Government Medical College ,Surat

Group:-3

Batch No:- 95

Guidance by:- Dr.S.M.Patel

Group Members :

	Roll Num.
1) Chhatrala Janki	21
2) Chauhan Bhargav	22
3) Dalal Shivani	23
4) Darji Deepak	24

Case:- Snake Bite

History

➡ We visited patient at H-3 ward of the new civil hospital.

➡

The patient is male of 25 years old bitten by snake late evening
At 26 September 2016.
Admitted in the EMERGENCY WARD.

nake Specification :- Snake was 30 cm long with spot

➡ Snake bite was on the medial side of left ankle

And this area had edema

➡ The patient is currently on following medication

(1) Anti Snake Venom

(2) Neostigmine

➡ REPORT at night of 26th September 2016

Prothrombin Time – 28s

WBCs count – 15800

iochemistry REPORT at 27/9/2016 2:45 A.M.

TEST	RESULT	REFERENCE RANGE	ALERT
Albumin	3.7	3.5-5.2g/dl	
Alk.Phosphatase	117	42-128U/L	
ALT	37	<45U/L	
Direct Bilirubin	0.3	<0.4mg/dl	
Total Bilirubin	**2.5**	<1.3mg/dl	High Abnormal
Indirect Bilirubin	**2.2**	<1.3mg/dl	High Abnormal
Creatinine	0.8	0.8-1.3	
k+	**3.39**	3.5-5.1mmol/L	Low Abnormal
Na+	**135.47**	136-145mmol/L	Low Abnormal
Total Protein	**6.1**	6.4-8.3g/dl	Low Abnormal

27/9/2016 Pathology REPORT at 5 P.M.

Prothrombin Time – 13s (Normal)

Biochemistry REPORT at 28/9/2016 2:40 A.M

TEST	RESULT	REFERENCE RANGE	ALERT
Albumin	**3**	3.5-5.2g/dl	Low Abnormal
Alk.Phosphatase	81	42-128U/L	
ALT	**194**	<45U/L	High Abnormal
Direct Bilirubin	**2.1**	<0.4mg/dl	High Abnormal
Total Bilirubin	**3.7**	<1.3mg/dl	High Abnormal
Indirect Bilirubin	**1.6**	<1.3mg/dl	High Abnormal
Creatinine	1.2	0.8-1.3	
k+	**3.27**	3.5-5.1mmol/L	Low Abnormal
Na+	137.09	136-145mmol/L	
Total Protein	5.3	6.4-8.3g/dl	

athology REPORT at 28/9/2016 3.00 A.M

- Prothrombin Time – 25s (Abnormal)
- WBCs count – 16900

- Previous history of surgery and hospitalizatio
- Occasional drinker of alcohol.

LT (Alanine Transferase enzyme

- T is an Alanine transferase enzyme. It is a mark
f hepatocellular damage which means
amage to liver which happens in hepatitis.

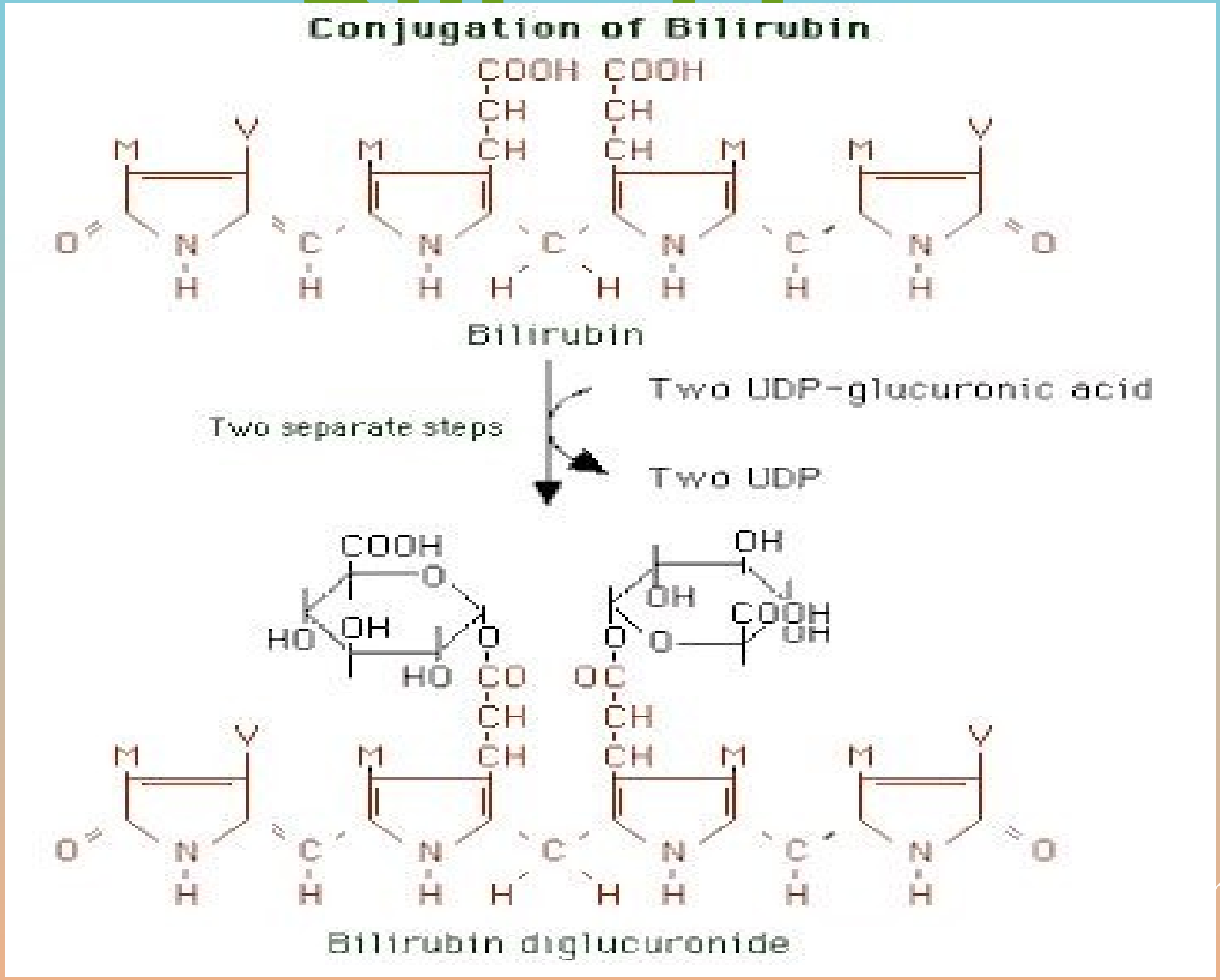
- may be of viral origin, congestive heart failure,
abetes, bile duct problem etc.
- increase in ALT suggests disease is in active o
ogressive phase.

Bilirubin

- **Conjugated bilirubin:** **Bilirubin bound to glucuronic acid is called conjugated bilirubin which has 6 hydroxyl group.**

This Conjugation occurs in liver and excreted in bi

Conjugated Bilirubin



his figure shows conjugated bilirubi

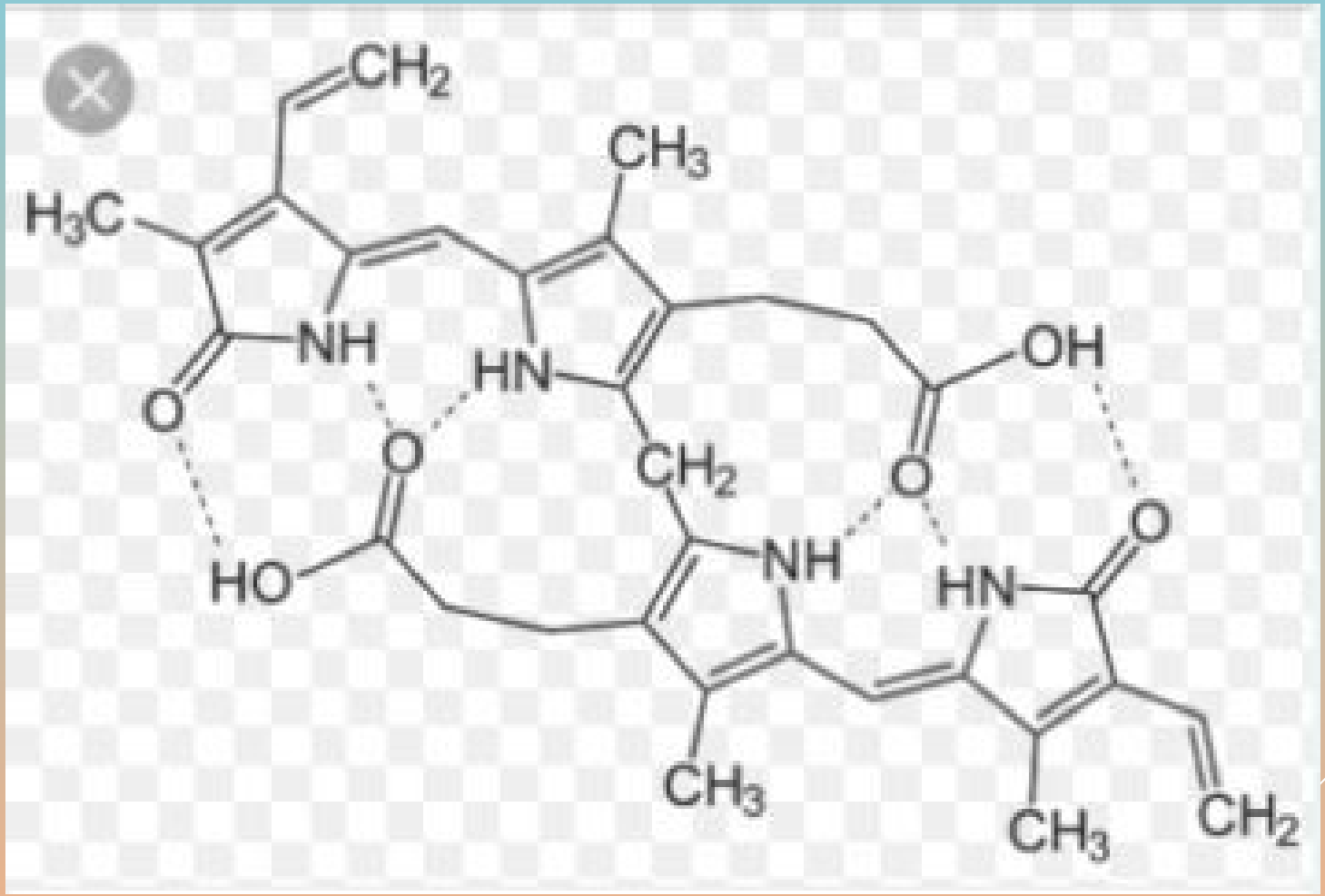
- ower part of figure shows two rings with many COOH group attached to main bilirubin molecule.
- ey are glucuronic acid. They are water soluble, making entir conjugated bilirubin water soluble

- **Unconjugated bilirubin :** Unconjugated bilirubin is not bound to glucuronic acid since it has only 2 hydroxyl groups it is not water soluble

- So it binds with plasma protein albumin

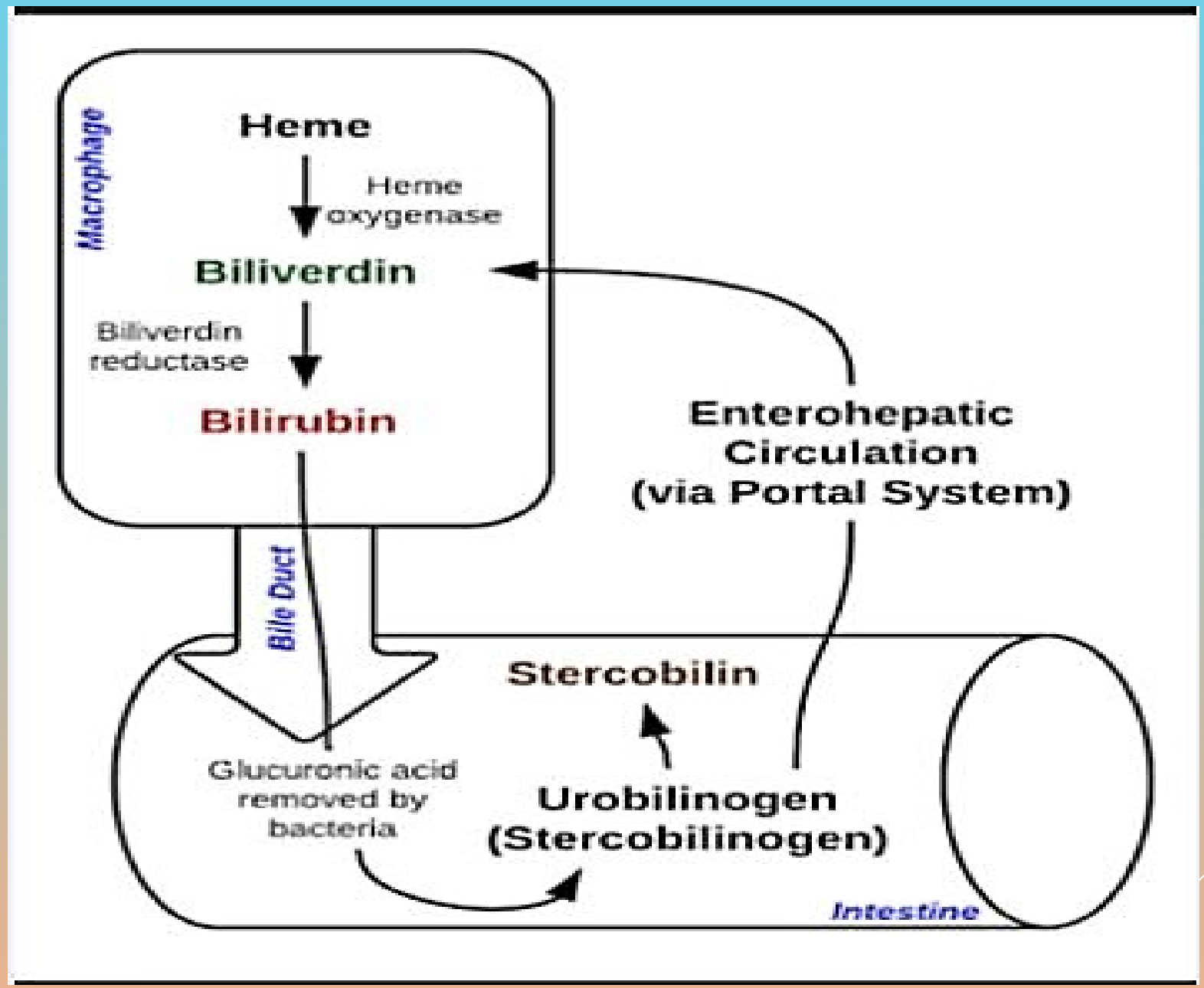
→ This way it circulates in blood with the support in albumin

Unconjugated Bilirubin



- Unconjugated bilirubin can form six hydrogen bonds, they are intramolecular.
- So it can not form H bond with water. So it is very less soluble in water.
- Once it is conjugated, there are many H bonds possible with bilirubin and glucuronide attached, making conjugated bilirubin very water soluble.

- In Lab, bilirubin is measured by it’s ability to form red colored compound with certain chemical.
- The compound which produces red color with bilirubin is called diazotised sulphanilic acid .
- onjugated bilirubin directly reacts water so it is called direct bilirubin .
- nconjugated bilirubin does not reacts with water. Hence this is called Indirect bilirubin .
- We can seprat it from albumin by adding methanol.


Macrophage
Heme
Heme oxygenase
Biliverdin
Biliverdin reductase
Bilirubin
Enterohepatic Circulation (via Portal System)
Bile Duct
Stercobilin
Glucuronic acid removed by bacteria
Urobilinogen (Stercobilinogen)
Intestine

Differential Diagnosis

Condition 1:- Destruction of large number of RBC, as in malaria, haemotoxic snake venom, sickle cell anaemia, extravascular haemolysis etc.

Observation:- Increased level of indirect bilirubin

Reason:- When lot of bilirubin is formed by macrophages, liver capacity for conguation is full. So unconjugated bilirubin accumulate.
There is good capacity of liver to throw conjugated bilirubin in bile.
So conjugated bilirubin does not increase when RBC killed.

Condition 2:- Destruction of hepatocytes as in viral hepatitis, alcoholism etc

Observation:- Increased level of indirect (unconjugated) and direct (conjugated) bilirubin.

Reason:- Hepatocytes convert unconjugated bilirubin into conjugated but due to their destruction unconjugated bilirubin level increase.
Also due to inflammation of liver, on destruction of hepatocytes, bile canals are blocked hence conjugated bilirubin level also rises

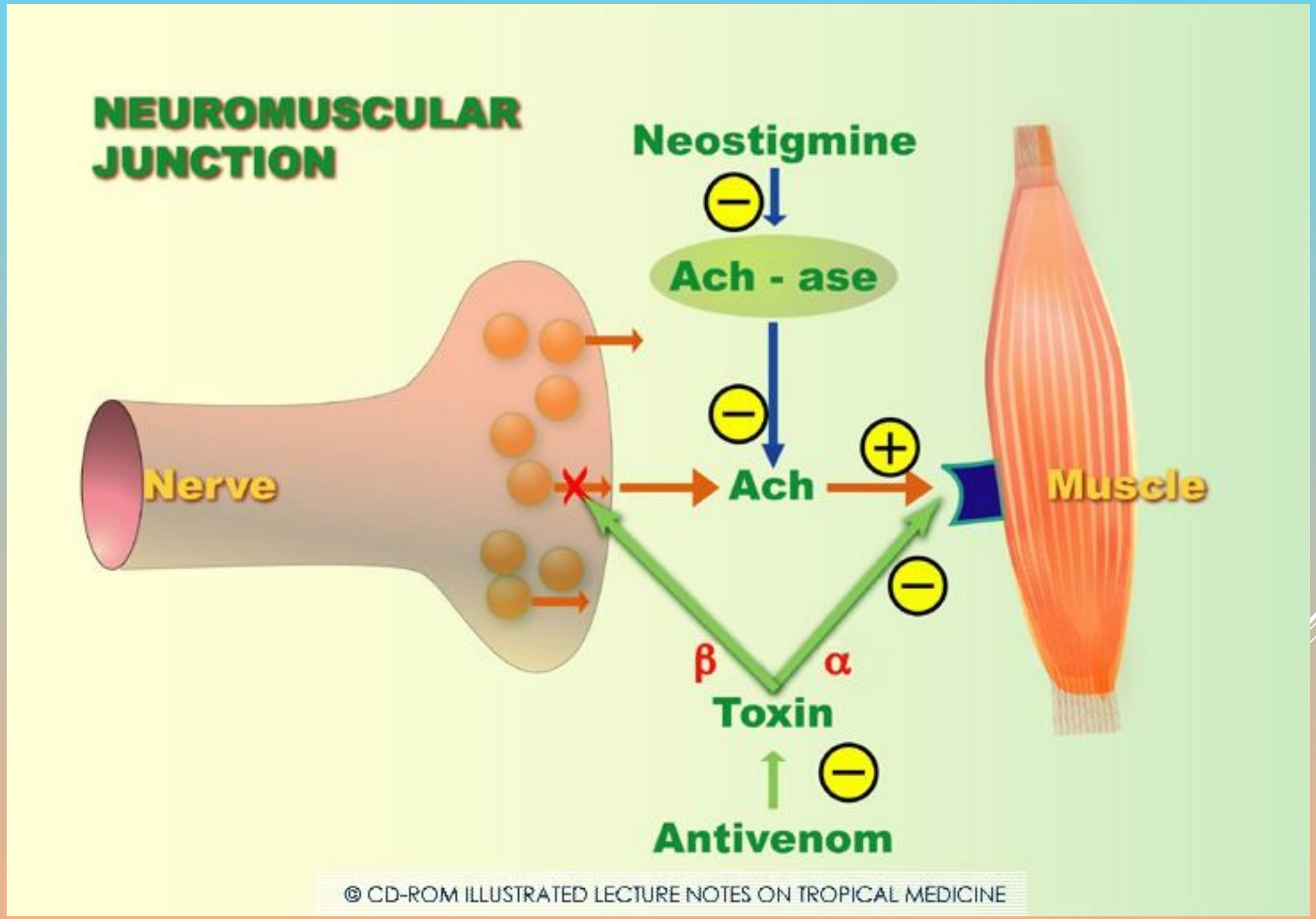
Condition 3:-Bile canaliculi is blocked as in obstructive jaundice.
Bile canaliculi are small canal which get pressed and blocked by liver inflammtion

Observation:- Increased level of direct bilirubin in liver

Reason:- Due to accumulation of bile in liver along with it the conjugated bilirubin formed inside the liver is also accumulated.

- Prothrombin time is about a blood report that measures the time required to

clot blood. Normal value is about 12- 14 second.


NEUROMUSCULAR JUNCTION
Neostigmine
Ach - ase
Nerve
Ach
Muscle
β
α
Toxin
Antivenom
© CD-ROM ILLUSTRATED LECTURE NOTES ON TROPICAL MEDICINE

1. Why some snake bite cause muscle paralysis ?

- Answer: Refer to figure above to understand following
  - alpha refers to alpha-toxin (alpha bungarotoxin), which is a protein. Beta refers to beta-toxin (beta bungarotoxin). It is also a protein.
  - Ach(Acetylcholine) is released from Nerve and Bind receptor protein on Muscle -> Causing muscle contraction
    alpha bind receptor protein on Muscle and prevent Ach from binding muscle -> Muscle do not contract -> Paralysis.
  - Beta-toxin is a phospholipase A2, probaby damaging nerve cell membrane. This results in decreased release of ACh from Nerve. -> Muscles do not contract -> Paralysis

- Acetylcholinesterase is the enzyme which catalyses ACh to acetic acid n choline molecule. It is found in neuromuscular junction and cholinergic synapse.

- Inhibition of AChE leads to accumulation of ACh in the synaptic cleft and causes transmission of nerve signal which causes increased muscle contraction

Snake Venom

- Snake venoms are used in the production of snake antivenom as hyperimmunizing antigens. Snake venoms are complex substances that, depending on the species, can contain a variety of toxins.

- Toxin components can include proteases, nucleases, phosphodiesterases, and Other enzymes which disrupt physiological processes and cellular integrity.

- The venom toxins are largely classified as neurotoxins, cytotoxins, myotoxins, and cardiotoxins.

- Venomous snake bites may cause a variety of symptoms, including pain, swelling, tissue necrosis, hypotension, neuromuscular collapse, blood clotting dysfunction,
respiratory paralysis, kidney failure, coma and death.

Manufacturing and Production of Antivenom

- Antivenoms are typically produced using a donor animal, such as a horse or sheep.
- The donor animal is hyperimmunized with non- lethal doses Of one or more venoms to produce a neutralizing antibody response.
- Then, at certain intervals, the blood from the donor animal is collected and neutralizing antibodies are purified from the blood to produce an antivenom.

- PLA2 is enzyme naturally occuring in our body cells. But when it is present in excess in ECF due to snake bite , it destroy cell membrane containing phospholipid.

- ✓ The result is: Cell death —> Inflammation (high WBC) —-> Oedema
  RBC lysis —> Hemolysis —> increased indirect bilirubin
  Hepatocyte lysis —> hepatitis —> increased indirect + direct bilirubin

- Pro coagulant- serine proteinase or metalloproteinase and Anti coagulant type - PLA2
  They cause variation in prothrombin time

What Is Alkaline Phosphatase?

- Alkaline phosphatase is an enzyme found in your bloodstream.
  ALP helps break down proteins in the body and exists in different forms, depending on where it originates.
- It is mostly produced in your liver, but some is also made in your bones, intestines, and kidneys. In pregnant women, ALP is made in the placenta

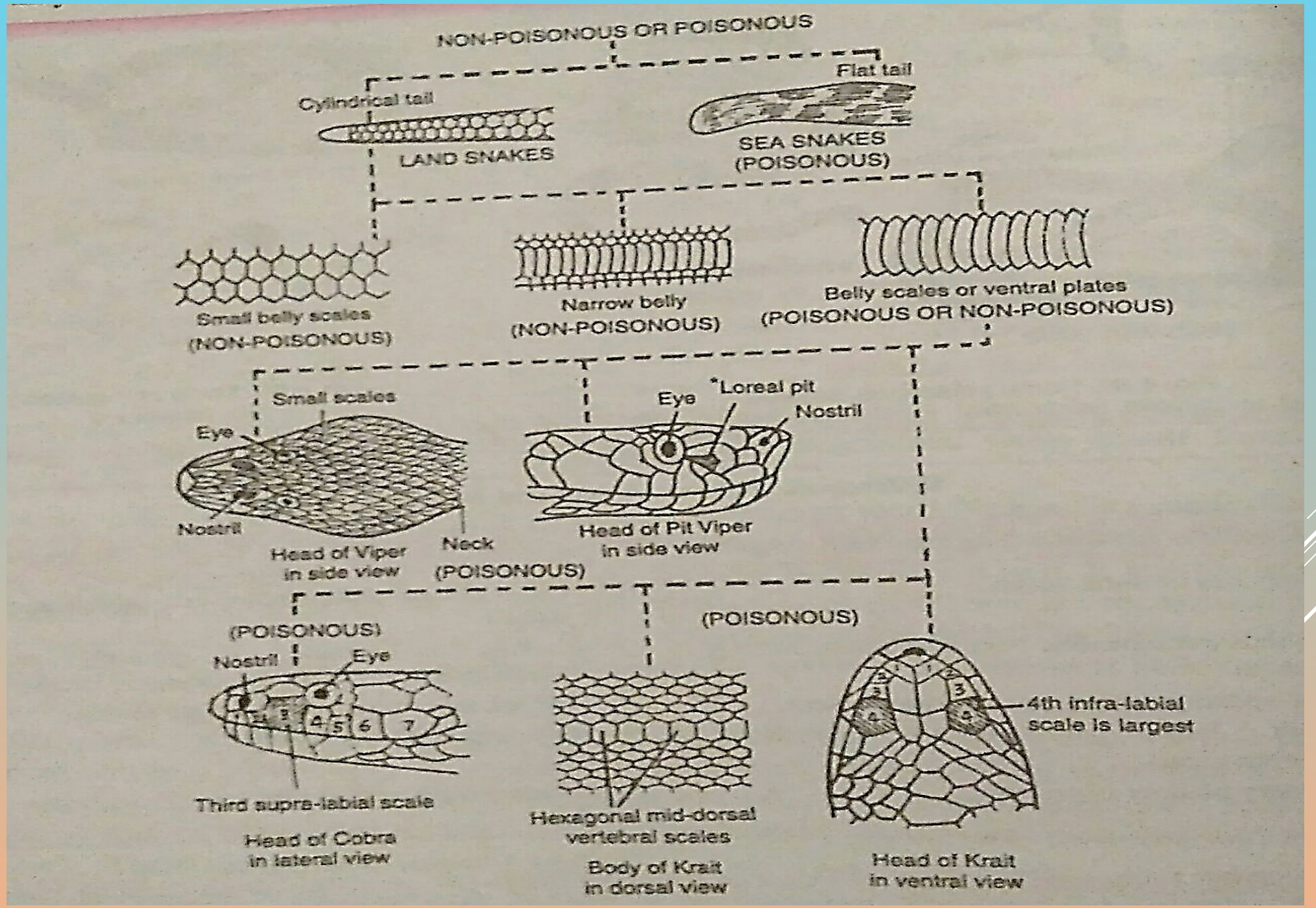
❖ Liver and Gall Bladder

- Checking ALP levels in the blood is a routine part of a liver function and gall bladder tests. Symptoms such as jaundice (yellowing of the skin and eyes), abdominal pain, vomiting, and nausea may lead your doctor to suspect there is something wrong with your liver or gallbladder

- The ALP test can be helpful in identifying conditions such as:
  - hepatitis (inflammation or infection of the liver)
  - cirrhosis (scarring Of the liver)
  - cholescystitis (inflammation of the gallbladder)
  - blockage of bile ducts (from gallstone, inflammation, or cancer)

- You may also need an ALP test if you are taking a medication that has the potential to damage your liver, such as acetaminophen (Tylenol). Measuring ALP is one way to check for that damage.

Identification of snakes


NON-POISONOUS OR POISONOUS
Cylindrical tail
LAND SNAKES
Flat tail
SEA SNAKES
(POISONOUS)
Small belly scales
(NON-POISONOUS)
Narrow belly
(NON-POISONOUS)
Belly scales or ventral plates
(POISONOUS OR NON-POISONOUS)
Small scales
Eye
Nostril
Head of Viper
in side view
Neck
(POISONOUS)
Loreal pit
Eye
Nostril
Head of Pit Viper
in side view
(POISONOUS)
Nostril
Eye
Third supra-labial scale
Head of Cobra
in lateral view
(POISONOUS)
Hexagonal mid-dorsal
vertebral scales
Body of Krait
in dorsal view
(POISONOUS)
4th infra-labial
scale is largest
Head of Krait
in ventral view

Structures	Characters	Nature	Snakes
1.Tail	A)Tail laterally compressed,oar-like	Poisonous, Hydrophis, Enhydrina	Sea snakes
	(b) Tail cylindrical, tapering	Poisonous or non-poisonous, Examine further	Land snakes
2. Belly scales or ventrals	(a) Belly scales small, continuous with dorsals	Non-poisonous	
	(b) Ventrals not fully broad to cover belly	Non-poisonous	Pythons
	(c) Ventrals broad. filly covering belly	Examine further	

Structures	Characters	Nature	Snakes
3. Head scales sub-caudals	(a) Head scales small, head triangular. No loreal pit	Poisonous	Pitless vipers Vipera russelli
	(i) Loreal pit, (ii)Subcaudals single	Poisonous Poisonous	Ancistrodon Echis carinata
	(b) Head scales shield like. A loreal pit present between nostril and eye	Poisonous	Pit vipers
	(c) Head with large shields. No loreal pit	Examine further	

Structures	Characters	Nature	Snakes
4. Vertebra's, 4th infralabial 3rd supralabial	(a) Vertebrals enlarged, hexagonal 4th infra-labial largest	Poisonous	Krait, Bungarus
	(bVertebrals not enlarged, 3rd supra-labial touches eye and nostril	Poisonous	
	(i) Neck with a hood and spectacle mark	Poisonous	Cobra, Naja
	(ii) Hood absent. Coral spots on	Poisonous	Coral snakes
	(c) No such characters	Non-poisonous	

Thank You