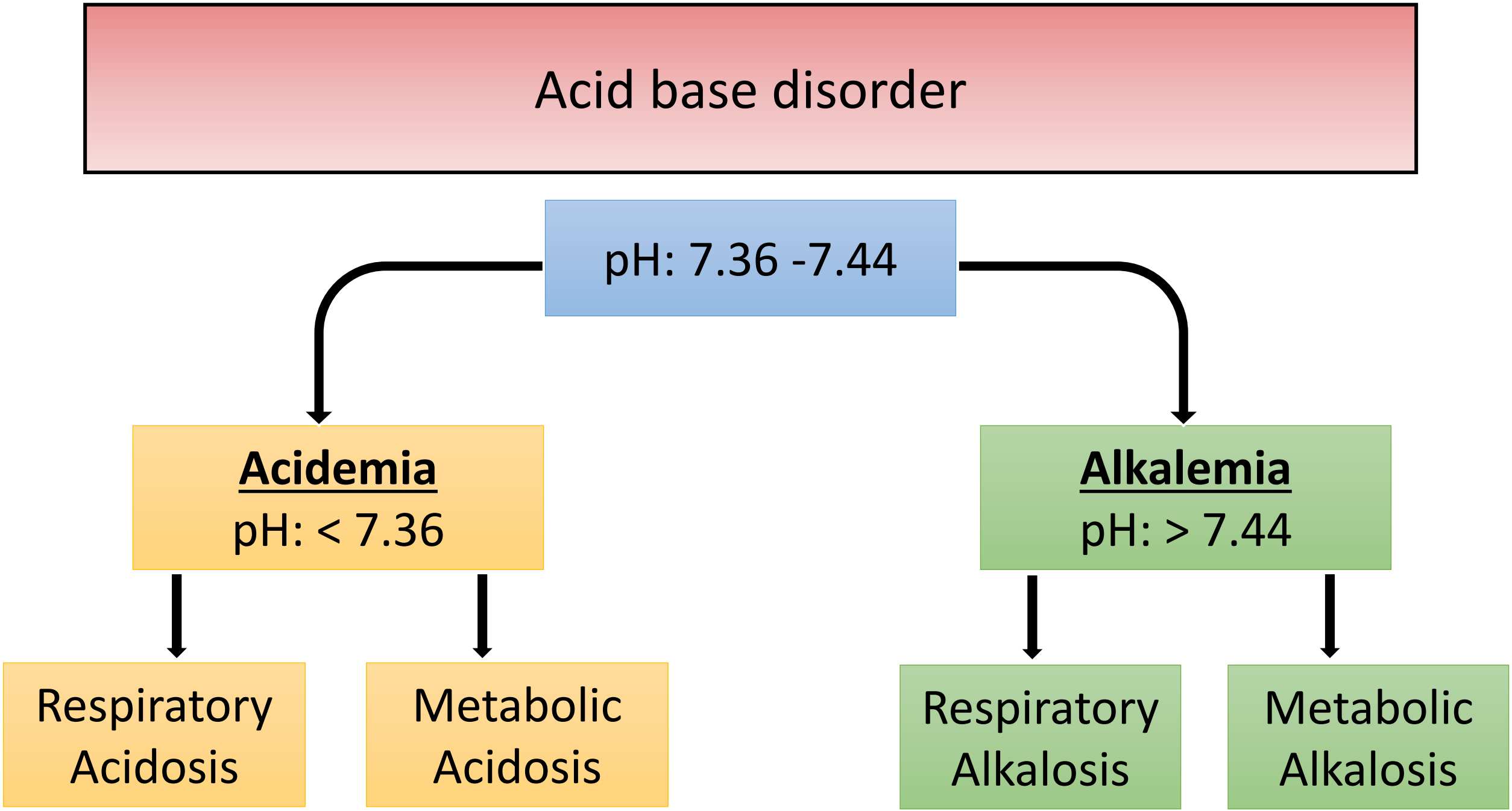
-Dr. A. Hariharan M.D.,
(Biochemistry)

Recap of acid base balance

- What is a acid?
- What is a base?
- What is pH?
- What is normal pH of blood?
- What is a strong acid and weak acid?
- What is a buffer?

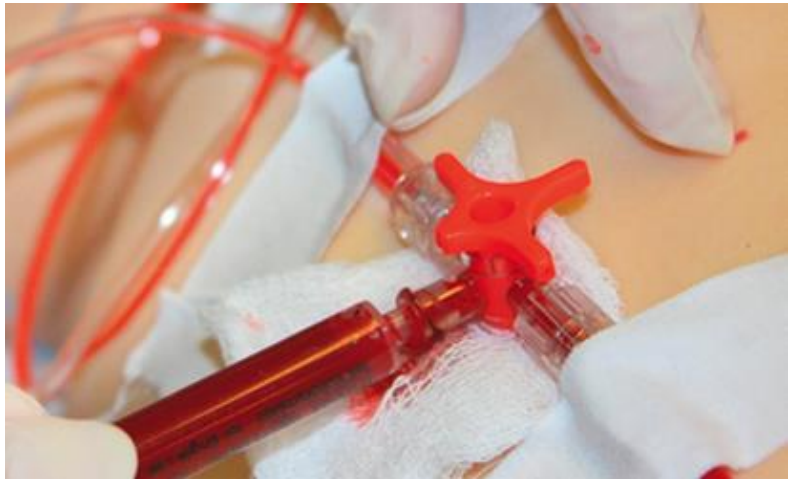
Recap of acid base balance

- Which is the major buffer present in ECF?
- What are the 3 major mechanism by which pH can be regulated?
- What is the normal value of pH, pCO_2, pO_2, HCO_3?


Acid base disorder
pH: 7.36 -7.44
Acidemia
pH: < 7.36
Alkalemia
pH: > 7.44
Respiratory Acidosis
Metabolic Acidosis
Respiratory Alkalosis
Metabolic Alkalosis

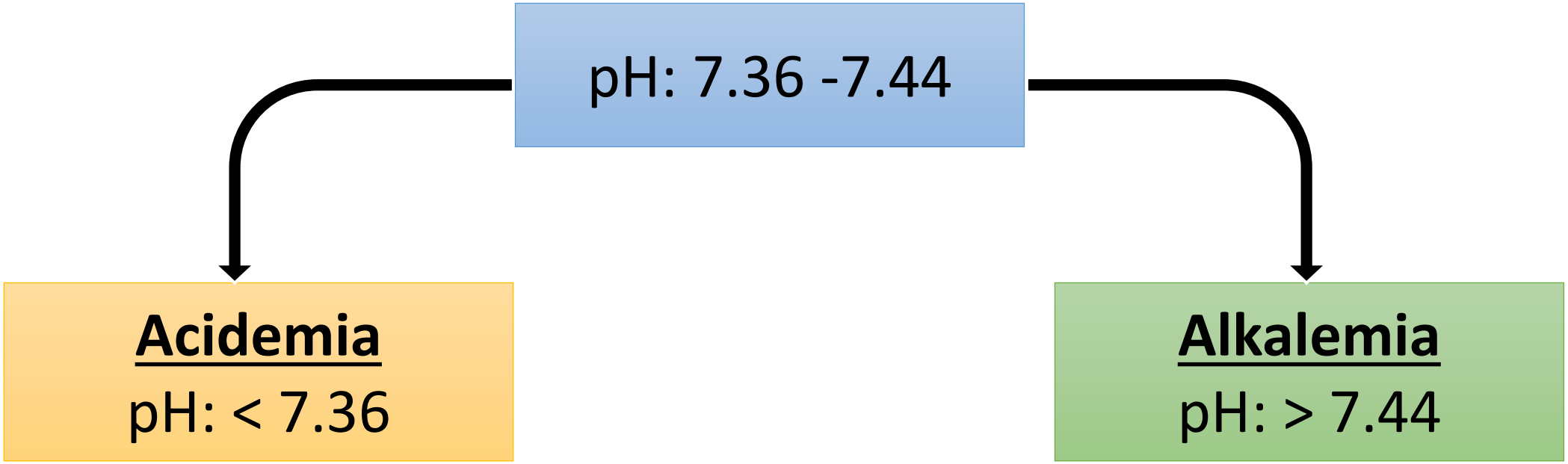
Step - 1

- Whether the sent sample for Arterial blood gas analysis is arterial or venous sample?
  - Arterial sample pO_2 > 70 mmHg.
  - Arterial sample is brighter than venous sample.



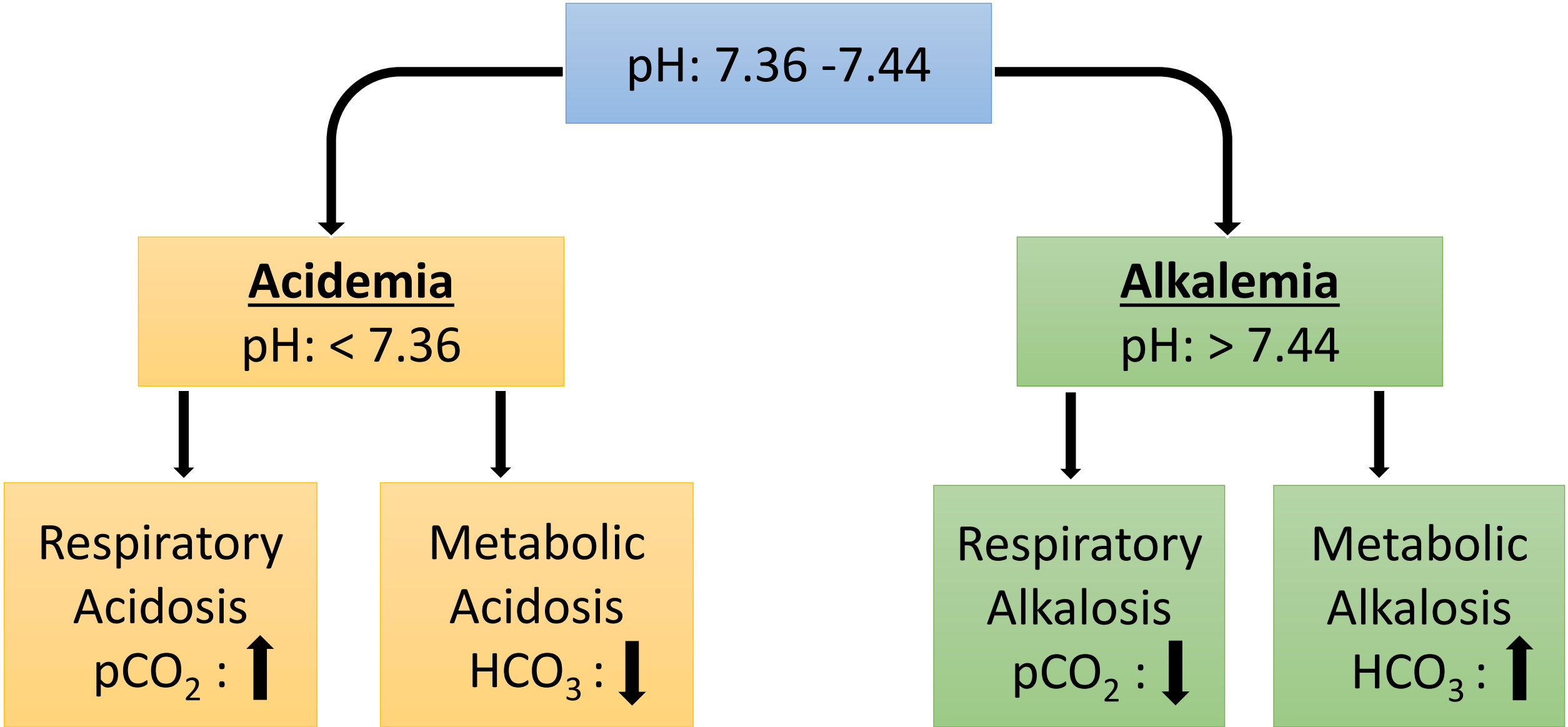
Step – 2

- Acedemia or Alkalemia.



Step - 3

- Identify the primary disorder.



Question: 1

- A 15 year old boy is brought from examination hall in apprehensive state with complain of tightness of chest.

pH: 7.54, HCO_3 : 21 mEq/L, $paCO_2$: 21 mmHg and

pO_2 : 100 mmHg.

Answer : Respiratory alkalosis

Question: 2

- Arterial blood gas analysis of a patient with CHF on frusemide is as follows:
- pH: 7.48, HCO_3 : 34 mEq/L, pCO_2 : 48mmHg,

  pO_2 : 98 mmHg.

Answer : Metabolic alkalosis

Question: 3

- Following sleeping pills ingestion, patient presented in drowsy state with sluggish respiration with respiratory rate 9/min.

pH: 7.1, HCO_3 :28 mEq/L, pCO_2 :80 mmHg, pO_2 : 70mmHg.

Answer : Respiratory acidosis

Question: 4

- A patient with poorly controlled type 1 DM missed his insulin for 3 days.

pH: 7.1, HCO_3 :8 mEq/L, pCO_2 :20 mmHg, pO_2 : 70mmHg.

Answer : Metabolic acidosis

Step - 4

- Compensation for primary disorder:

1. Metabolic Acidosis:

pH : Reduced.

HCO_3 : Decreased.

Compensation :

Decreased in pCO_2 → Respiratory alkalosis.

$pCO_2 = (HCO_3 \times 1.5) + 8.$

Step - 4

- Compensation for primary disorder:

2. Metabolic Alkalosis:

pH : Increased.

HCO_3 : Increase.

Compensation :

Increase in pCO_2 → Respiratory acidosis.

$pCO_2 = (HCO_3 \times 0.7) + 21.$

Step - 4

- Compensation for primary disorder:

3. Respiratory Acidosis:

pH : Reduced.

pCO_2 : Increase.

Compensation :

Increase in HCO_3 → Metabolic alkalosis.

Step - 4

- Compensation for primary disorder:

4. Respiratory Alkalosis:

pH : Increased.

pCO_2 : Decreased.

Compensation :

Decrease in HCO_3 → Metabolic acidosis.

Step - 4

	Acute	Chronic
Respiratory acidosis (for every 10 mmHg increase in pCO_2)	HCO_3 increased by 1 mmHg	HCO_3 increased by 4-5 mmHg
Respiratory alkalosis (for every 10 mmHg decrease in pCO_2)	HCO_3 decrease by 2 mmHg	HCO_3 decrease by 4-5 mmHg

Step - 4

Primary disorder	Compensation
Metabolic acidosis	Respiratory alkalosis
Respiratory acidosis	Metabolic alkalosis
Metabolic alkalosis	Respiratory acidosis
Respiratory alkalosis	Metabolic acidosis

Question - 5

- A 40 yr old male a known case of bronchial asthma came with complains of breathlessness for past 1 day and his arterial blood gas analysis shows,

pH: 7.33, pCO_2 :70mmHg, HCO_3 :27 mEq/dL, pO_2 :70 mmHg.

Answer : Compensated Respiratory acidosis

Question - 6

- A 56 year old man who smoked for many years develops cough with purulent sputum and was admitted to the hospital because of difficult in breathing for past 20 days. His arterial blood gas analysis shows,

pH: 7.20, pCO_2 :70mmHg, HCO_3 :27 mEq/dL, pO_2 :70 mmHg.

Answer : Uncompensated Respiratory acidosis

Question - 7

- A 5 year old child a known case of type 1 DM brought to hospital in unconscious state and her arterial blood gas analysis shows,

pH: 7.34, pCO_2 : 36 mmHg, HCO_3 :19 mEq/dL, pO_2 :70 mmHg.

Answer : Compensated Metabolic acidosis

Question - 8

- A 5 year old child a known case of type 1 DM brought to hospital in unconscious state and her arterial blood gas analysis shows,

pH: 7.34, pCO_2 : 40 mmHg, HCO_3 :18 mEq/dL, pO_2 :70 mmHg.

Answer : Uncompensated Metabolic acidosis

Question - 9

- A patient on ventilator for past 8 hours has following arterial blood gas value,

pH: 7.49, pCO_2 : 30 mmHg, HCO_3 :22 mEq/dL, pO_2 :91 mmHg.

Answer : Compensated Respiratory alkalosis

Question - 10

- A patient on ventilator for past 19 days has following arterial blood gas value,

pH: 7.49, pCO_2 : 20 mmHg, HCO_3 :20 mEq/dL, pO_2 :95 mmHg.

Answer : Uncompensated Respiratory alkalosis

Question - 11

- What is the primary acid base disorder if arterial blood gas analysis shows,
- pH: 7.58, pCO_2 : 42 mmHg, HCO_3 :30 mEq/dL, pO_2 :85 mmHg.

Answer : Compensated Metabolic alkalosis

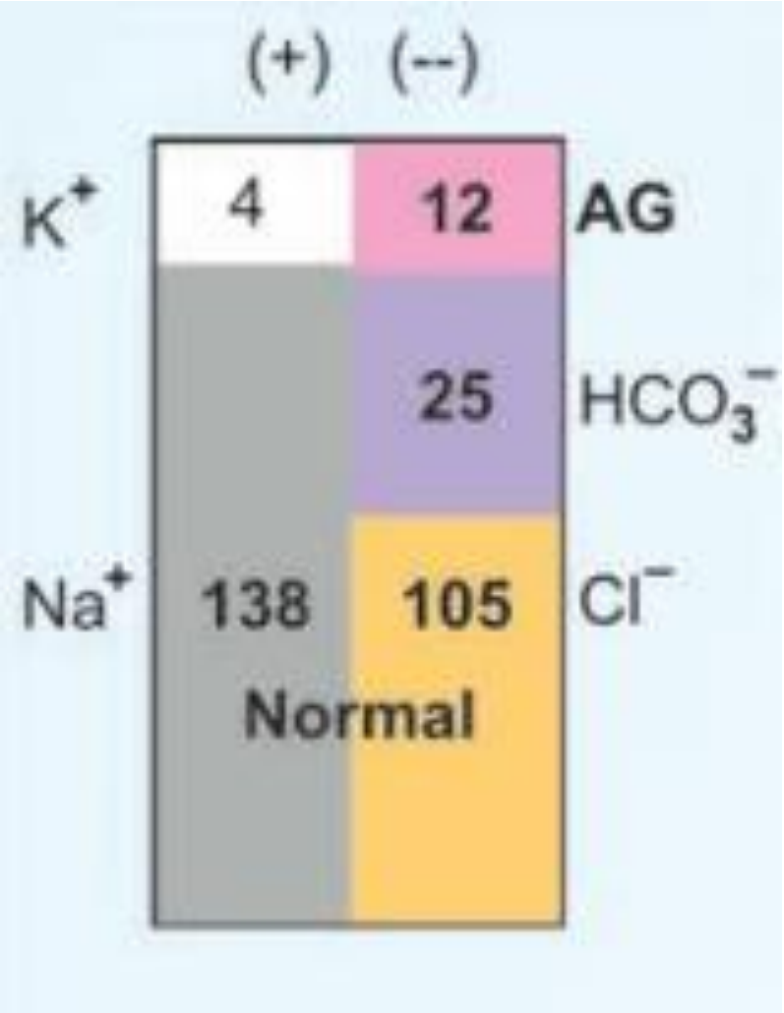
Question - 12

- What is the primary acid base disorder if arterial blood gas analysis shows,

pH: 7.86, pCO_2 : 40 mmHg, HCO_3 :33 mEq/dL, pO_2 :90 mmHg.

Answer : Uncompensated Metabolic alkalosis

Anion Gap



Anion Gap (AG)

- Unmeasured anion is called as anion gap.
- AG = $(Na^+ + K^+) – (Cl^- + HCO_3)$.
- Normal range = 12 – 16.
- Metabolic acidosis can be further subdivided into two type based on anion gap.
  - High anion gap metabolic acidosis.
  - Normal anion gap metabolic acidosis.

Question - 13

- A patient with poorly controlled type 1 DM missed his insulin for 3 days.

pH: 7.1, HCO_3 : 8 mEq/L, pCO_2 :20 mmHg, pO_2 : 70mmHg.

Na: 135, K =5, Cl: 106.

Answer : High anion gap Metabolic acidosis

Question - 14

- What is the primary acid base disorder if arterial blood gas analysis shows,

pH: 7.26, pCO_2 : 40 mmHg, HCO_3 :20 mEq/dL, pO_2 :90 mmHg.

Na: 135, Cl: 106, K =4.

Answer : Normal anion gap Metabolic acidosis

VELAMMAL MEDICAL COLLEGE
HOSPITAL AND RESEARCH INSTITUTE
MADURAI - 625009

Department of Biochemistry

Report on Certificate course on Arterial Blood Gas Analysis

Topic: Arterial Blood Gas analysis

Date: 22.03.2019

Venue: Biochemistry Demonstration Room

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

Number of Participants: 113

Event Report: The event started with the welcome address by Dr.P.K.Mohanty. Following that Dr. A.Hariharan started the lecture on Arterial Blood Gas Analysis. He explained the basics of acid base balance and steps in interpretation of arterial blood gas analysis.

Outcome: Participants should be able to interpret the arterial blood gas analysis.

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VELAMMAL MEDICAL COLLEGE
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MADURAI - 625009

Department of Orthopaedics

Report

Topic : Certificate course on Osteosarcoma Management

Date : 04.08.2018

Venue : Ortho OPD Demo Hall, VMCH & RI

Target Audience : CRRIs

Number of participants: 24

A certificate course on Osteosarcoma Management In Orthopaedics was organised by the Department of Orthopaedics to CRRIs on 04.08.2018. 24 CRRIs participated in the course. The program began by 08.30 am with a pretest followed by Introduction, Investigations, Surgical techniques, Complications Assessment & discussion. 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 Osteosarcoma Management. The gained idea about Osteosarcoma Management.

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VELAMMAL MEDICAL COLLEGE
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MADURAI - 625009

Department of Orthopaedics

Report

Topic : Certificate course on LBA- Causes & Management

Date : 22.04.2019

Venue : Ortho OPD Demo Hall, VMCH & RI

Target Audience : Prefinal year students

Number of participants: 25

A certificate course on LBA- Causes & Management in Orthopaedics was organised by the Department of Orthopaedics to Prefinal year students on 22.04.2019. 25 Prefinal year students participated in the course. The program began by 08.30 am with a pretest followed by Introduction, Causes, Investigations, Conservative & Surgical management, 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 LBA- Causes & Management. They gained an idea about LBA- Causes & Management.

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VELAMMAL MEDICAL COLLEGE
HOSPITAL & RESEARCH INSTITUTE

DEPARTMENT OF RADIODIAGNOSIS

IMAGING OF CRANIOFACIAL TRAUMA

CERTIFICATE COURSE

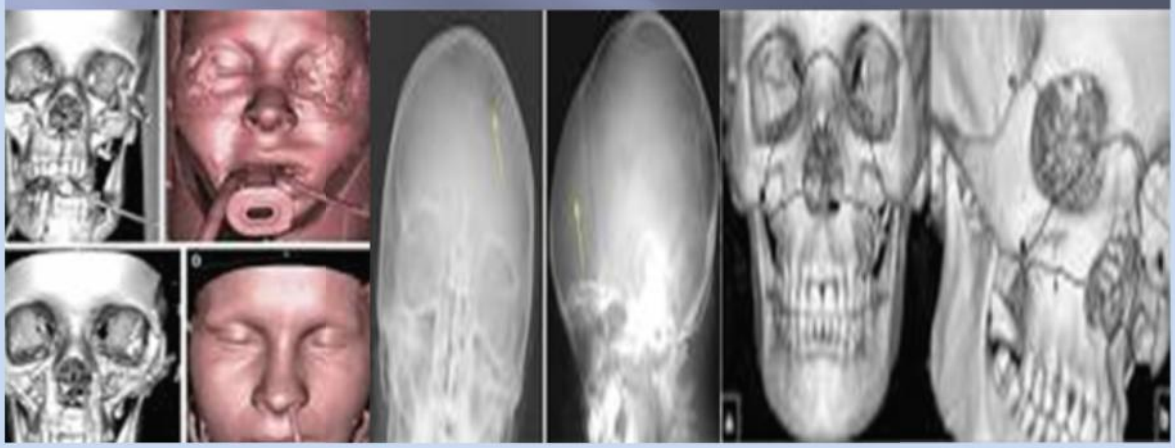
VELAMMAL MEDICAL COLLEGE HOSPITAL & RESEARCH INSTIUTE

IMAGING OF CRANIOFACIAL TRAUMA

CERTIFICATE COURSE

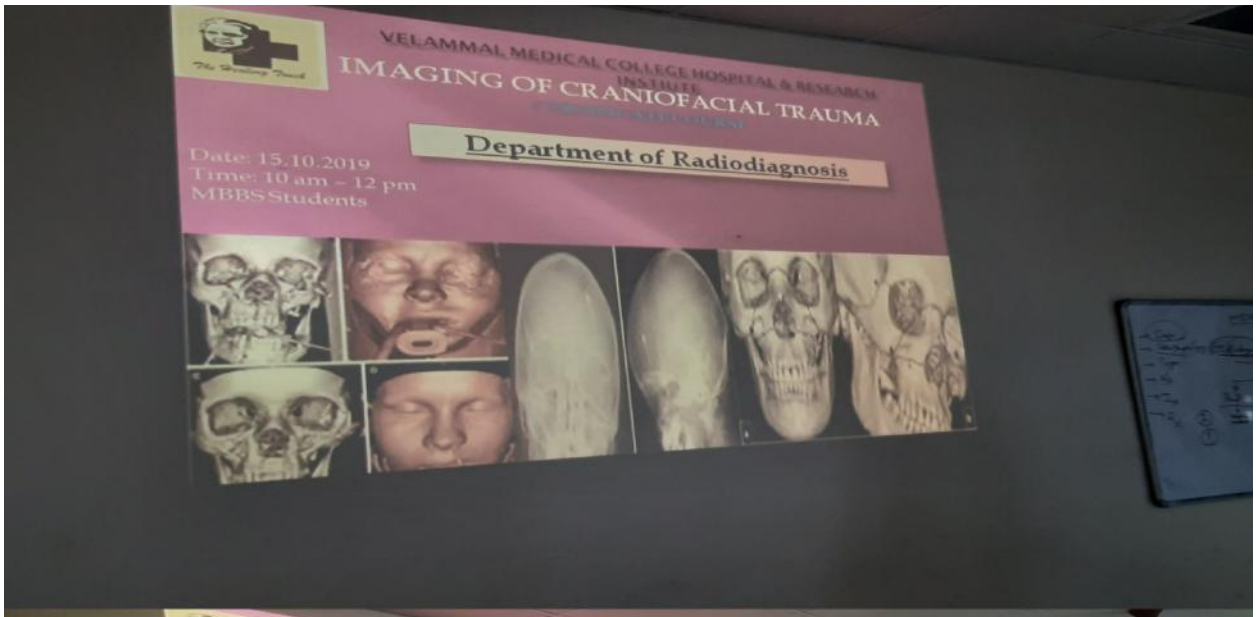
Department of Radiodiagnosis

Date: 15.10.2019
Time: 10 am – 12 pm
MBBS Students



DEPARTMENT OF RADIODIAGNOSIS
CERTIFICATE COURSE –CRANIOFACIAL TRAUMA
15.10.2020

S.No	Name	Mobile Number	Signature
1.	M. Vidhyasree	9344590737	
2.	Swastika Balaji	7339333611	
3.	Vathsala C	8825808372	
4.	R. SMRITI	7358215596	
5.	R. Sindudevi	8903084883	
6.	Swathi M	8608894784	
7.	Varsha S	9944403882	
8.	P Sandhiya	7094155939	
9	M. Sarika	8523982586	
10.	J. Sathana Jenifer	7397645111	
11.	R.K. Sharad Eswar	6382103374	
12.	T. Shasmila Nandhini	9597429059	
13.	M. Sriram	9361618143	
14.	Suba Hasini P	9789184858	
15.	B.T. Surya Krishna	9444041407	
16	Sushma IBS	9384644886	
17.	N. Suvesha	9003881884	
18	P. Swarna Gowri	6383951050	
19.	B.K. Thasrun	9840100400	
20.	S. Thayala Sundaram	8610657923	
21.	G.S.V. Rishi Rahul	9894894502	
22	Varshini K	8489569829	
23	S.A Vinudhaagaa	9790222846	
24	Yamini R	9150531299	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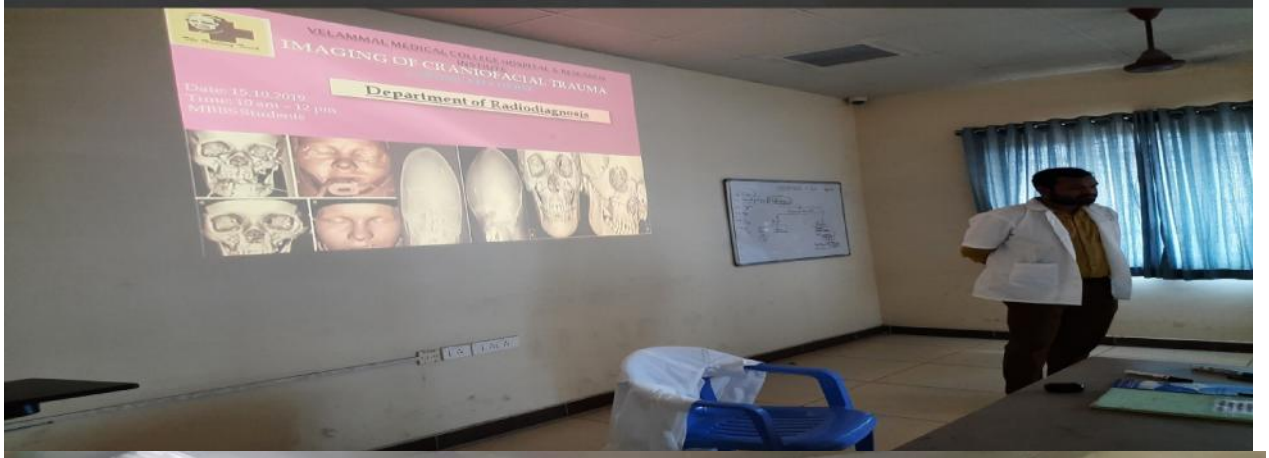
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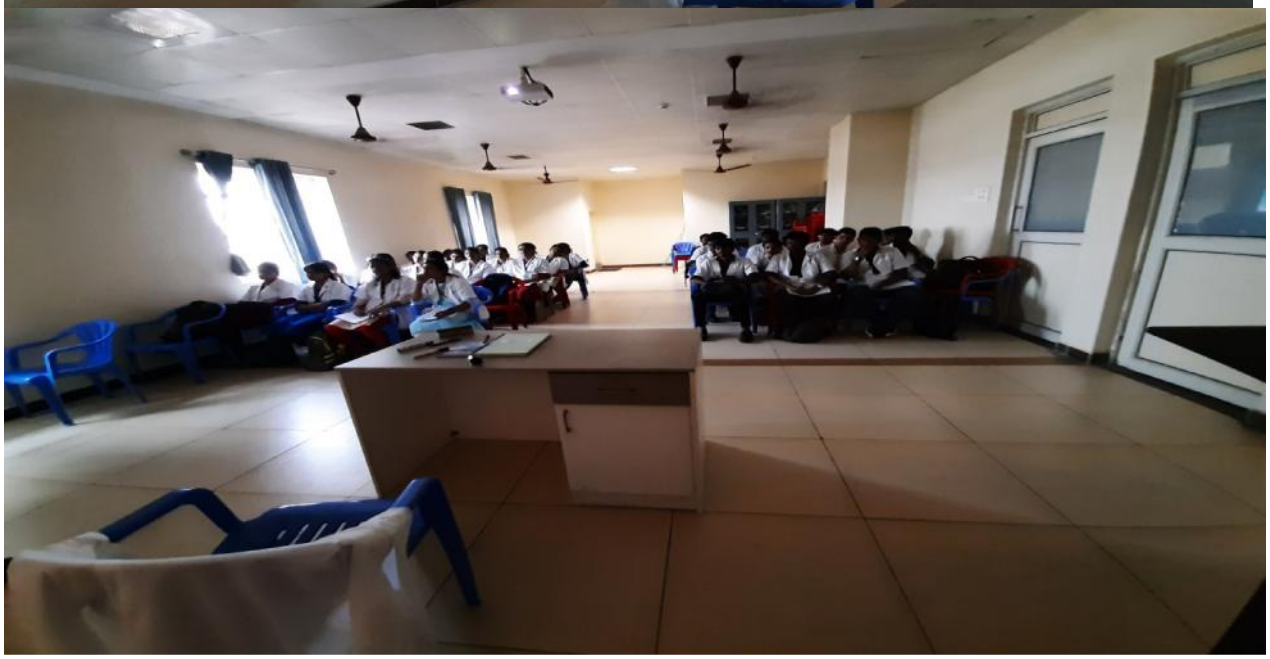
Department of Radiodiagnosis

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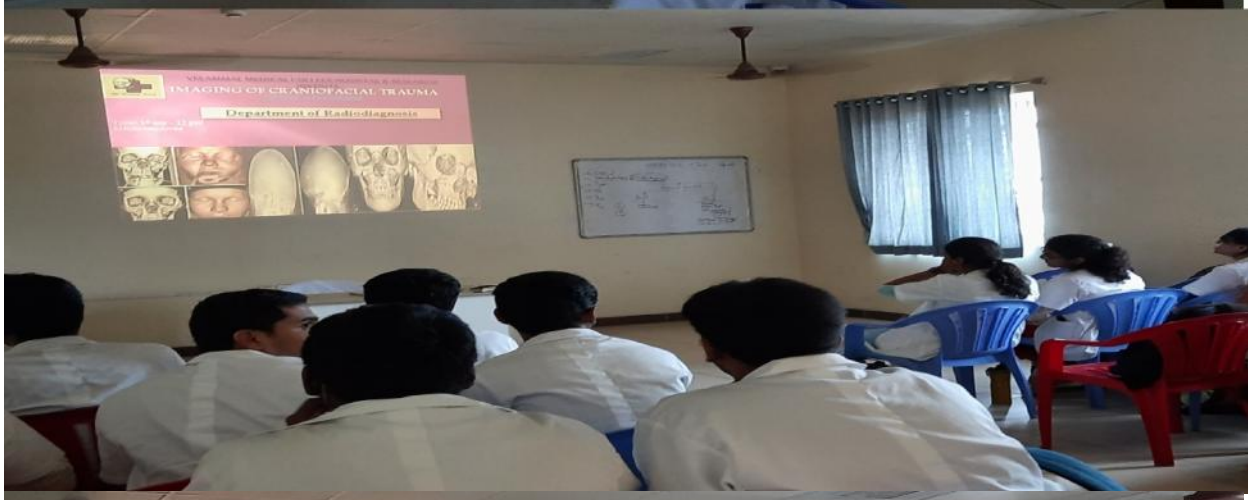
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Department of Radiodiagnosis


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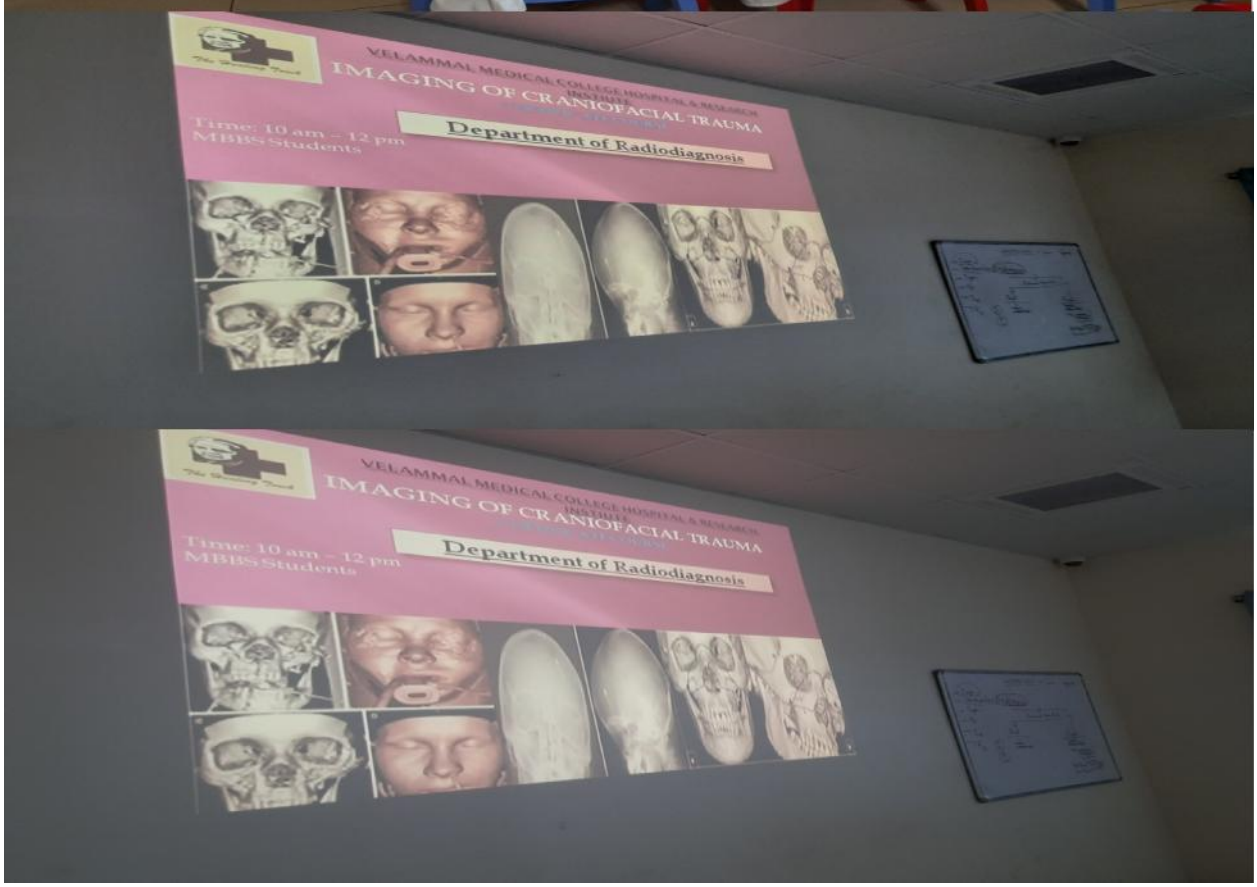
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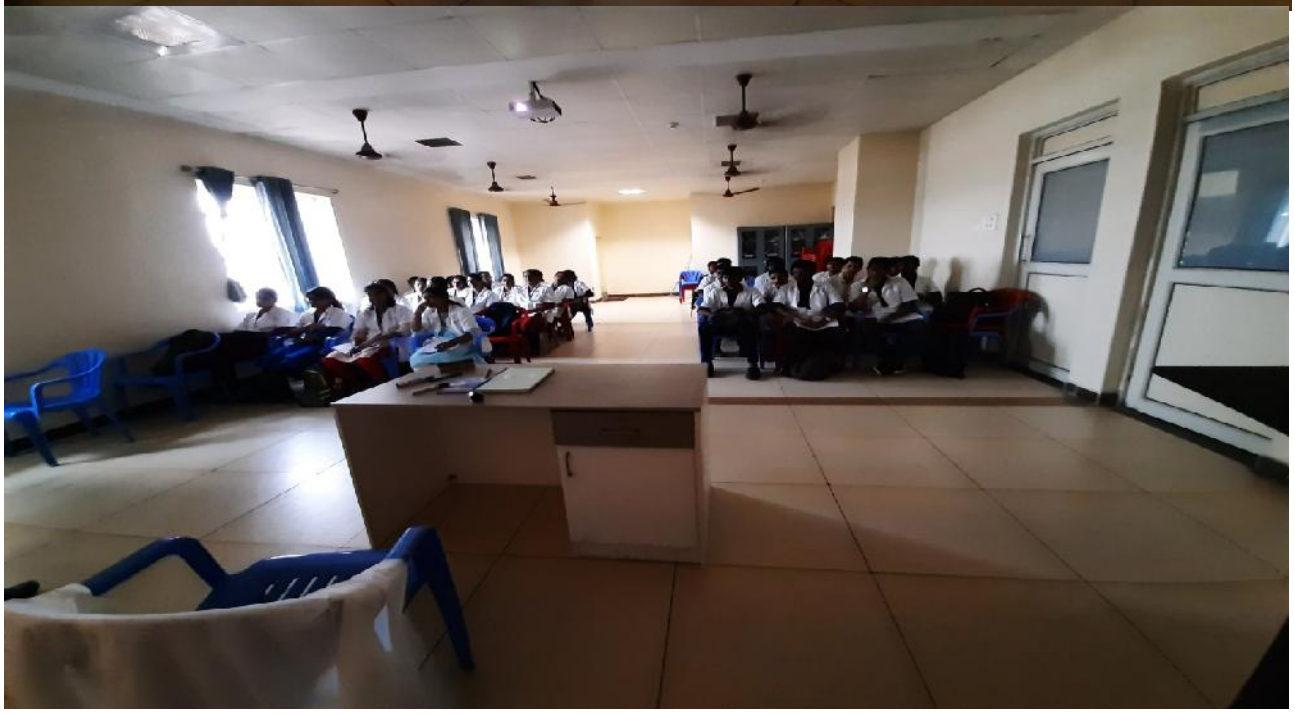

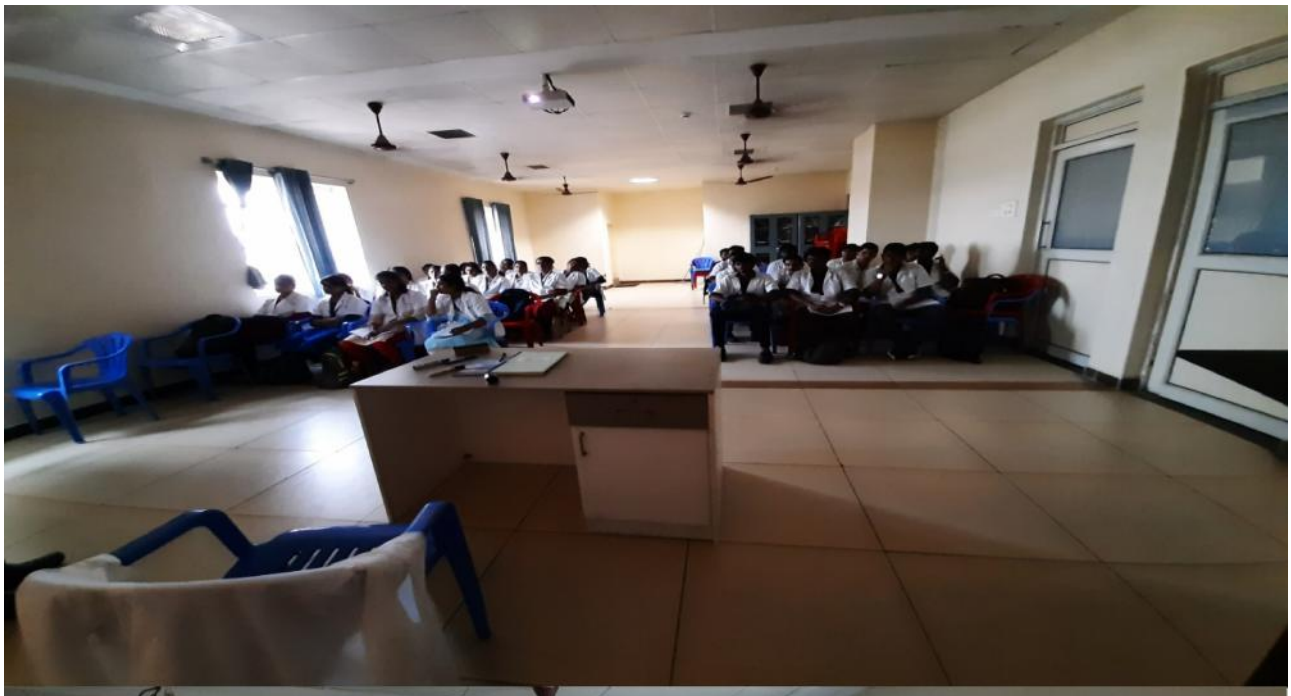


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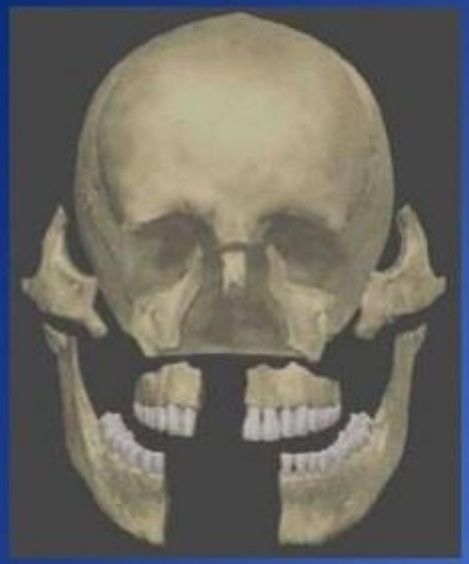
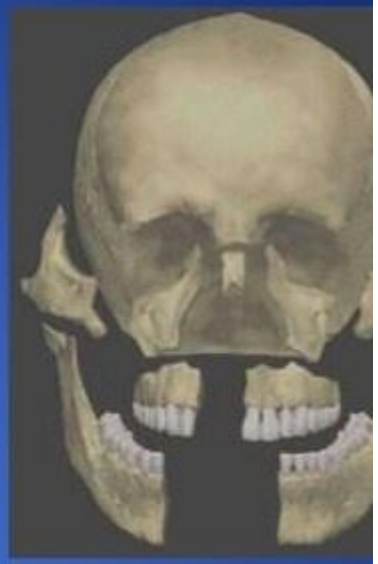
Department of Radiodiagnosis



Facial Trauma

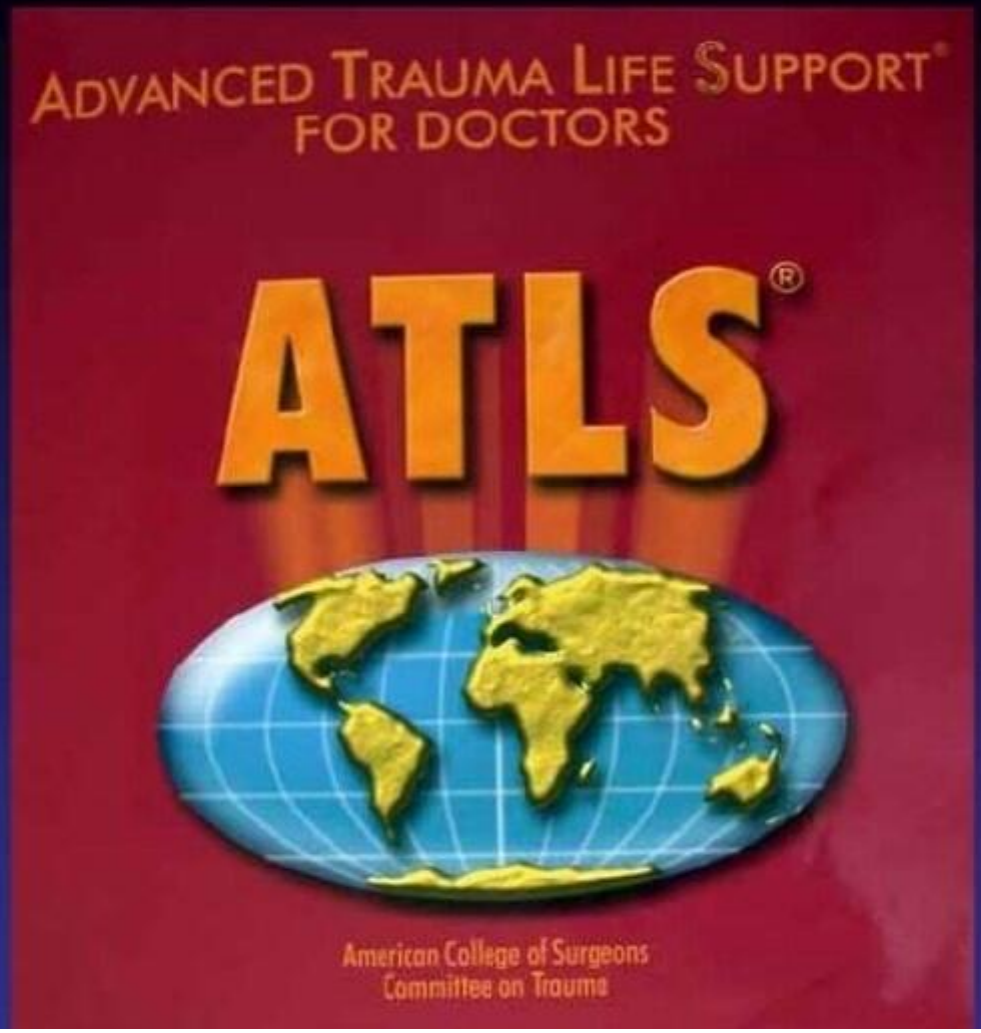


Plastic and Reconstructive Surgery Department
SCGH

Dr Sepehr Lajevardi

Initial Approach

- ABC's - Don't be distracted by obvious
- Stabilize patient & rule out life- or limb-threatening injuries
- Establish airway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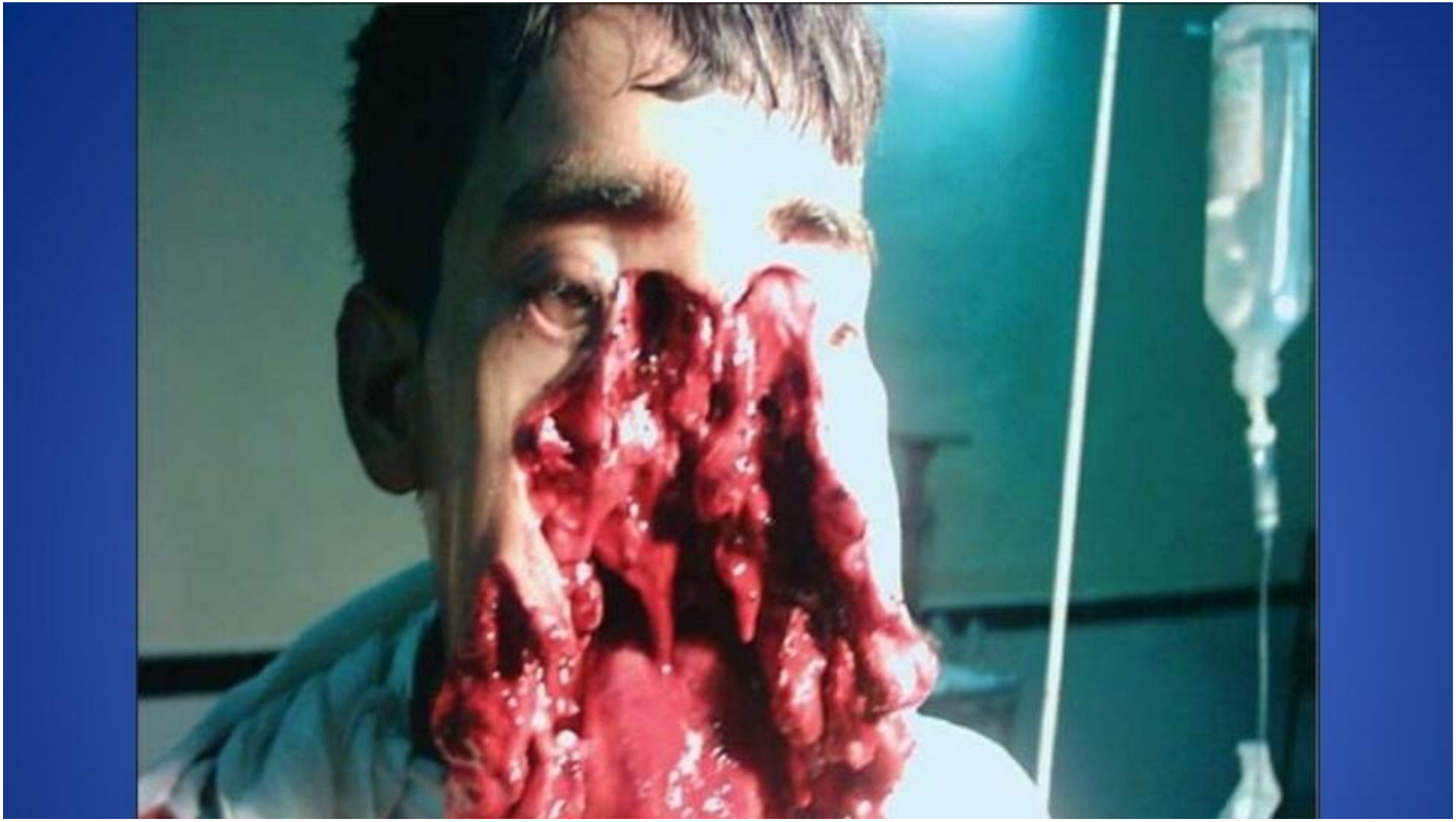
American College of Surgeons
Committee on Trauma

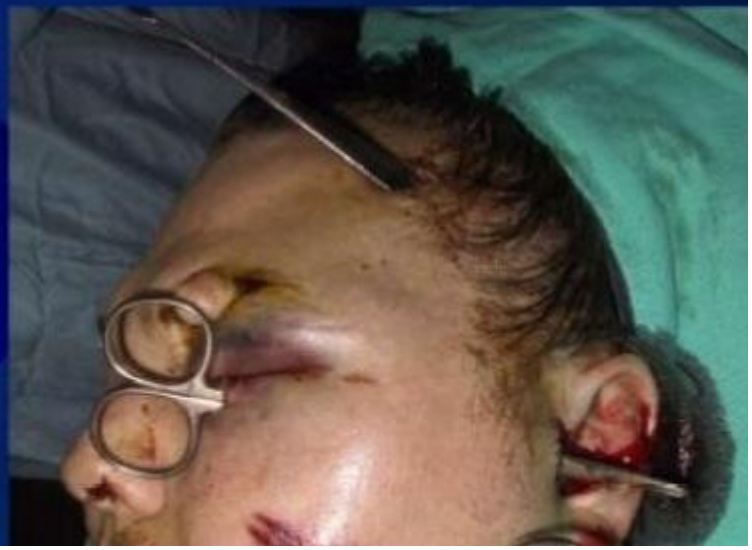


Facial Injuries

- Intracranial, Ocular and C-spine injuries must be ruled out / managed
  - ➡ these injuries are frequently assc. with facial trauma
- Patient rarely die from facial injuries.

Airway Management

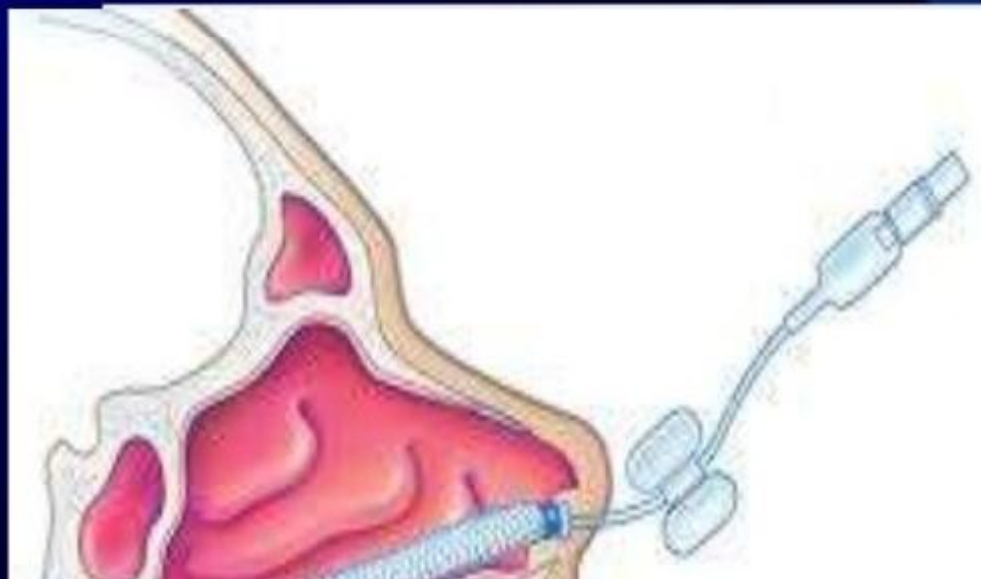
- Major facial trauma assc. with risk of upper airway obstruction
- Protect C-spine (5-10%)
- FB/blood, ↓ LOC, Loss

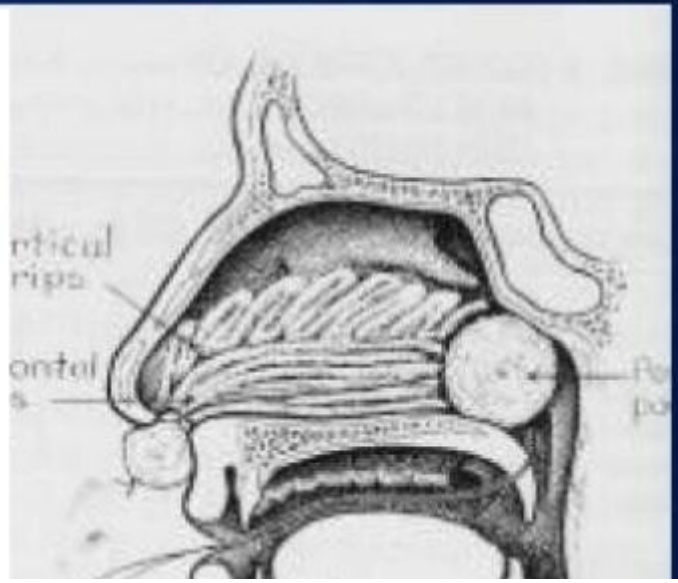


Massive Hemorrhage

- Life-threatening bleeding occurs in 1.2-5% of all LeFort fractures
- Hemorrhage is generally controlled by direct pressure
- Brisk epistaxis may respond to anterior

Massive Hemorrhage





Standard Clinical Assessment

History

Mode of injury, loss of consciousness, symptoms at examination, medications, alcohol or drugs and fasting status

General Examination

External Examination for Hard / Soft injury

Inspection looking for oedema, ecchymosis

Standard Clinical Assessment

Neurologic Examination

Cranial Nerves V and VII

Intra-oral Examination

Intra-oral examination in good lighting.

Inspect for ecchymosis in buccal or lingual sulci. Mucosal haematoma over underlying fractures

Standard Clinical Assessment

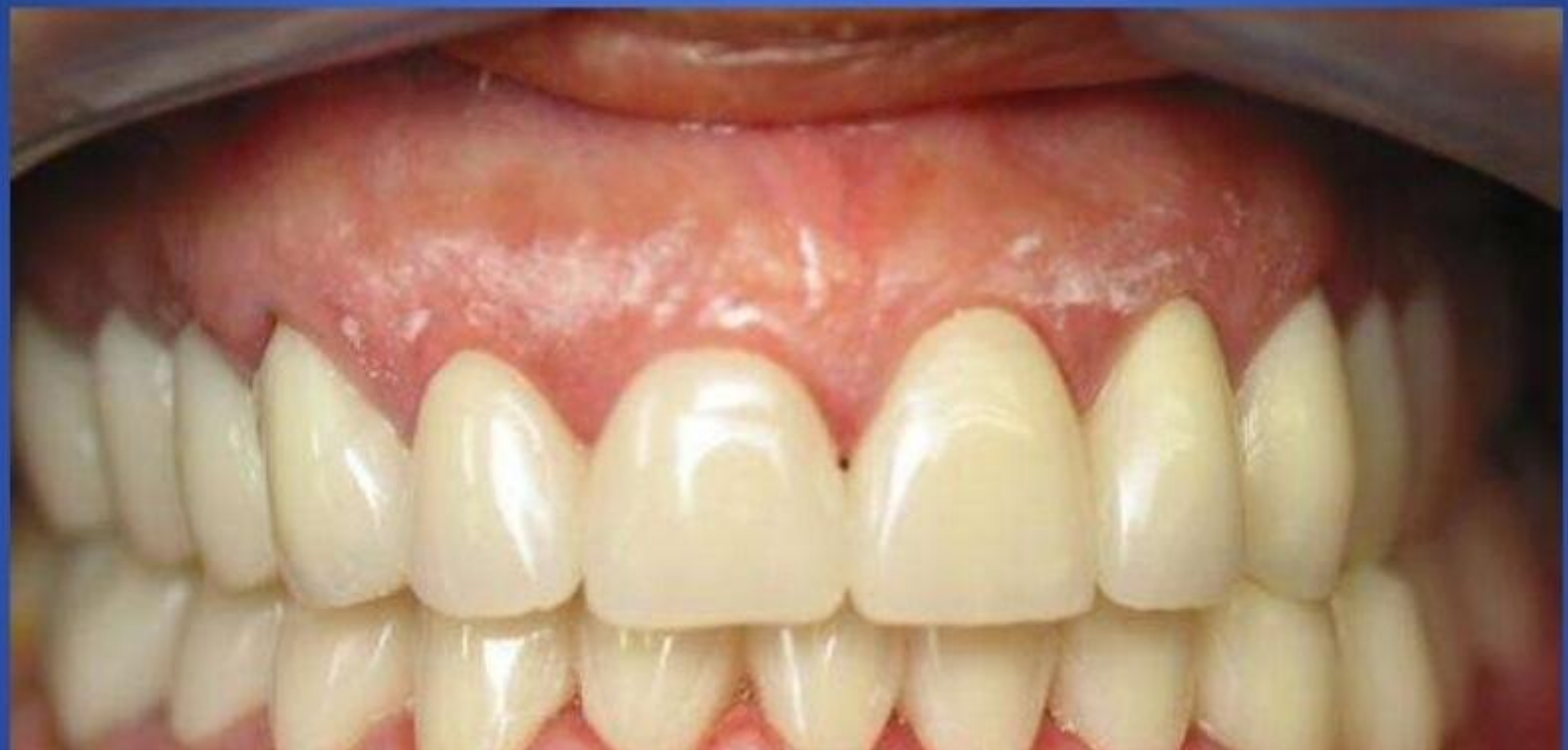
Orbital Examination

Ocular movements, ocular position. Intra-ocular pathology.

Close observation essential in zygoma fractures.

Nasal Examination

Occlusion



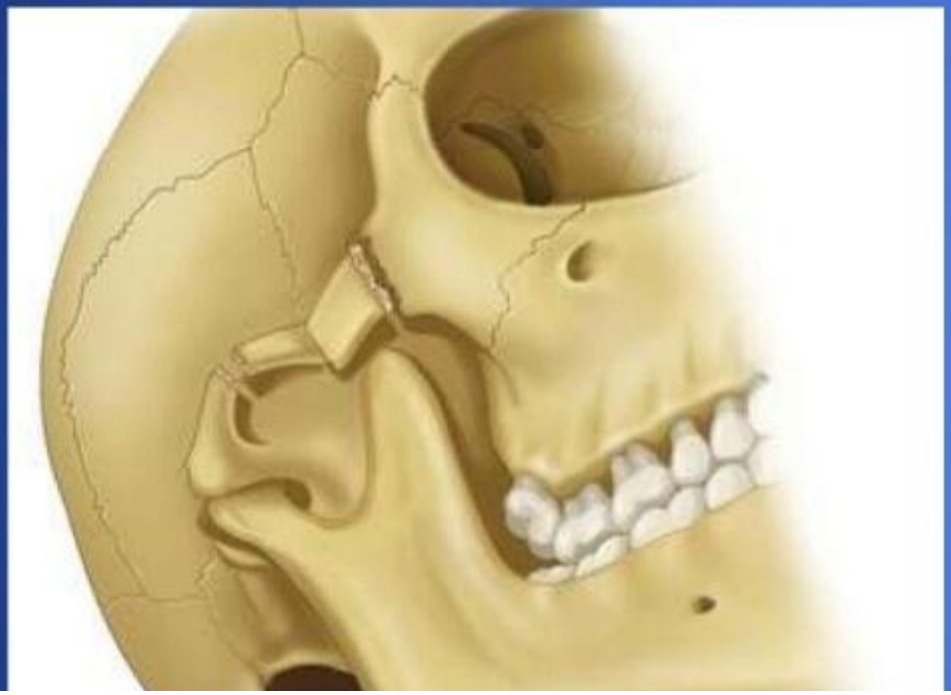
Bony Injuries

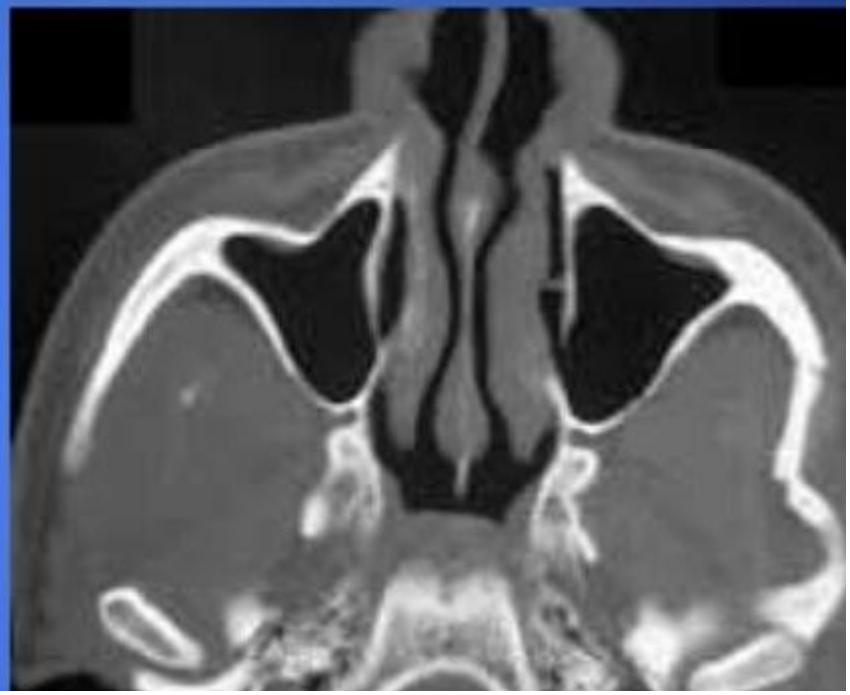
- Common Presentation
  - Nasal
  - Orbital Floor
  - Zygoma
    - Zygomatic Arch
  - Infra-orbital rim
  - Mandibular
- Less common

CLASSIFICATION OF FACIAL FRACTURES

1. **Upper ⅓**	2. **Middle ⅓**	3. **Lower ⅓**
Frontal Bone	*Maxilla*	*Mandible*
	Nasal	
	Naso-ethmoids	
	Orbitozygomatic	
	"Pure" blowout	

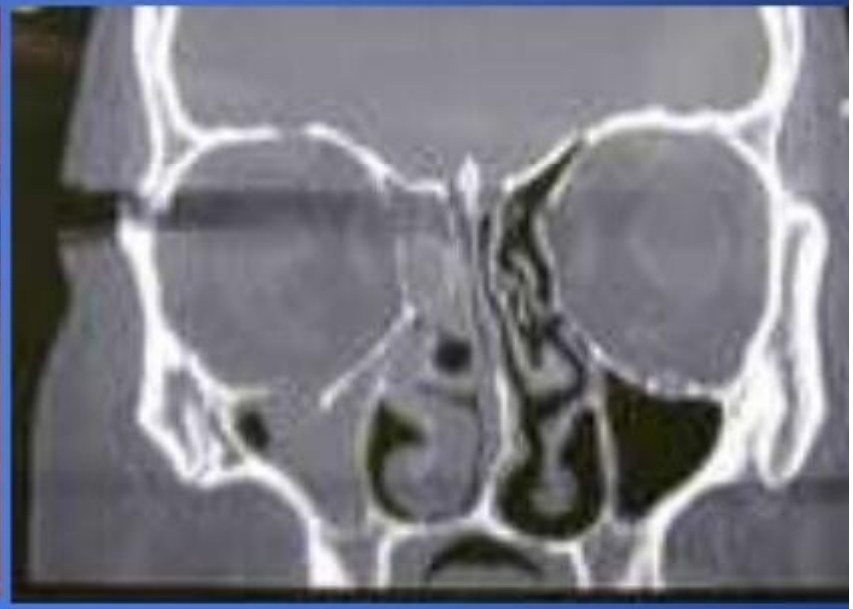
Zygomatic Arch



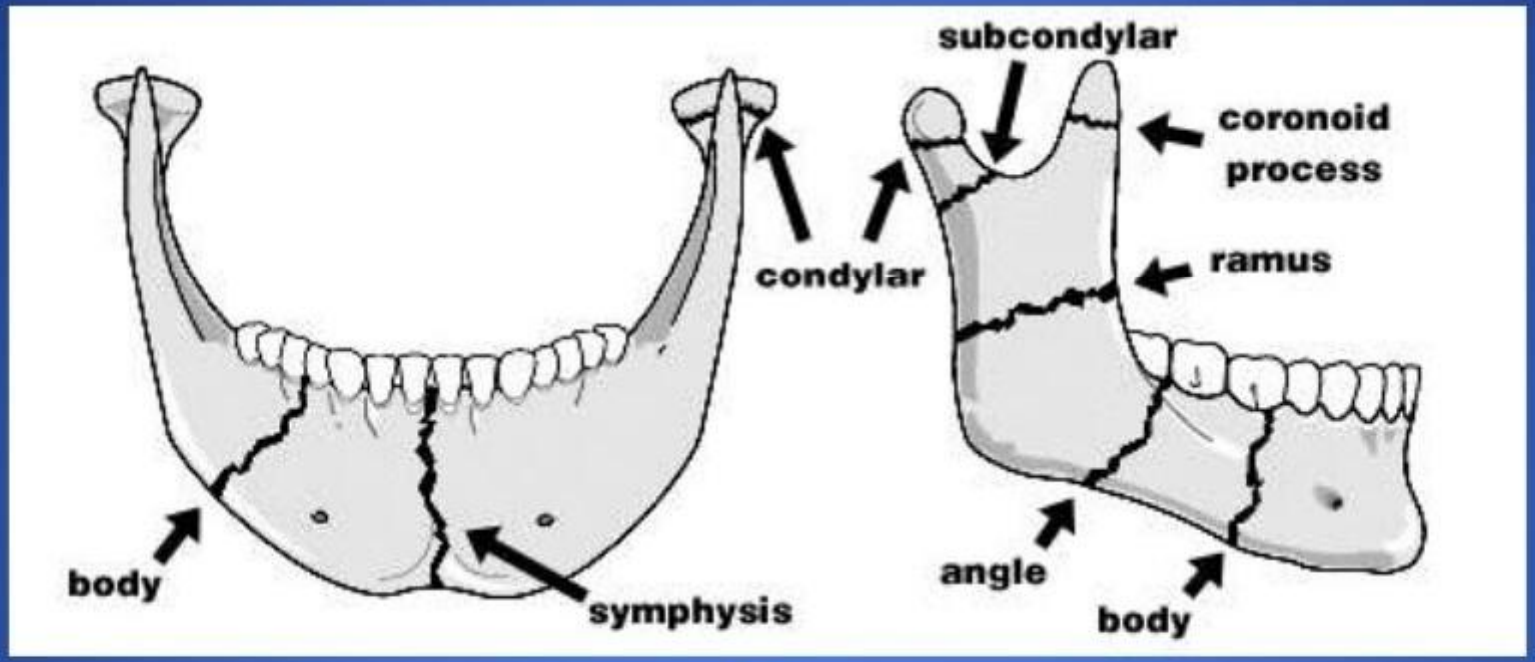


Zygoma Fracture

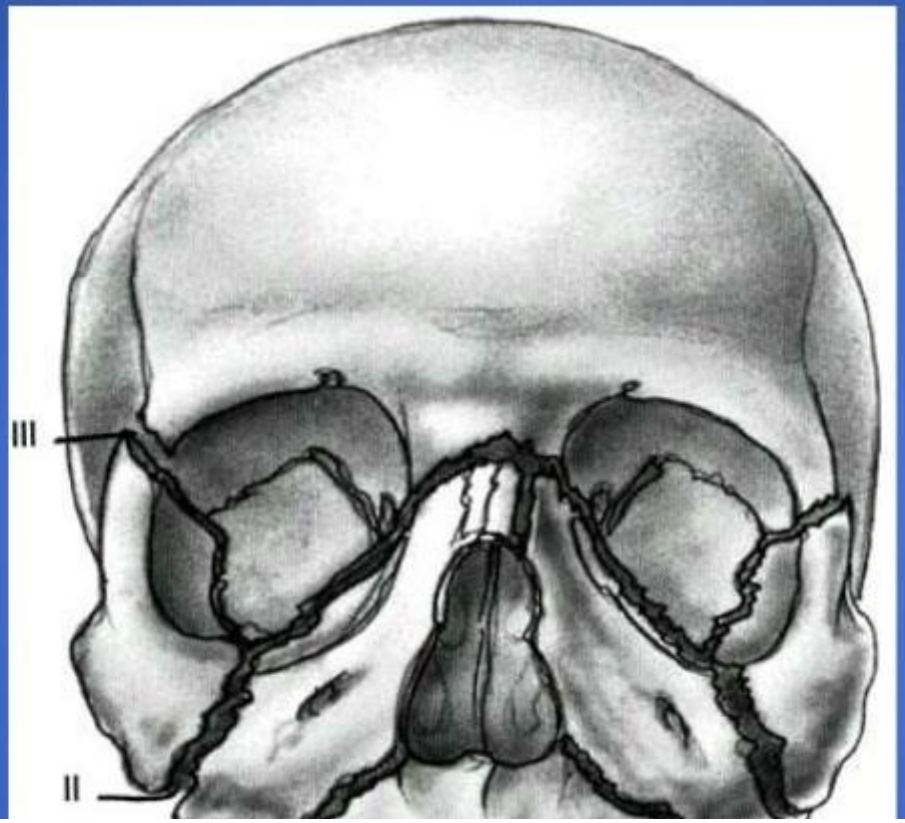
Orbital Floor



Mandible

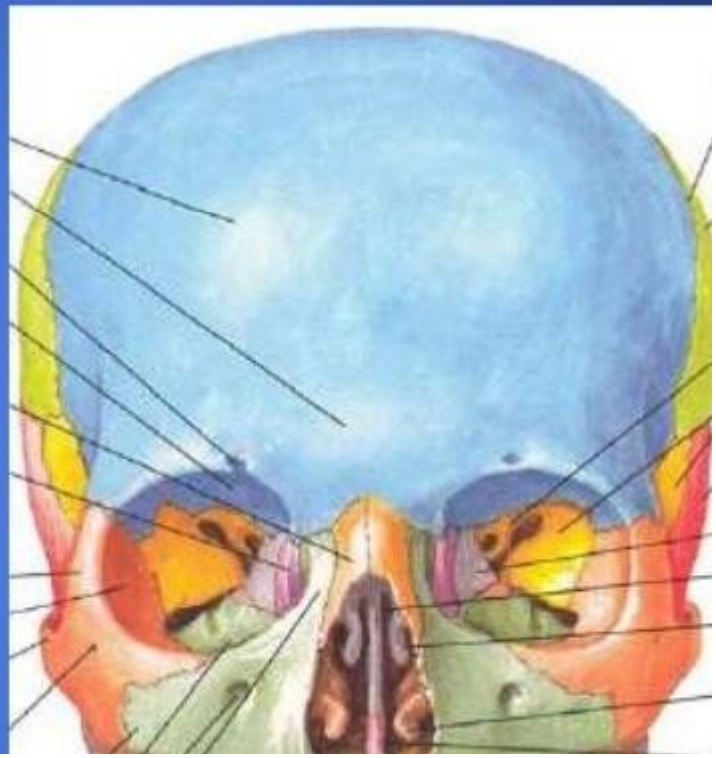

subcondylar
coronoid process
ramus
condylar
body
symphysis
angle
body

Le Forte Fractures



Local Anaesthetics in the Face

- Infra-orbital nerve
- Supra-orbital nerve
- Mental nerve
- Dorsal nasal nerves



ED referral for Facial #s

- On CT ensure facial bones and mandible included + 3D reconstruction views
- Call Plastics registrar post imaging
- Opthal review for orbital floor fractures
- Most patients can be reviewed as outpatient in PDC the next day (eReferral)
- Advise on no nose blowing

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DEPARTMENT OF RADIODIAGNOSIS

CHEST IMAGING

CERTIFICATE COURSE

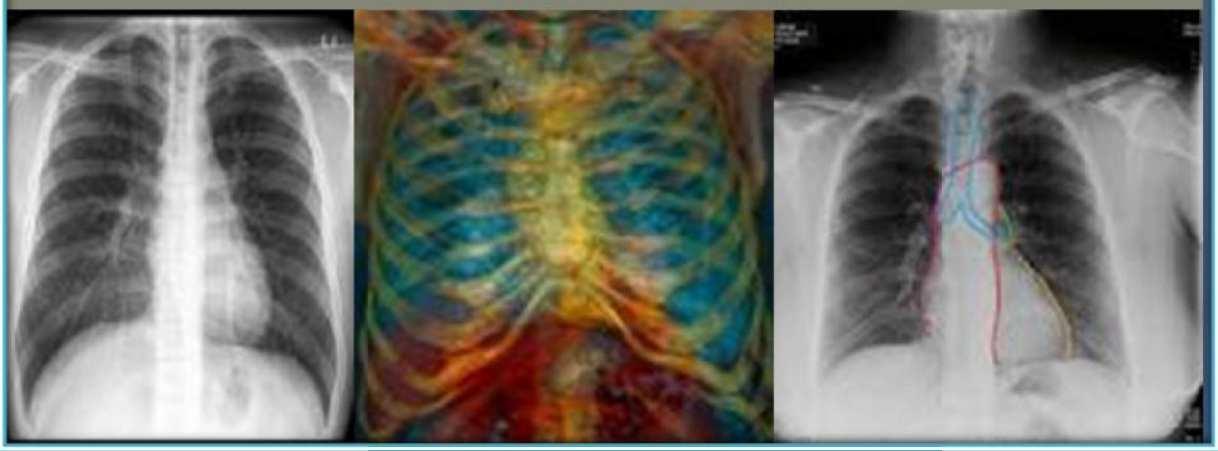
VELAMMAL MEDICAL COLLEGE HOSPITAL & RESEARCH INSTIUTE

CHEST IMAGING

CERTIFICATE COURSE

Department of Radiodiagnosis

Date: 14.05.2019
Time: 10 am – 12 pm
MBBS Students



DEPARTMENT OF RADIODIAGNOSIS
CERTIFICATE COURSE – CHEST IMAGING
14.05.2019

S.No	Name	Mobile Number	Signature
1.	Arsha. S.P	9442192277	
2.	Dhivya. R	8610454506	
3.	Bhargavi. G	9840399222	
4.	H. Gayathri	9994687897	
5.	V.S Aswathi	9445434474	
6.	Dharshini Priya. E	9677365774	
7.	K. Divya Bharathi	9489187202	
8.	Gayathri. S	9444020195	
9.	Gayathri P.J.	8300296126	
10	GAYASREE S	7373858088	
11	Akshita Singh	9488149441	
12	A. Anitta Xavier	95855 33371	
13	Amina Marwa Sabreen. A	9384736511	
14.	Ameya Venkatesh.	7010255369	
15	Akshitha Meenakshi	7094790909	
16.	Adlin Trinita	9786013799	
17	Aarina Krishnan	8848746609	
18	Dumpa Roshni	8939031174	
19.	Bailey Prakash	8610638498	

VELAMMAL MEDICAL COLLEGE
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MADURAI - 625009

Department of Anesthesiology

Invitation

CME And Workshop On Basic & Advanced Life Support

26th May 2019 & 2nd June

Organized by: Department of Anesthesiology.

Dear Colleagues ,

The Department of Anesthesia takes immense pleasure in inviting you for the Continuing Medical Education programme and workshop on **"Basic Life Support and Advanced Life Support"** for Internees and Practitioners.

We request all practitioners and CRRI'S to enroll and strengthen skills in managing cardiac arrest scenarios.

Dr. S.C. Ganesh prabhu,
Organizing chairman.

Dr.P.Ramadevi,
Organizing secretary.

Venue : Main Operation Theatre 2nd floor , VMCH &RI
Date : 26.05.2019

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

202

VELAMMAL MEDICAL COLLEGE
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CME and Workshop on Basic & Advanced Life Support

Format For Scientific Session

Date : 26.05.2019 & 02.06.2019

08.30 – 09.00am	**Introduction to CME**	**Dr. S.C.Ganesh Prabu**	
Time	**Topic**	**Chairpersons Or Other Resource Persons**	**Speaker**
09.00 - 09.45 am	Lecture on basic life support	Prof. Dr. T.Thirunavukkarasu	Dr. S.Sethumadhava Kumar
09.45 -10.30 am	Lecture on advanced life support	Prof. Dr. S.P.Meenakshi Sundaram	Dr. P.Ramadevi
10.30 -10.45 am	**Tea Break**		
10.45 – 11.30 am	Drugs used in life support	Prof. Dr. T.Nirmaladevi	Dr. K.Kala
11.30 – 12.15 pm	Equipment	Prof. Dr. P.Mageswari	Dr. R. Lavanya
12.15 – 1.00 pm	**Lunch Break**		
1.00- 2.00 pm	Workshop on basic life support	Prof. Dr. T.Thirunavukkarasu	Dr. S.Renganathan Dr. S.Sathia Narayanan
2.00- 3.00 pm	Workshop on advanced life support	Prof. Dr. S.P.Meenakshi Sundaram	Dr. S.Renganathan Dr. S.Balaji
3.00 - 3.15pm	**Tea Break**		
3.15 – 4.15 pm	Demonstration of equipment used in life support	Prof. Dr. P.Mageswari	Dr. R.Arun Shankar Dr. T.Malarvizhi
4.15 – 5.15 pm	Drugs used in life support	Prof. Dr. T.Nirmaladevi	Dr. J.N.C.Hamilton

Prof. T. THIRUNAVUKKARASU, M.D.,D.A.,
Dean
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Anuppanadi, Madurai, T.N.-625 009

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HOSPITAL AND RESEARCH INSTITUTE
MADURAI - 625009

From

Dr. S.C Ganeshprabhu
Professor & HOD
Department of Anaesthesiology
Velammal medical college & Research institute
Madurai -9

To

The Dean
Velammal medical college & Research institute
Madurai -9

Respected Sir:

Subject: Conducting BLS/ ACLS CME/ Workshop for CRRI on 26.05.2019 & 02.06.2019

As we are conducting CME on BLS & ACLS- Workshop on 26.05.2019 & 02.06.2019. I request you to kindly give permission to use the third floor auditorium on may 26th and june 2nd from 8.30 am -11.30am and to use our department halls for workshop 11.30am -2.00pm on the same days.

21.05.2019
Madurai - 9

Thanking you

Yours faithfully

21/5/19

(Dr. S.C Ganeshprabhu)

PROFESSOR & HEAD
Dept. of Anaesthesiology

21/5/19

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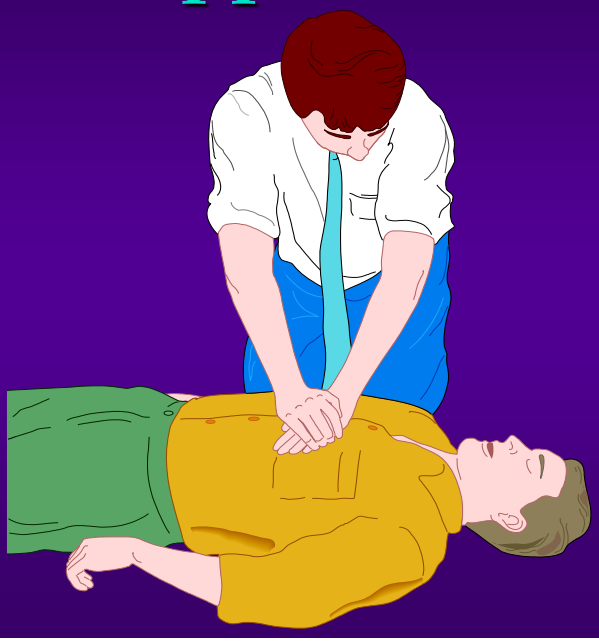
Basic Life Support
CPR



Introduction

- ASU basketball game
- Student: You saved my child's life
- Introduce Annie

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BLS & ACLS FOR RESCUERS

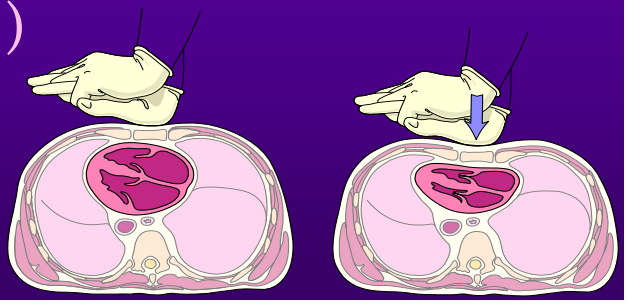
PROFESSOR OF
ANAESTHSIOLOGY
VELAMMAL MEDICAL
COLLEGE

CPR

- Combines rescue breathing and chest compressions
- Revives heart (cardio) and lung (pulmonary) functioning
  - Use when there is no breathing and no pulse
- Provides O2 to the brain until ACLS arrives

How CPR Works

- Effective CPR provides 1/4 to 1/3 normal blood flow
- Rescue breaths contain 16% oxygen (21%)

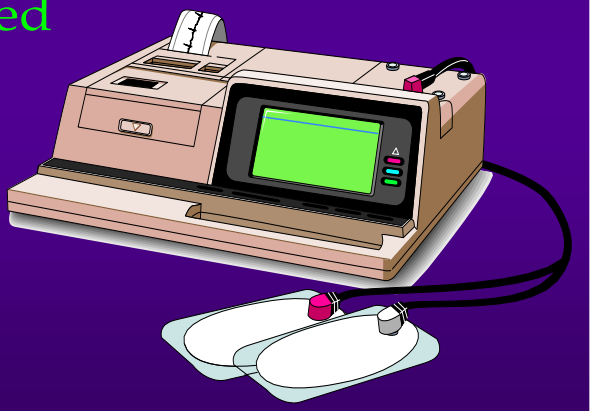


Start CPR Immediately

- Better chance of survival
- Brain damage starts in 4-6 minutes
- Brain damage is certain after 10 minutes without CPR

Even With Successful CPR, Most Won't Survive Without ACLS

- ACLS (Advanced Cardiac Life Support)
- ACLS includes defibrillation, oxygen, drug therapy



Survey The Scene, then: RAP

- R - Responsiveness
  - Tap shoulder and shout "Are you ok?"

RAP

- A - Activate EMS (if unresponsive)
  - YOU - call 911 – come back and let me know what they said (another can stay by the phone)
  - You may have to make the call

ABCD

- **Airway**
- **Breathing**
- **Circulation - Bleeding**
- **Disability** (keep this in mind from the beginning)
  - If victim is unconscious but does display vital signs, place on left side

Checking Vital Signs

- A – Airway
  - Open the airway
  - Head tilt chin lift

B – Check For Breathing

- Look, listen and feel for breathing
  - No longer than 10 seconds seconds

Breathing

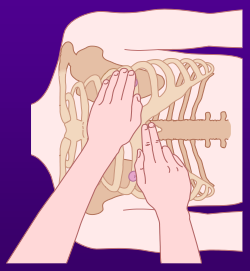
- If the victim is not breathing, give two breaths (1 second or longer)
  - Pinch the nose
  - Seal the mouth with yours
- If the first two don't go in, re-tilt and give two more breaths (if breaths still do not go in, suspect choking)

Breathing: Mouth To Nose (when to use)

- Can't open mouth
- Can't make a good seal
- Severely injured mouth
- Stomach distension
- Mouth to stoma (tracheotomy)

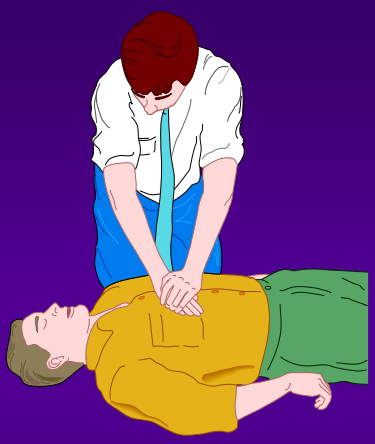
Compressions

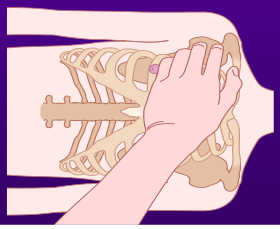
- After giving breaths…
- Locate proper hand position for chest compressions
  - Place heel of one hand on center of chest between the nipples **OR**



Compressions

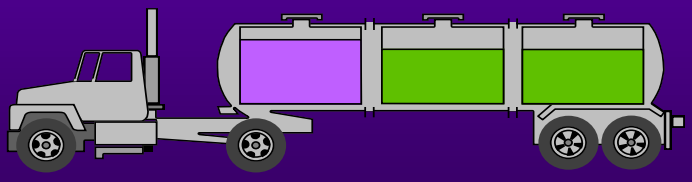
- Using both hands, give 30 chest compressions
  - Count 1, 2, 3 …
- Depth of compressions: 1 .5 to 2 inches
- For children: ½ to 1/3 of chest depth and use 1 or 2 hands (keep one hand on forehead if possible)





When to Stop CPR

- Victim revives
- Replaced by another rescuer
- Too exhausted
  - Trucker



Two Partner CPR

- Rescuer 1:
  - RAPAB
- Rescuer 2:
  - place hands for compressions
- Compression rate: 30:2
- Switch off when tired
- 1 and 2…..4 and change

Checking for CPR Effectiveness

- Does chest rise and fall with rescue breaths?
- Have a second rescuer check pulse while you give compressions

Mouth to Mouth Barrier Devices

- Masks
- Shields

If You Are Afraid to Perform CPR

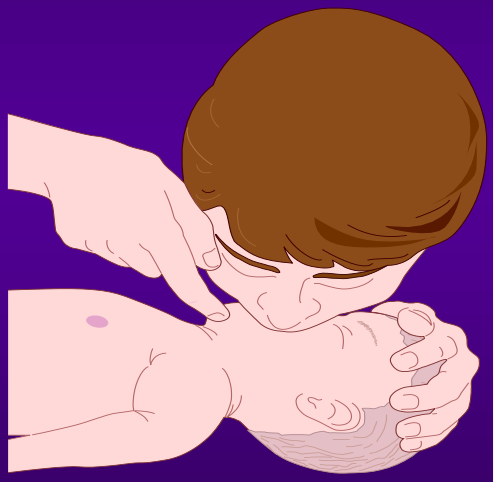
- Call EMS
- Open the airway
- Give chest compressions

If Victim Becomes Unconscious After Giving Thrusts

- Call 911
- Try to support victim with your knees while lowering victim to the floor
- Assess
- Begin CPR
- After chest compressions, check for object before giving breaths breaths

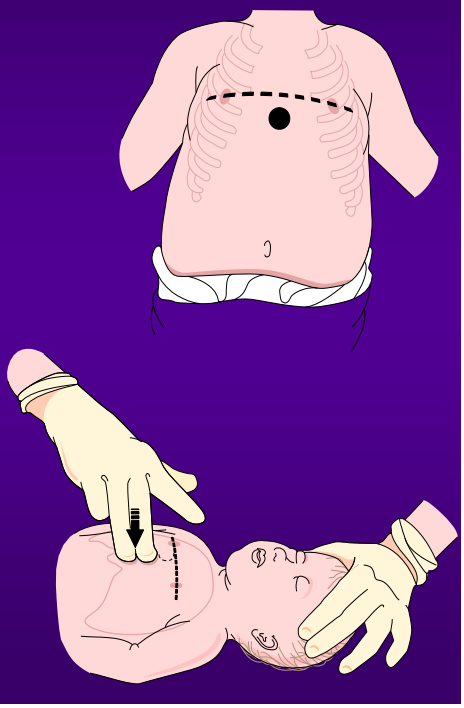
CPR for Infants (Under 1 Year of Age)

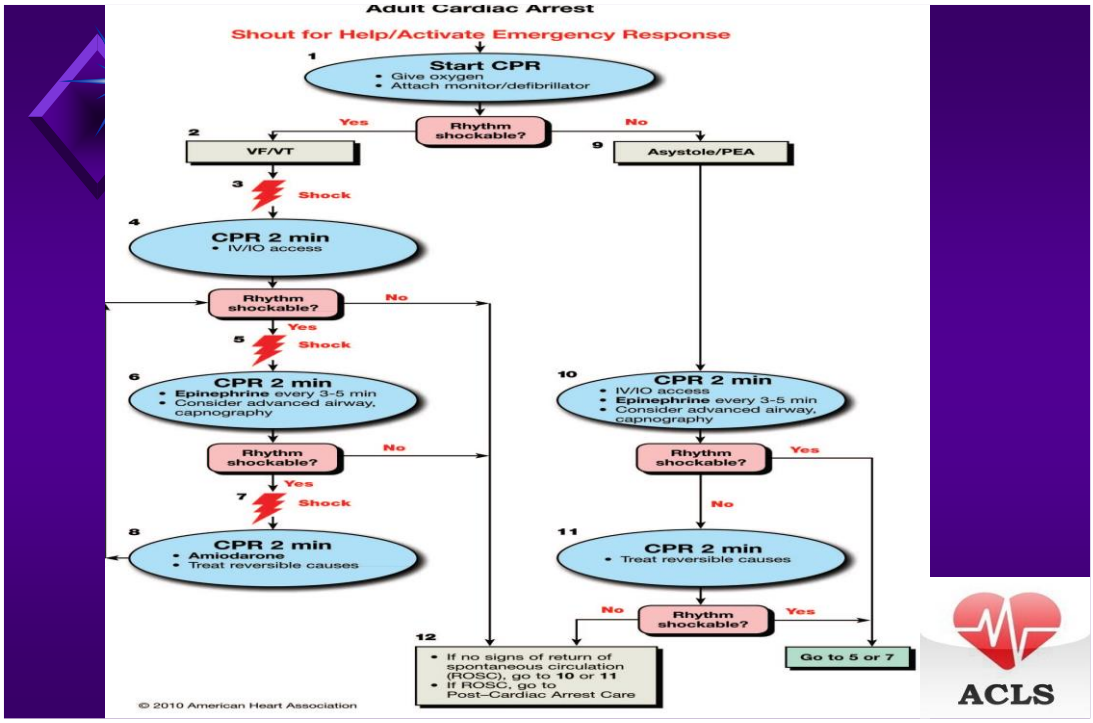
- Same procedures (RAPAB) except:
- Seal nose and mouth or nose only
- Give shallow "puffs"
-



CPR: Infants

- RAPAB
- Give CPR
  - Press sternum 1/2 to 1/3 depth of the chest
  - Use middle and ring finger
- 30 compressions to 2
- If alone, resuscitate for 2 minutes then call 911





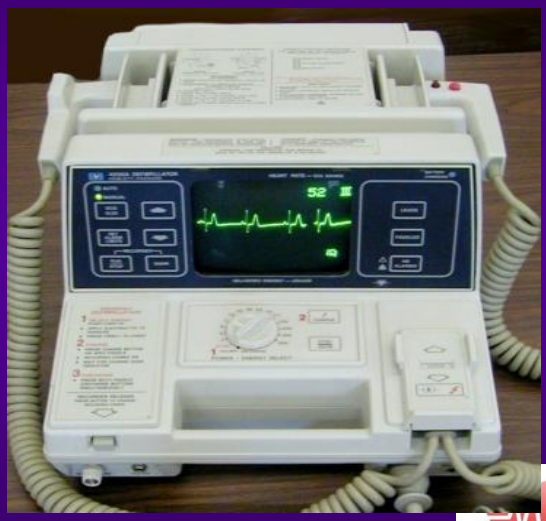
ADJUVANT DEVICES

- Interposed abdominal compression (IAC)
- Active compression-decompression CPR (ACD-CPR) by a device containing a suction header, bellows, and a compression area within the bellows
- Impedance threshold device (ITD)
- Circumferential compression of the chest with a load-distributing band (LDB)

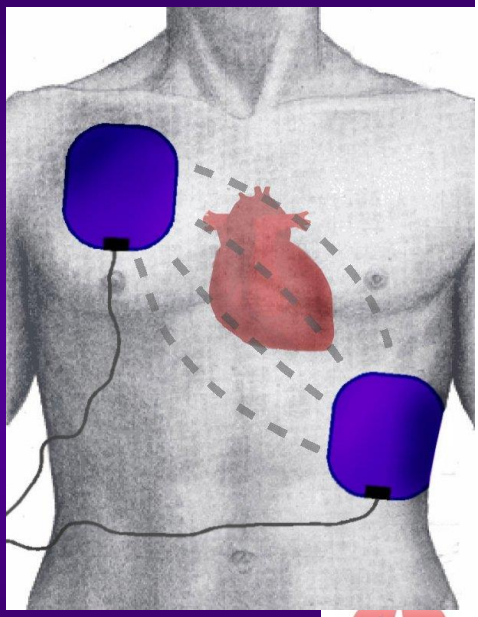
ACLS

DEFIBRILLATION

- Defibrillation consists of delivering a therapeutic dose of electrical energy to the affected heart with a device called a **defibrillator**.



ACLS



ACLS

TYPES

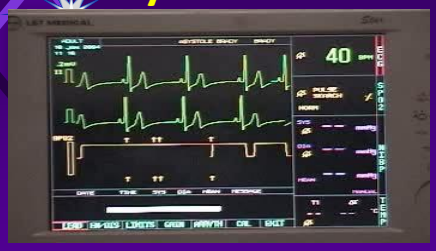
- Automated external defibrillators (AEDs)
- Semi-automated AED
- Standard defibrillators with monitors

ACLS

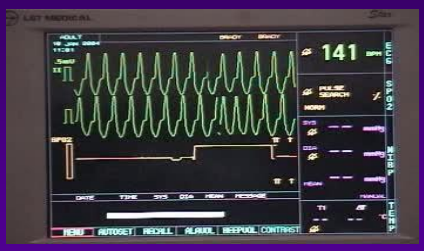
- Defibrillation is a nonsynchronized delivery of energy during any phase of the cardiac cycle
- Cardioversion is the delivery of energy that is synchronized to the large R waves or QRS complex.

ACLS

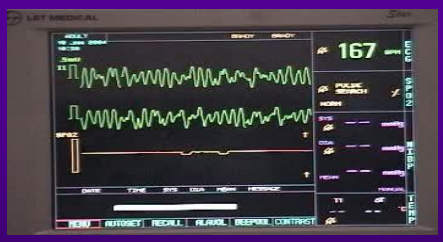
Bradycardia



PulselessVT



VF



Asystole

ACLS

DEFIBRILLATION

VF/VT

1shock
200j

CPR
30:2
For 2 min

Do not reassess the rhythm or feel pulse

CPR for 2min

Check rhythm and give shock

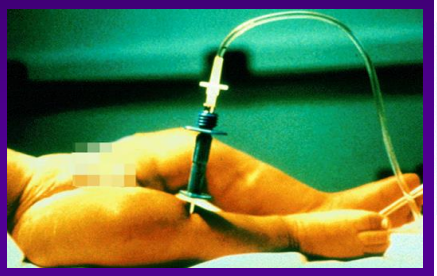
ACLS

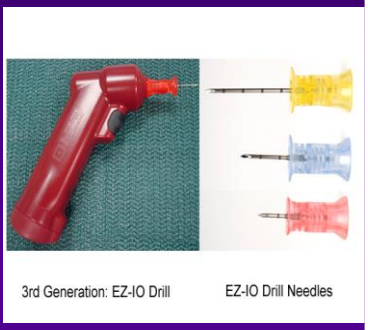
CARDIOVERSION

ARRHYTHMIAS	BIPHASIC ENERGY DOSE
SVT/A.Flutter	50 J – 100J
AF / VT	120 J- 200J

DRUGS ROUTE

- IO





- IV

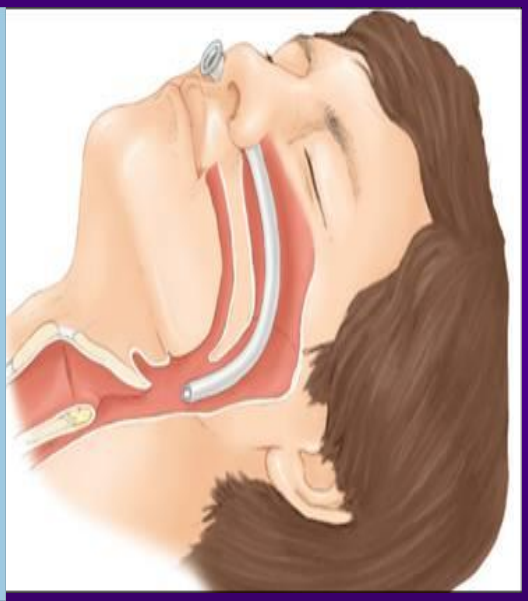
ACLS

~~Endotracheal~~

~~Intra cardiac~~

ACLS

NASOPHARYNGEAL



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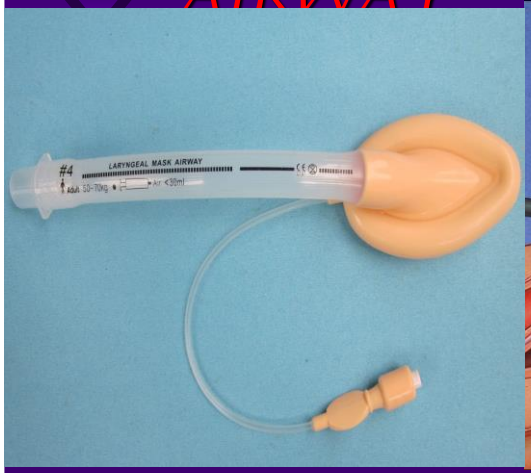
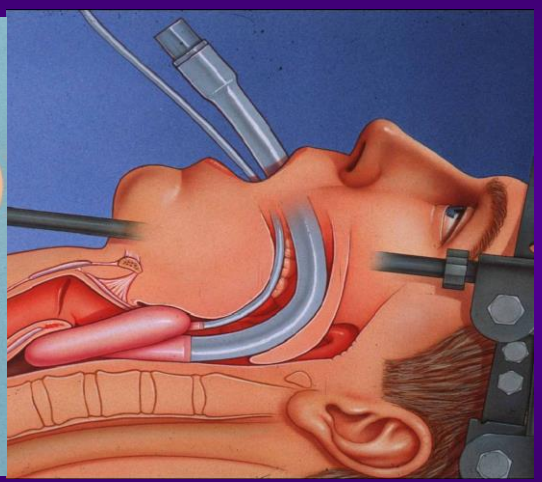
OROPHARYNGEAL AIRWAYS

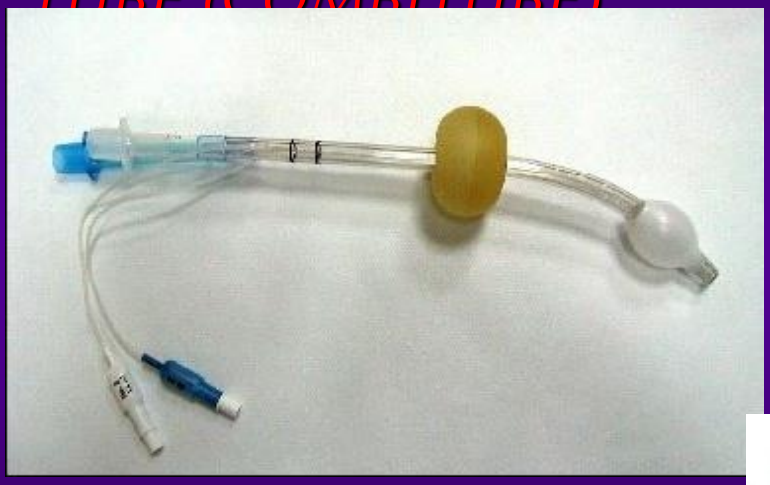
ACLS

LARYNGEAL MASK AIRWAY





ESOPHAGEAL-TRACHEAL TUBE (COMBITUBE)

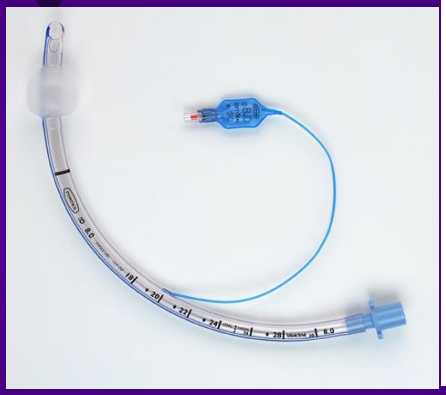


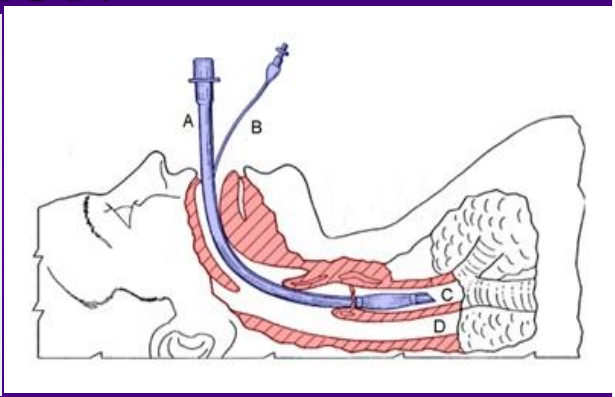
ACLS

LARYNGEAL TUBE OR KING LT

ACLS

ENDOTRACHEAL INTUBATION





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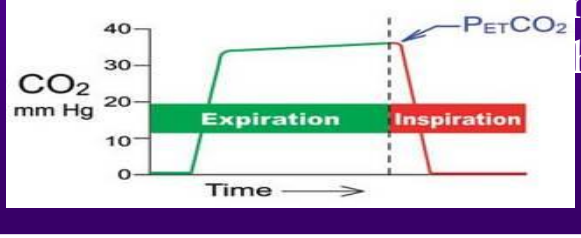
CAPNOGRAPHY

- PETCO2 is 10 mm Hg, it is reasonable to consider trying to improve CPR quality by optimizing chest compression parameters
- If PETCO2 abruptly increases to a normal value (35 to 40 mm Hg), it is [illegible]hat this is a[illegible]



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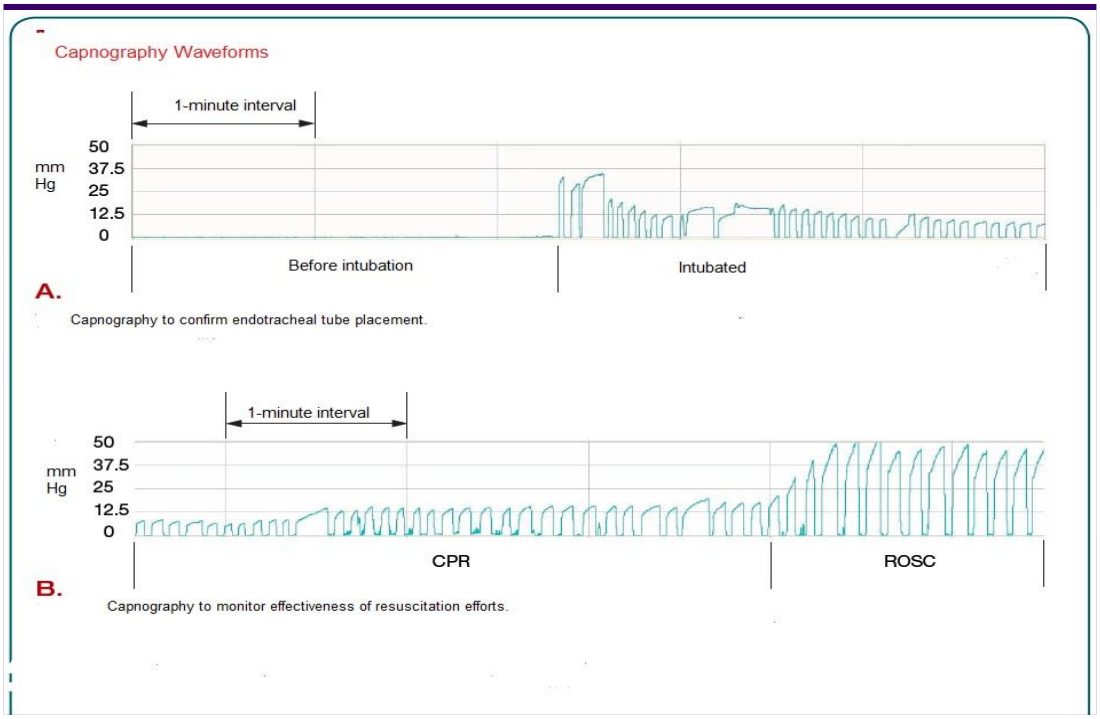
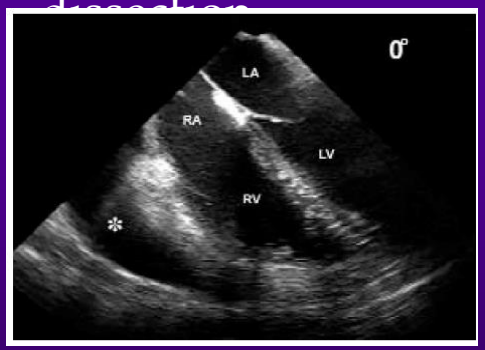
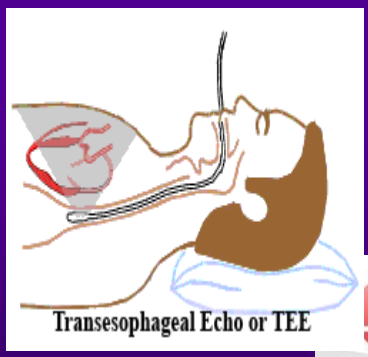


- Pulse Oximetry & ABG not reliable
- **Echocardiography:**

To diagnose treatable causes of cardiac arrest such as cardiac tamponade, pulmonary embolism, ischemia, and aortic dissection





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PEDIATRIC ADVANCED LIFE SUPPORT

- The initial defibrillation energy dose - 2 to 4 J/kg
- For second and subsequent doses, give at least 4 J/kg.

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- Adenosine :
  - 0.1 mg/kg (maximum: 6 mg),Repeat 0.2 mg/kg (maximum: 12 mg)
  - Contraindicated in Bronchial asthma, congestive cardiac failure ,Severe hypotension
- Amiodarone:
  5 mg/kg IV/IO; repeated up to 15 mg/kg
  Maximum: 300 mg

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- Atropine:
  - 0.02 mg/kg IV/IO
  - Maximum single dose: Child: 0.5 mg
    Adolescent: 1 mg
- Epinephrine:
  - 0.01 mg/kg (0.1 mL/kg 1 : 10,000) IV/IO
- Procainamide:
  - 15 mg/kg IV/IO over 30- to 60-min period

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SUMMARY

- Defibrillation - single shock is best.
- CPR is continued.
- Oxygenation is must.
- IV/IO
- Drugs -Epinephrine, amiodarone.
- To protect the brain.
- Algorithm is scientifically and evidence based.