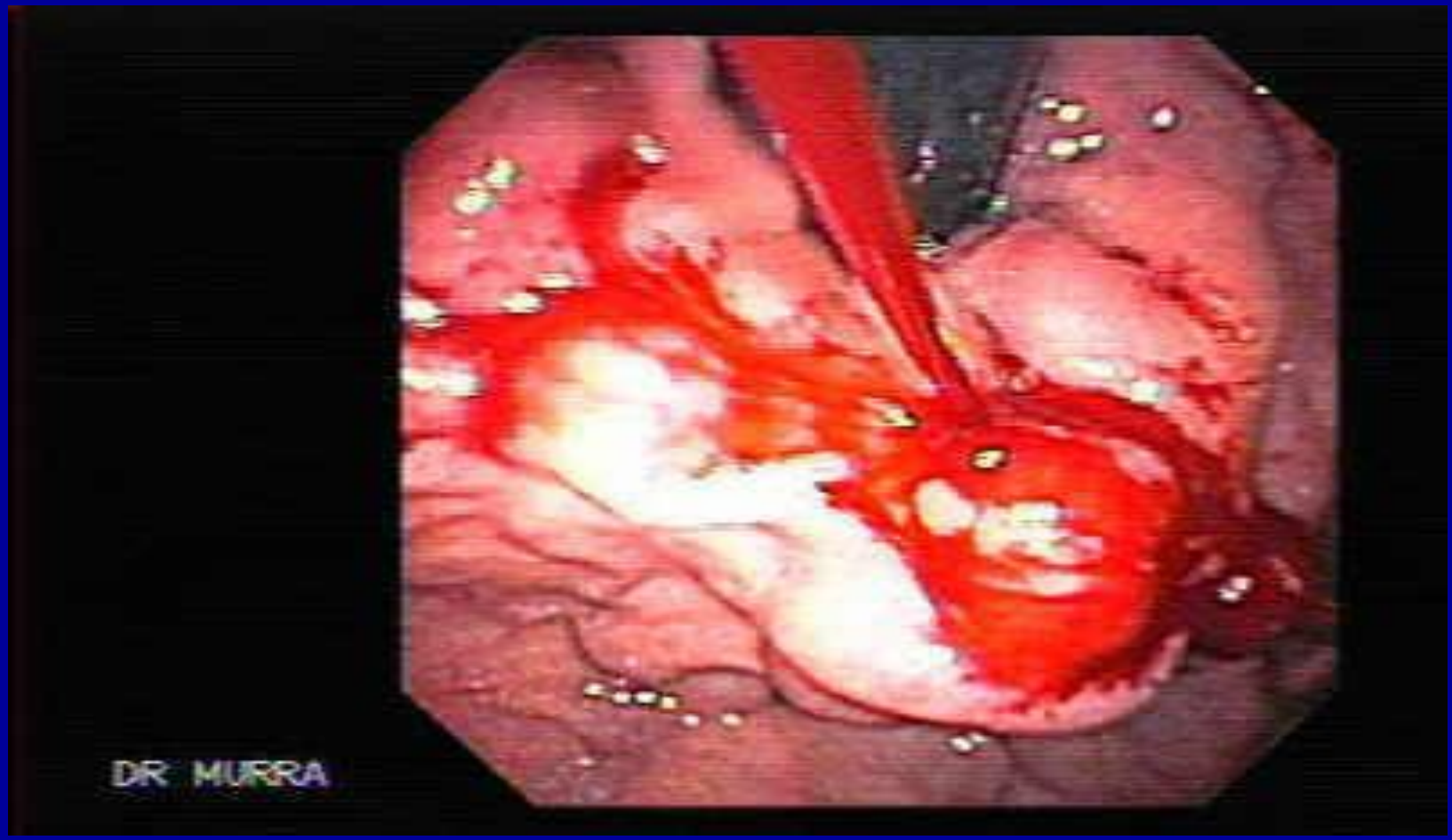
# Upper GI bleeding (above ligament of Treitz)



# UPPER GI BLEEDING

## Signs and Symptoms

- **Hematemesis** vomiting fresh blood
- **Melena** black, tarry, offensive in smell stool that is also loose & shiny
- **Dizziness** due to hypotension & volume loss
- **Abd. Pain and symptoms of Peptic ulcer disease**
- **Hx of NSAID's use**
- **Pallor** due to anemia
- **Hypotension**
- **Orthostasis** drop in BP while in standing position due to hypotension that may preceede the frank "
- **Jaundice and other stigmatas of chronic liver diseases**

So any patient with upper GI bleeding → you have to measure his BP while he's supine & while standing? he may have normal BP while supine, but his BP would drop upon standing up

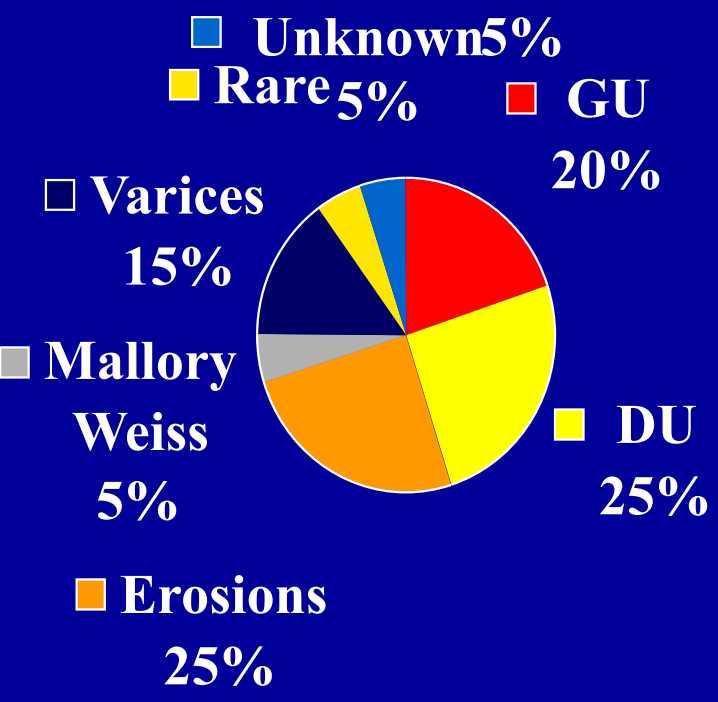
patients with massive upper GI bleeding
↳ fresh metal bleeding, blood is not having time to convert to the black color of melena (hematochezia)

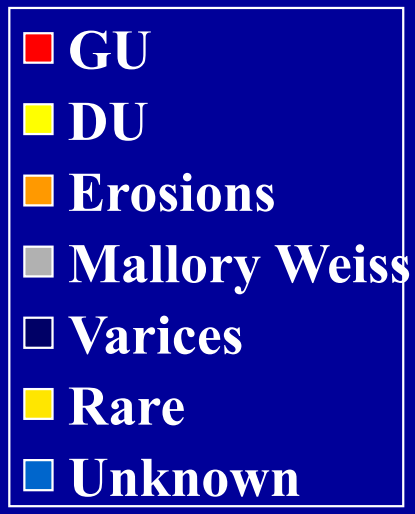
↑coughing ground vomiting

# UPPER GI BLEEDING

## CAUSES

1. mcc → PUD (gastric + duodenal ulcer)
2. esophageal varices
3. Mallory Weiss tear
4. erosions
5. other causes (ex: malignancies, arteriovenous malformations, haemobilia due to cholangiocarcinoma).




## RARE CAUSES

- Neoplasms
- AVM/Ectasia
- Dieulafoy's
- Stoma ulcers
- Esophageal ulcers
- Deodenitis
- Hemobilia
- Aorto-enteric fistulas
  - occurs in patients with aortic aneurysm that did a surgery for it, then a fistula forms between colon & aorta (MASSIVE BLEEDING)

# UPPER GI BLEEDING

## Peptic Ulcer Disease

- Defect in the GI mucosa extending through the muscularis mucosa.
- Decreasing incidence.
- Caused by imbalance between the aggressive and defensive factors.

# UPPER GI BLEEDING

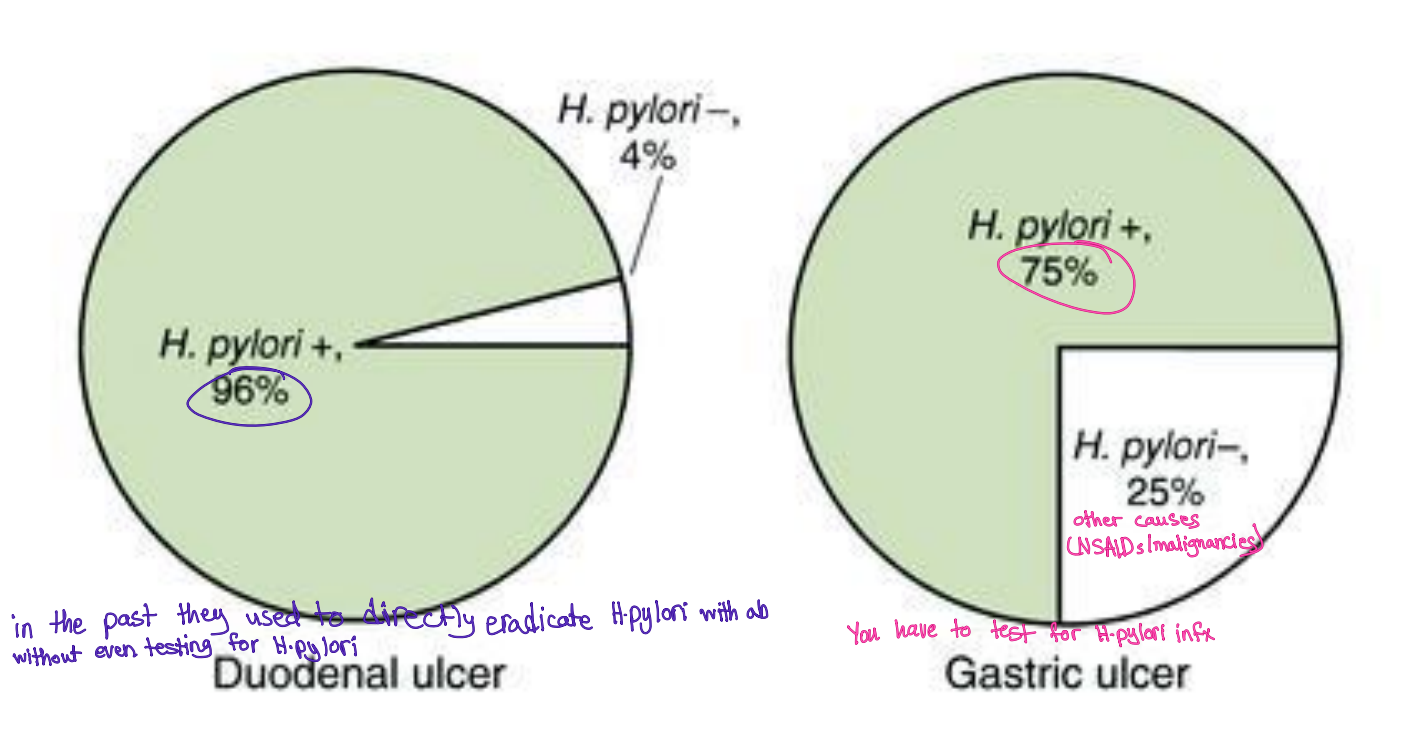
## Peptic Ulcer Disease

mcc

- Helicobacter Pylori
- NSAID's
- Acid Hypersecretory state.
- Antral G cell Hyperplasia

# UPPER GI BLEEDING

## Peptic Ulcer Disease



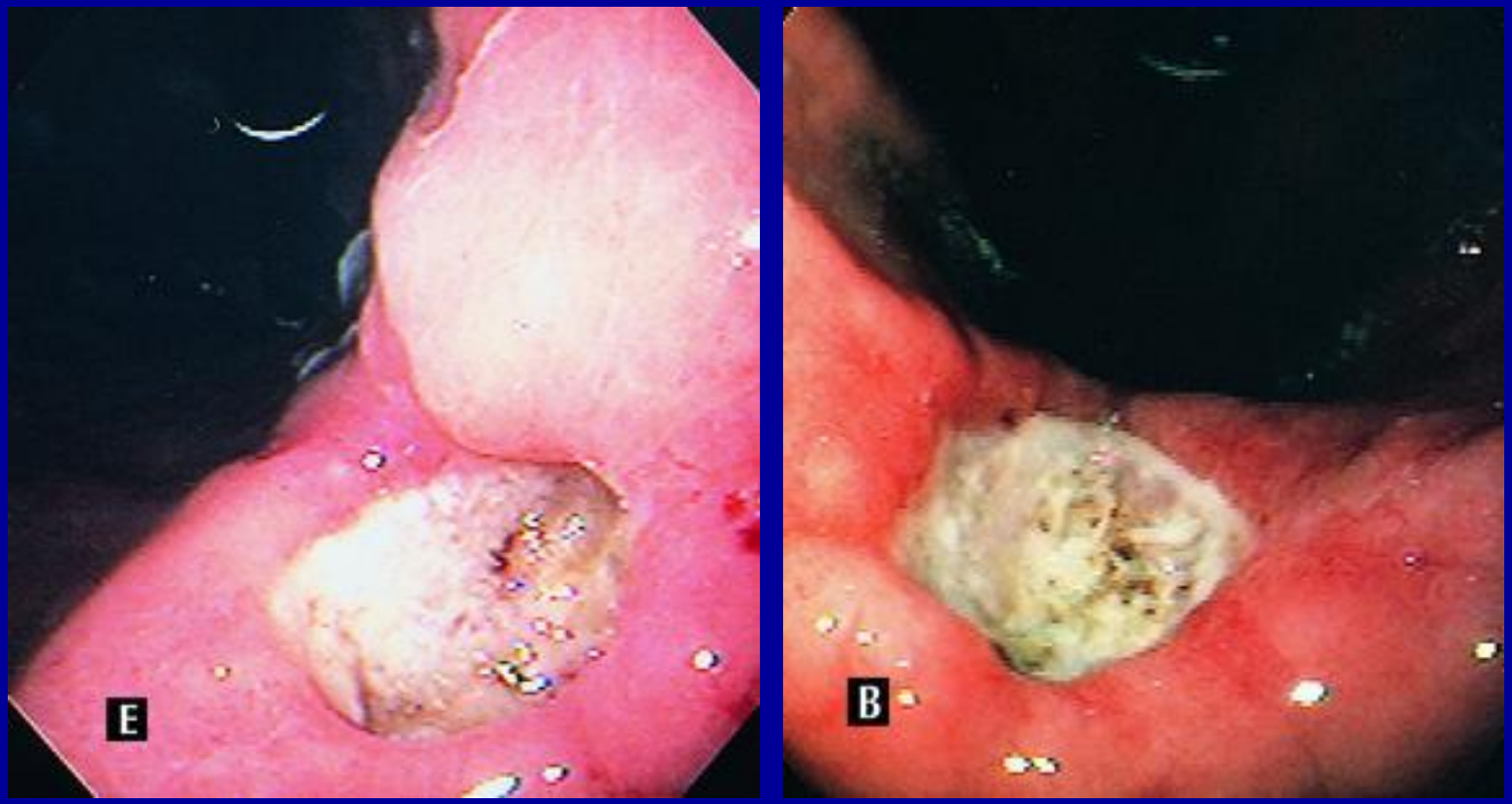

in the past they used to directly eradicate H.pylori with ab without even testing for H.pylori

You have to test for H.pylori infx

# UPPER GI BLEEDING

## Gastric Ulcers

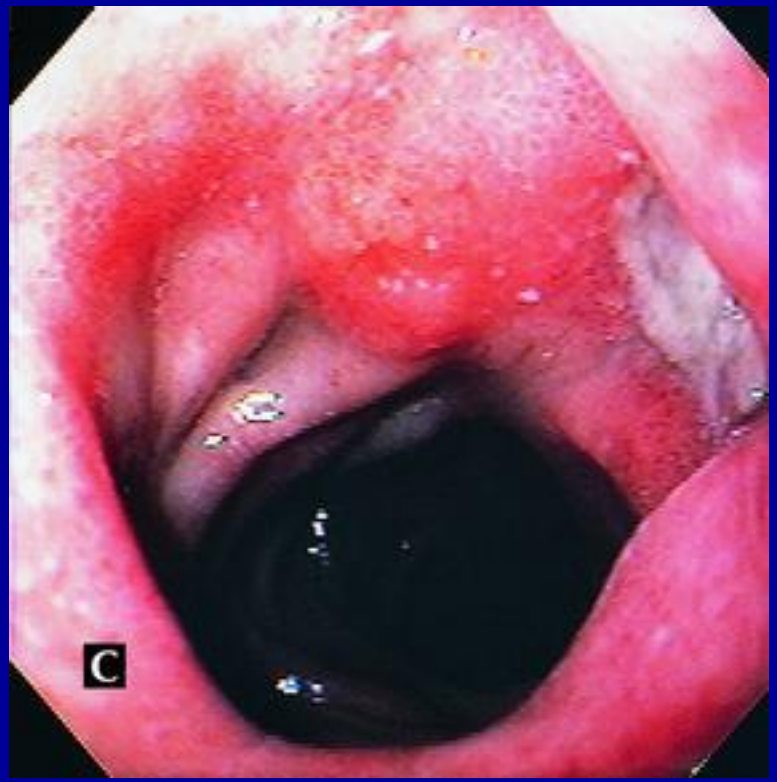
defect in normal mucosa
clean-based ulcer

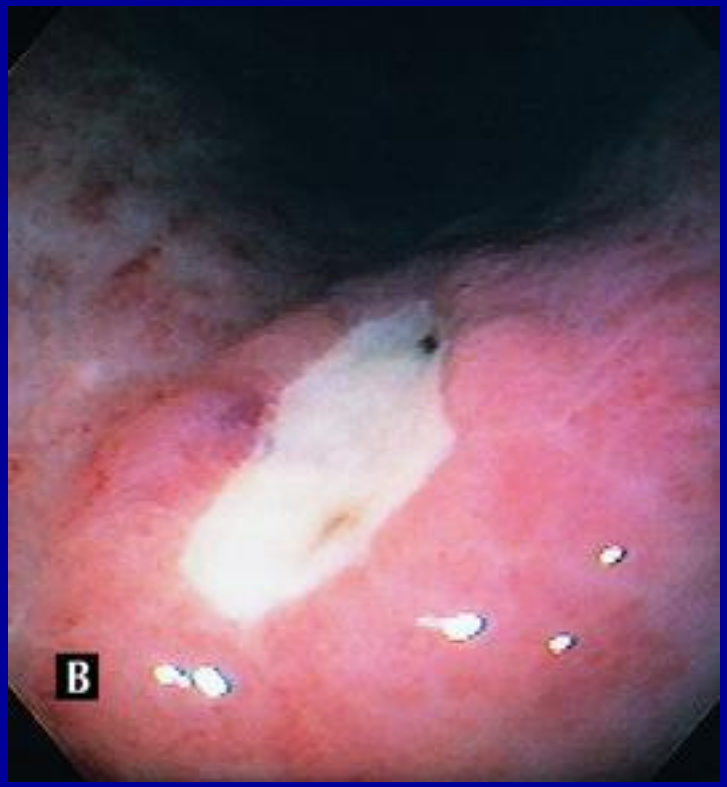


# UPPER GI BLEEDING

## Duodenal Ulcers

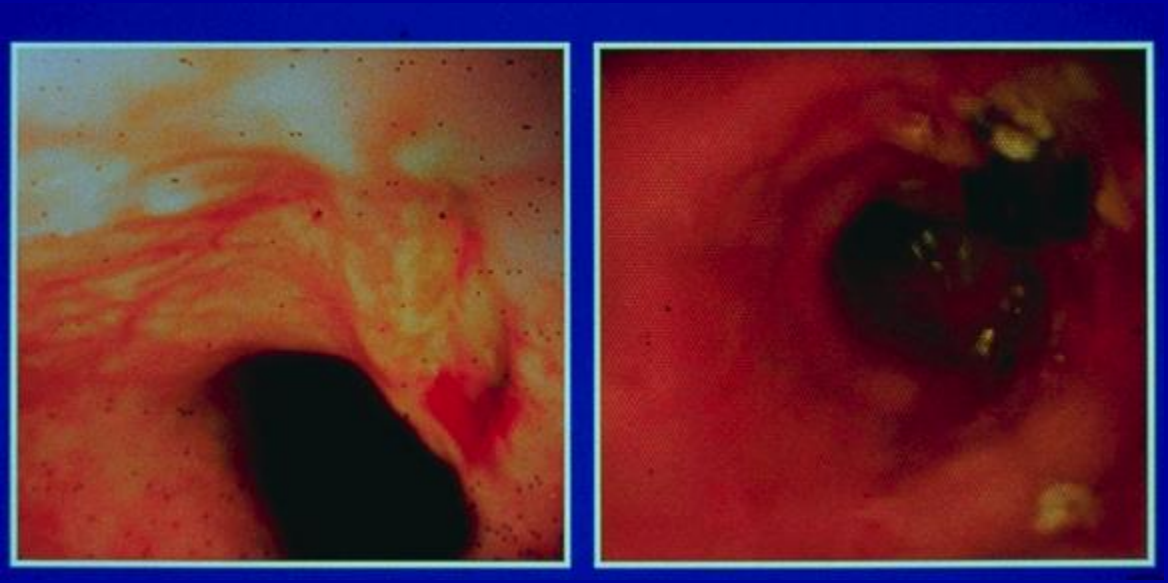
defect in healthy mucosa + surrounding inflammation



# UPPER GI BLEEDING

## Mallory - Weiss



(tear)

Laceration around the GE junction

Classical presentation as bleeding after episode of forceful vomiting

(ex: alcoholics, pregnant with hyperemesis gravidarum "excessive vomiting during the 1st trimester)

Classical presentation found in 50% only

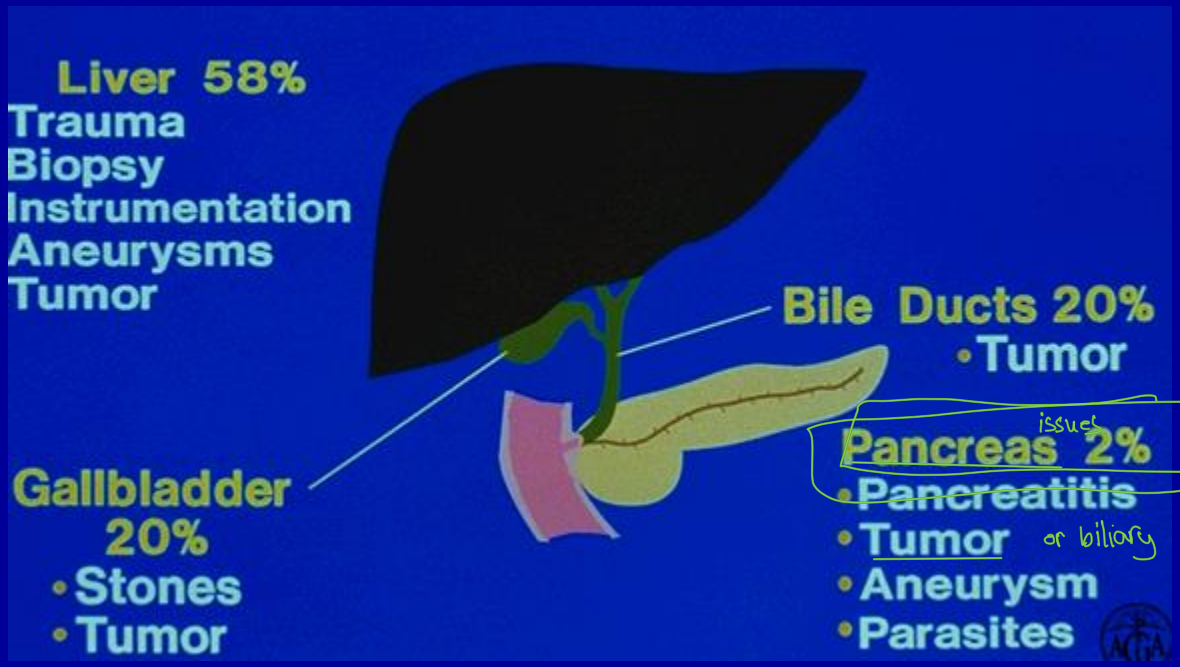
Self- limiting

usually minimal bleeding

# UPPER GI BLEEDING

## Hemobilia

blood coming out from bile tract



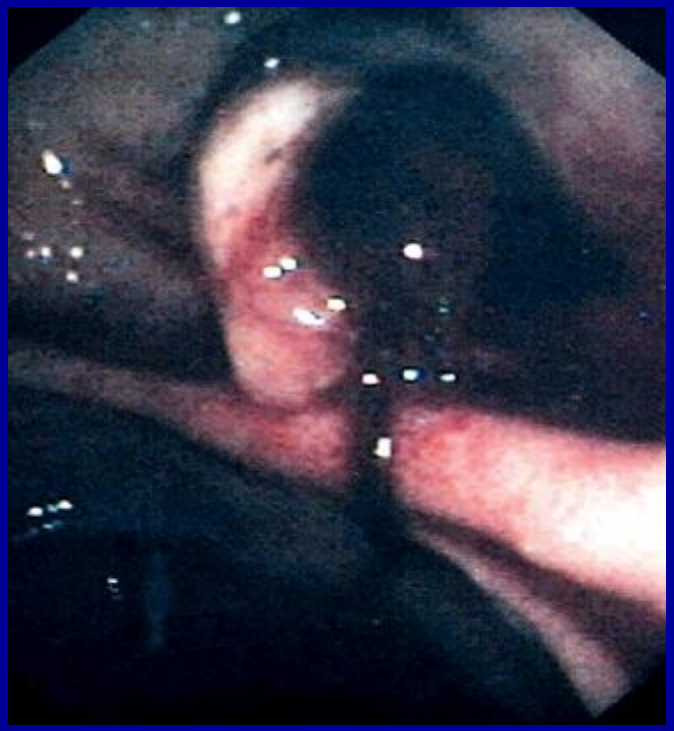

issues

or biliary cancer cholangiocarcinoma, stones ...

# UPPER GI BLEEDING

## Hemobilia

blood coming out from Ampulla of Vater

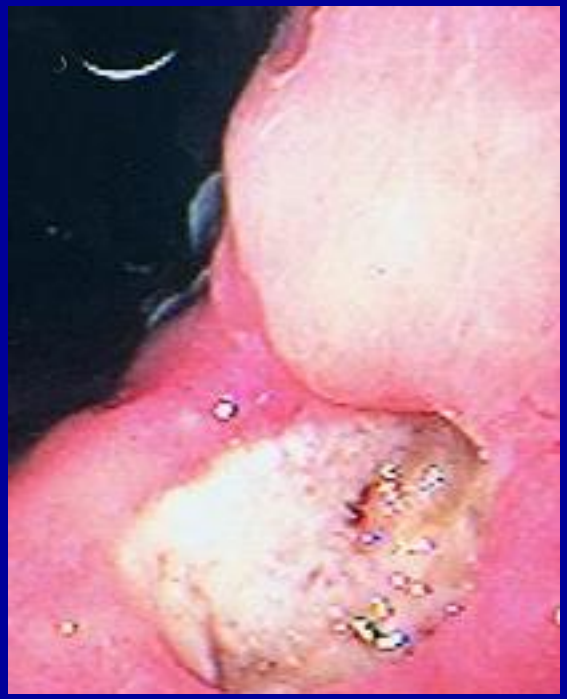


# UPPER GI BLEEDING

## stress ulcers

such as patients with head injury or burn patients (NOT related to H.pylori infx or NSAIDs uses)

- Caused by Vagal hyperstimulation and vascular hypoperfusion.
- Body and fundus more affected
- Multiple ≠ in H.pylori PUD → antrum* in critically ill patients
- Prophylaxis is indicated in critically ill ICU patients



Curling Extensive burn

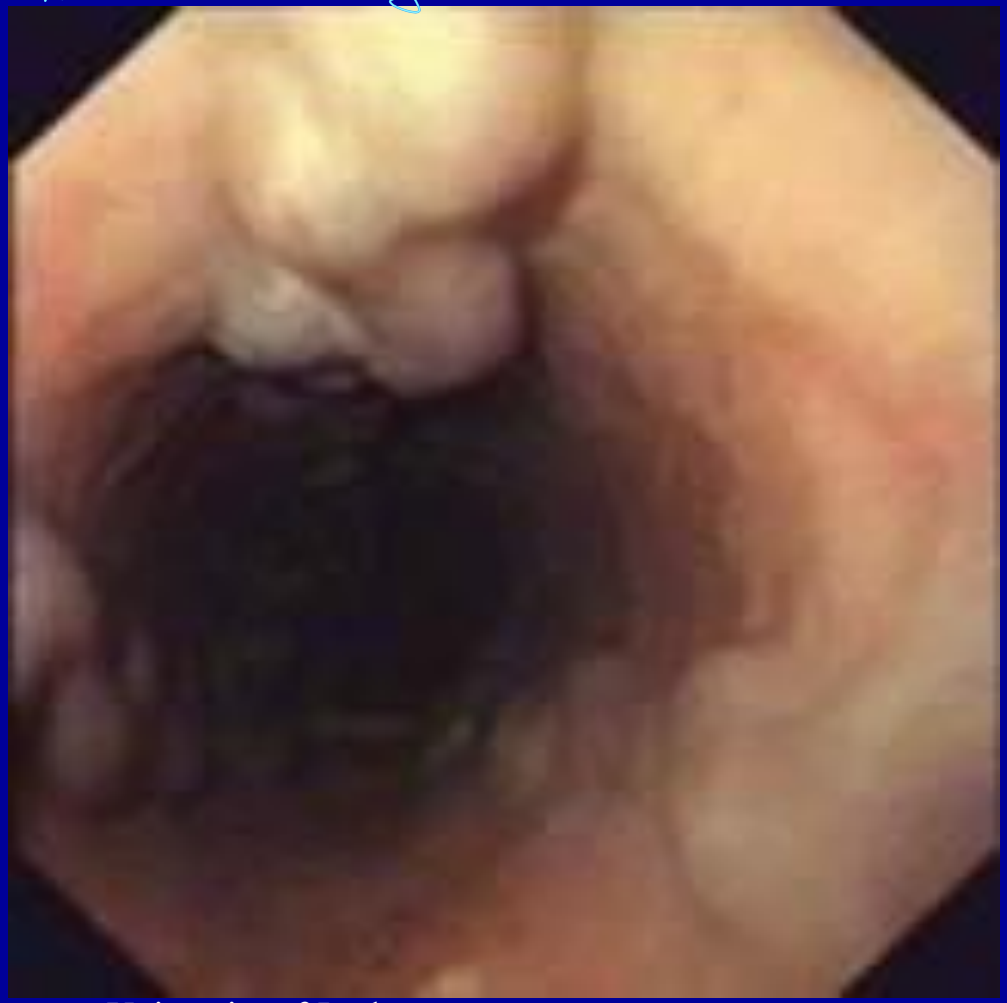
Cushing Head Injury

both are stress ulcers

# UPPER GI BLEEDING

## BLEEDING ESOPHAGEAL VARICEAL

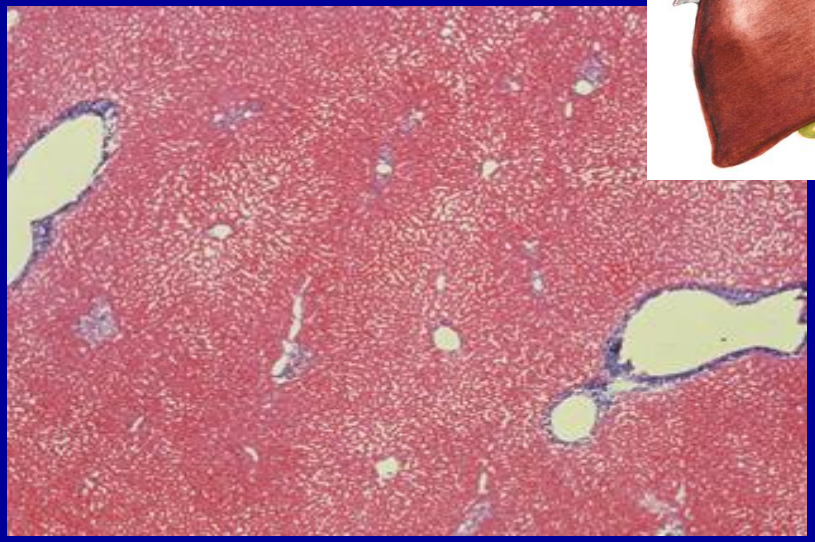
upper endoscopy image:

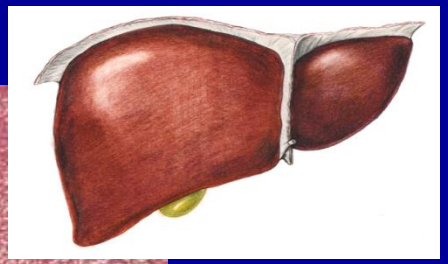


- Dilated tortuous veins of the lower and mid esophagus. (distal)
- Secondary to portal HTN
- 30% mortality after the first episode. (very high)
- 60% Rebleeding rate

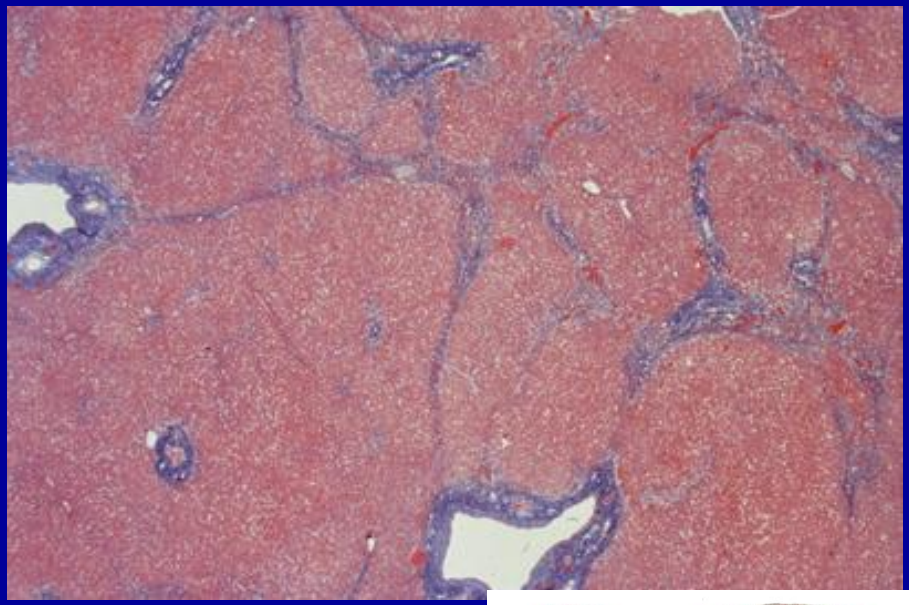
# Cirrhosis and Portal hypertension

## Healthy Liver



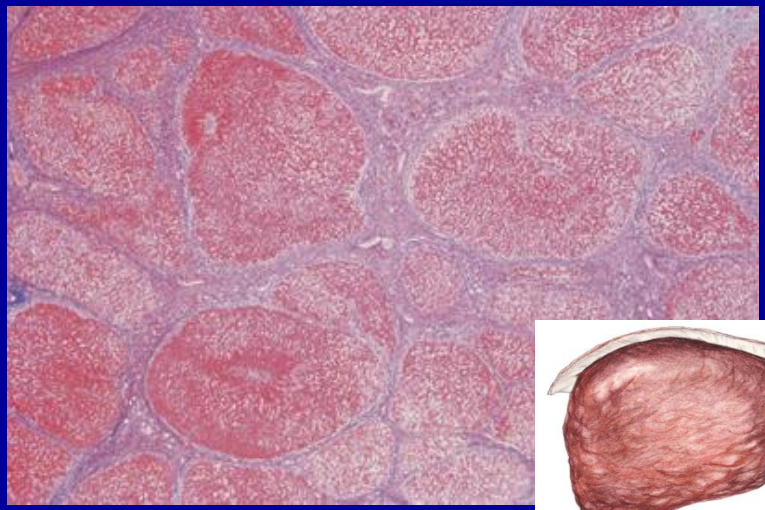


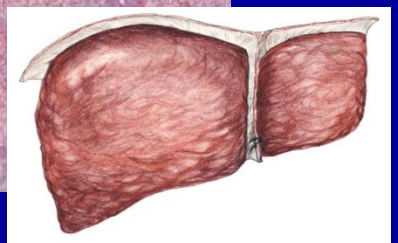
## Liver Fibrosis

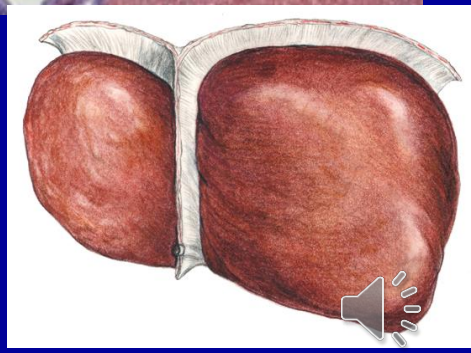


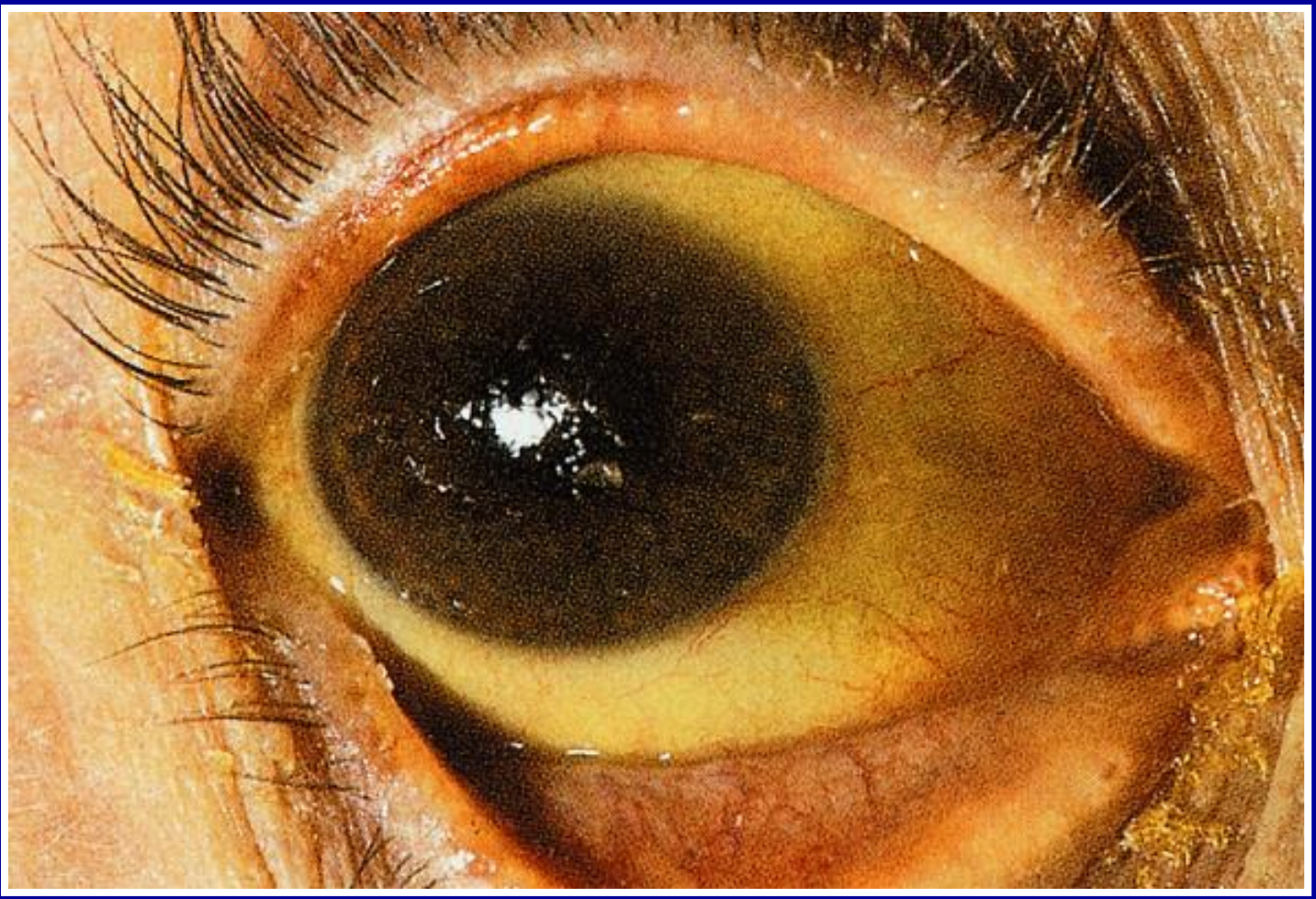
## Cirrhosis

nodular









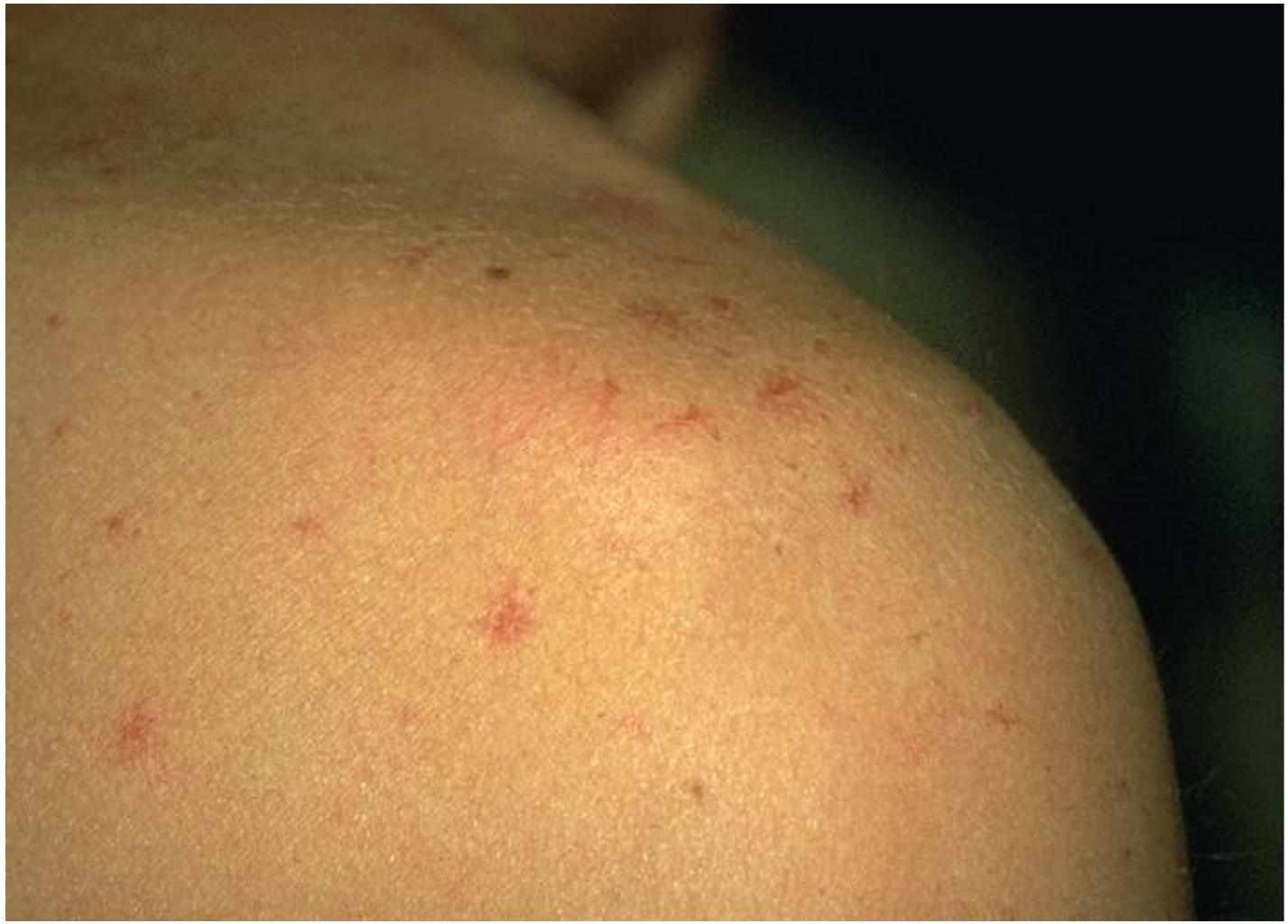
common manifestation of liver cirrhosis

# Jaundice

> 2.5 mg/dL or 3 → clinically detect jaundice

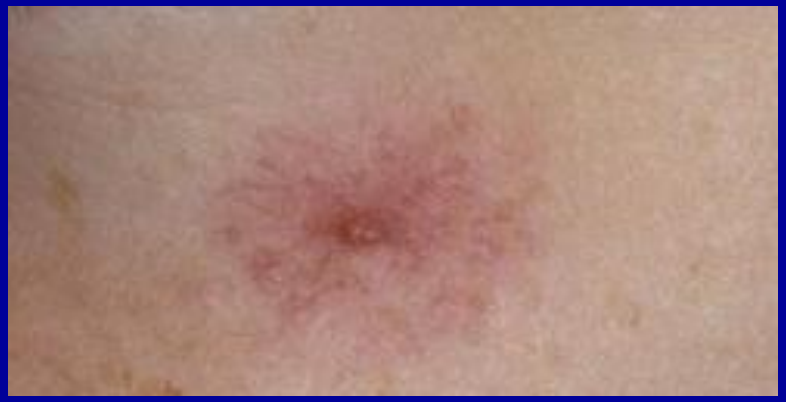
Accumulation of bilirubin in the blood stream causing yellowish discoloration of plasma and heavily perfused tissues

↳ sclera & mucus membranes



## Spider angiomas

- on ant. & post. part of chest
- capillaries like spiders due to hyperestrogenemia in patients with liver cirrhosis



## Spider Angiomas

Small, centrally raised bumps (papules) caused by a dilated arteriole (small artery). A network of dilated capillaries (tiny blood vessels) radiate from the arteriole. Pressing on the lesion causes the redness to disappear briefly, and there is a rapid return of redness once the pressure is lifted.

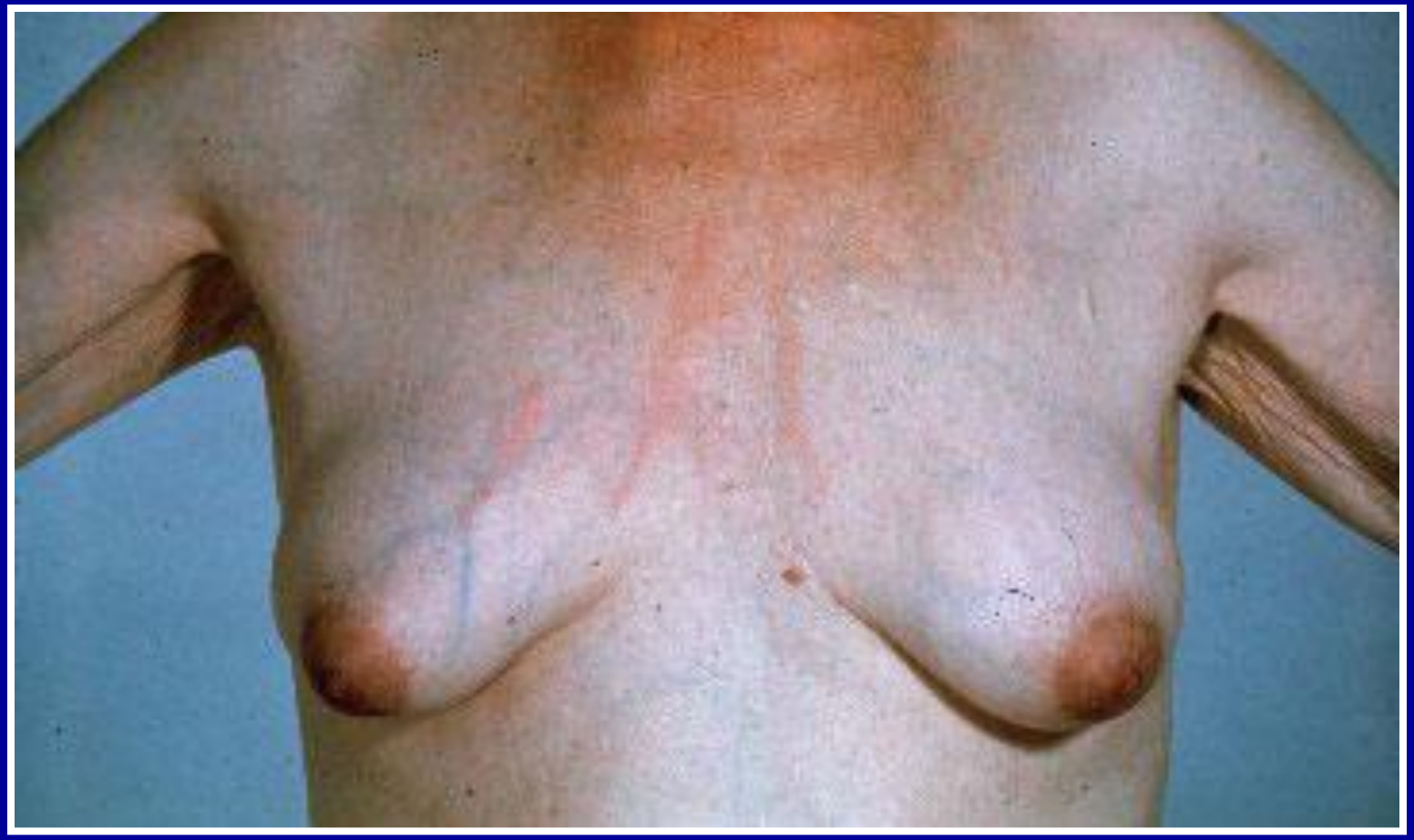
# Finger Clubbing

a condition where there is enlargement of the terminal end of the digit over the distal phalanx.

It is usually symmetrical and affects the fingers

*The angle between the nail & nailbed is lost at the beginning, then the curvature of the nail increases with time.
*Can be familial

*Common manifestation of liver cirrhosis, but it can occur in other diseases (ex: IBD, cyanotic congenital ♡ diseases)



Gynecomastia due to liver cirrhosis in men.

Breast development in men

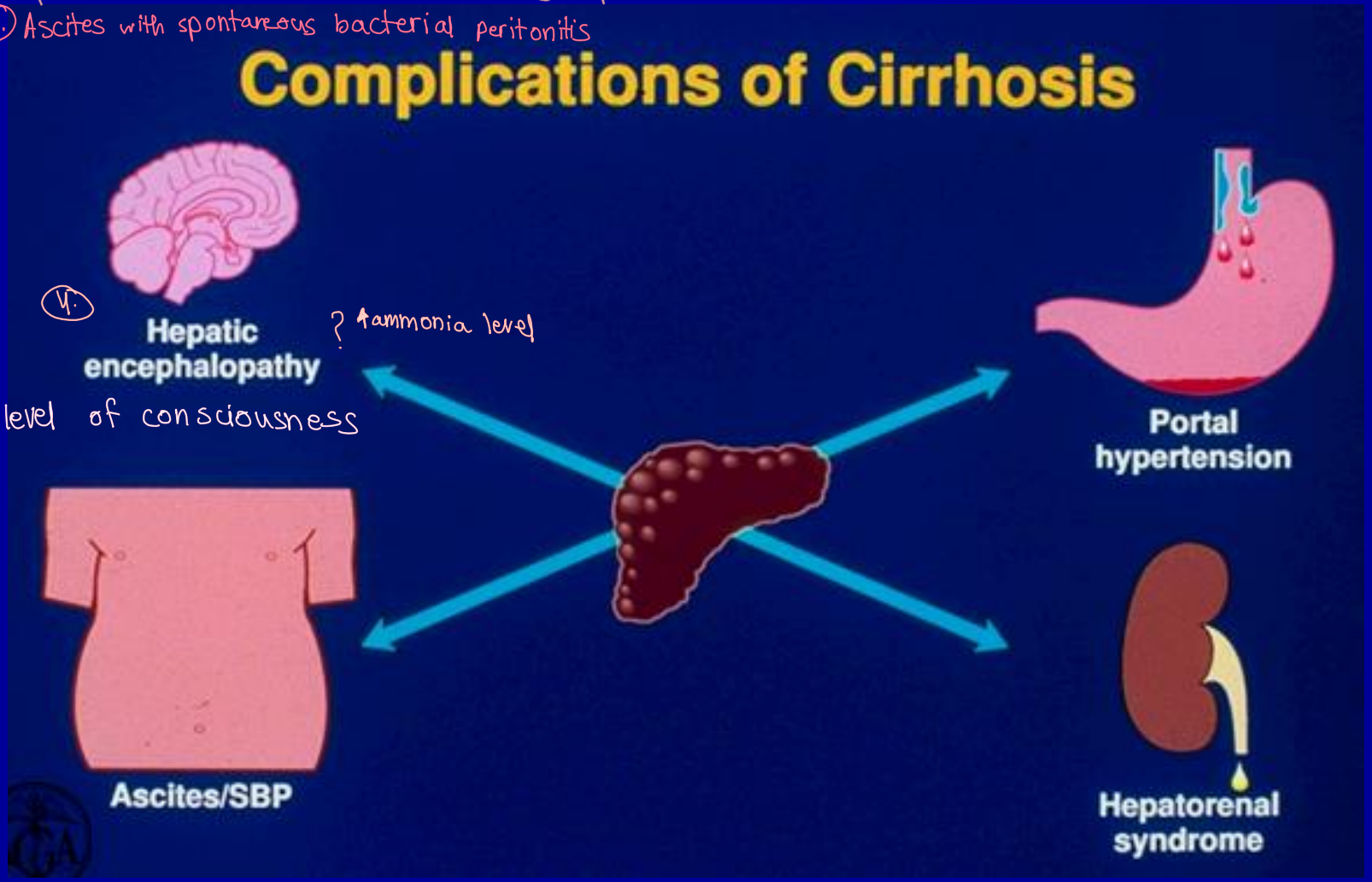
While women → breast atrophy

# Dupuytren's Contractures

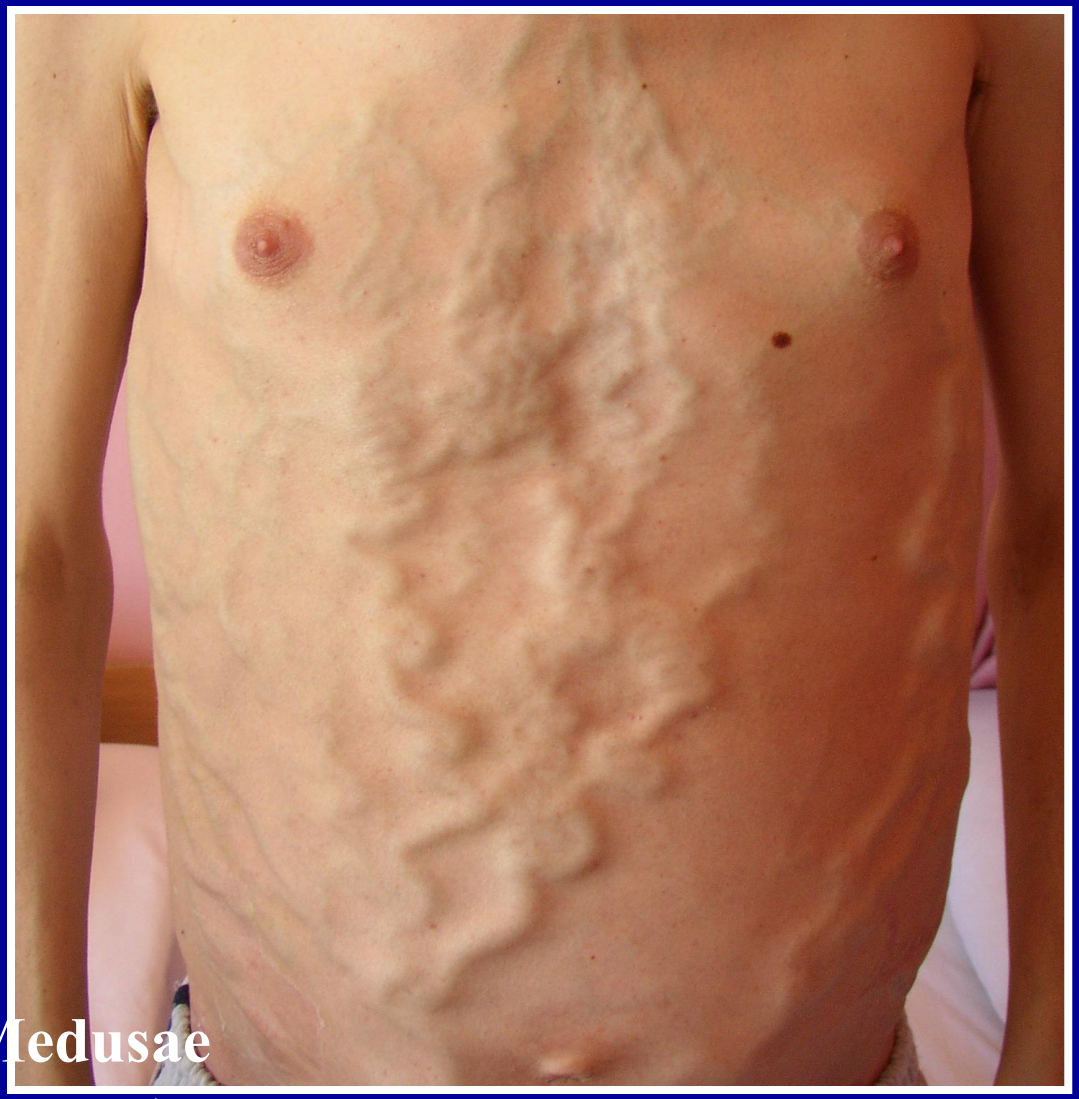
liver cirrhosis

Joint contractures

1. varicial bleeding (↑mortality rate)
2. hepatorenal disease (acute kidney injury in patients with advanced liver disease)
3. Ascites with spontaneous bacterial peritonitis

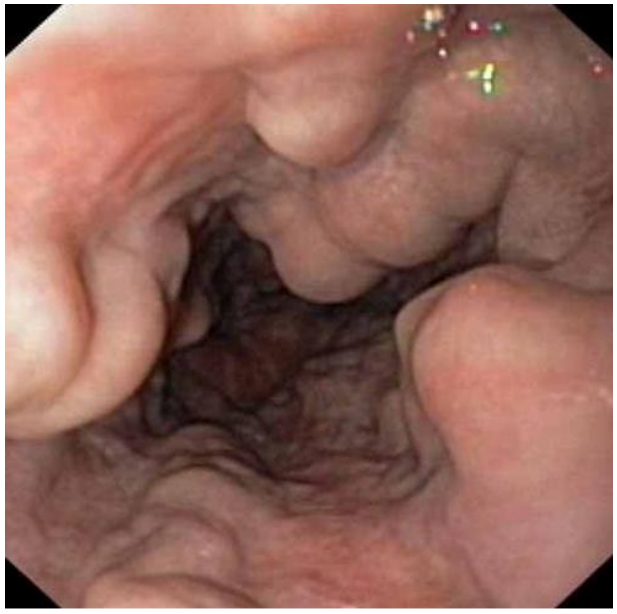


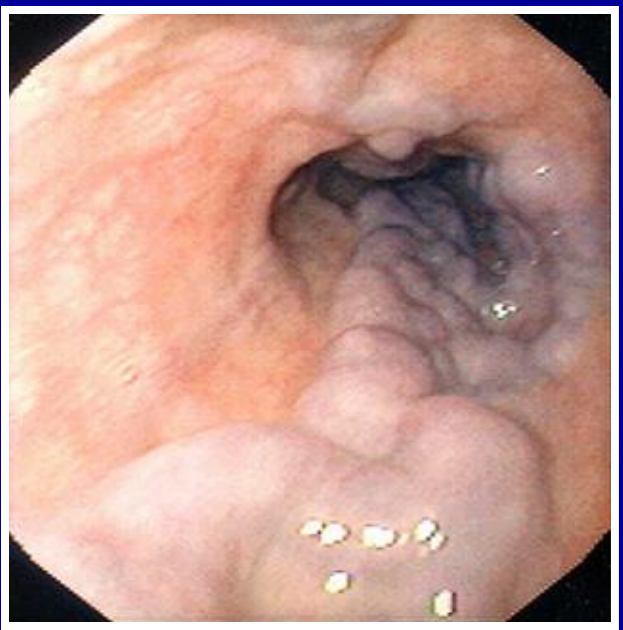

4. ? ↑ammonia level
5. ↓level of consciousness

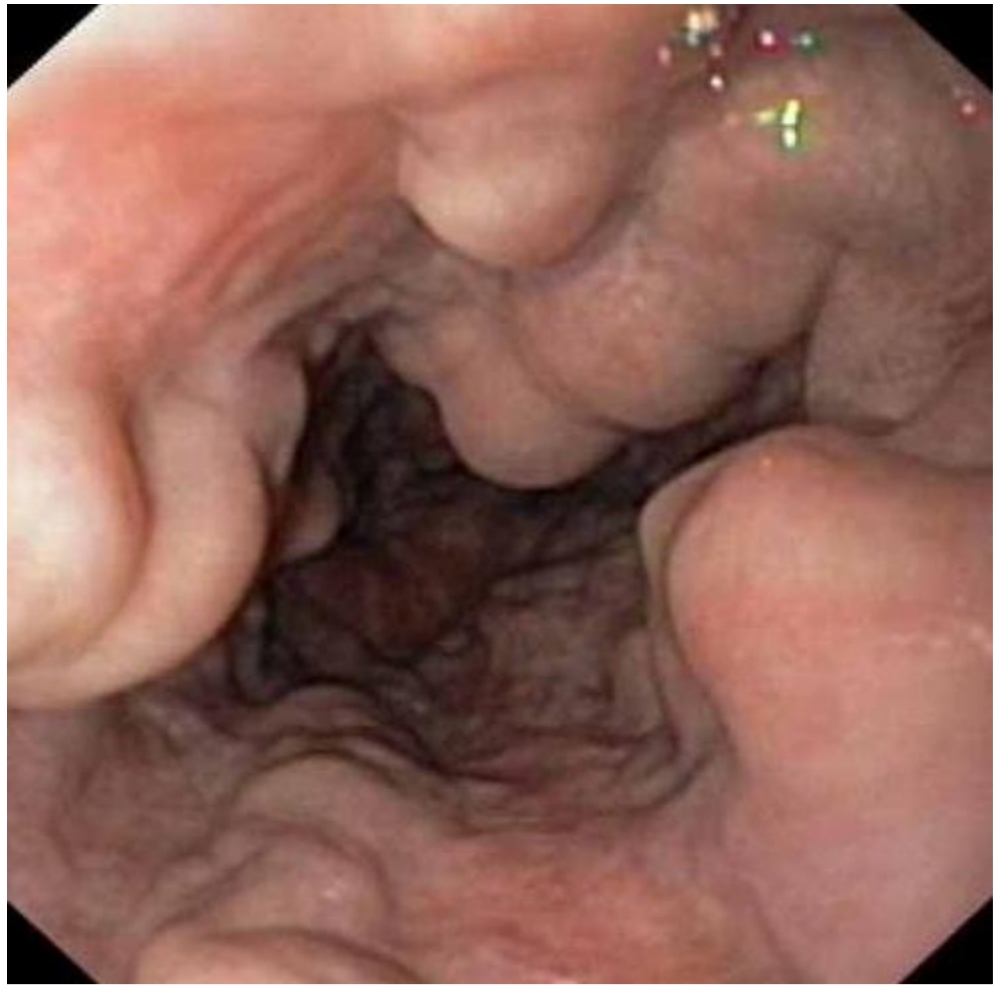


**Caput Medusae**

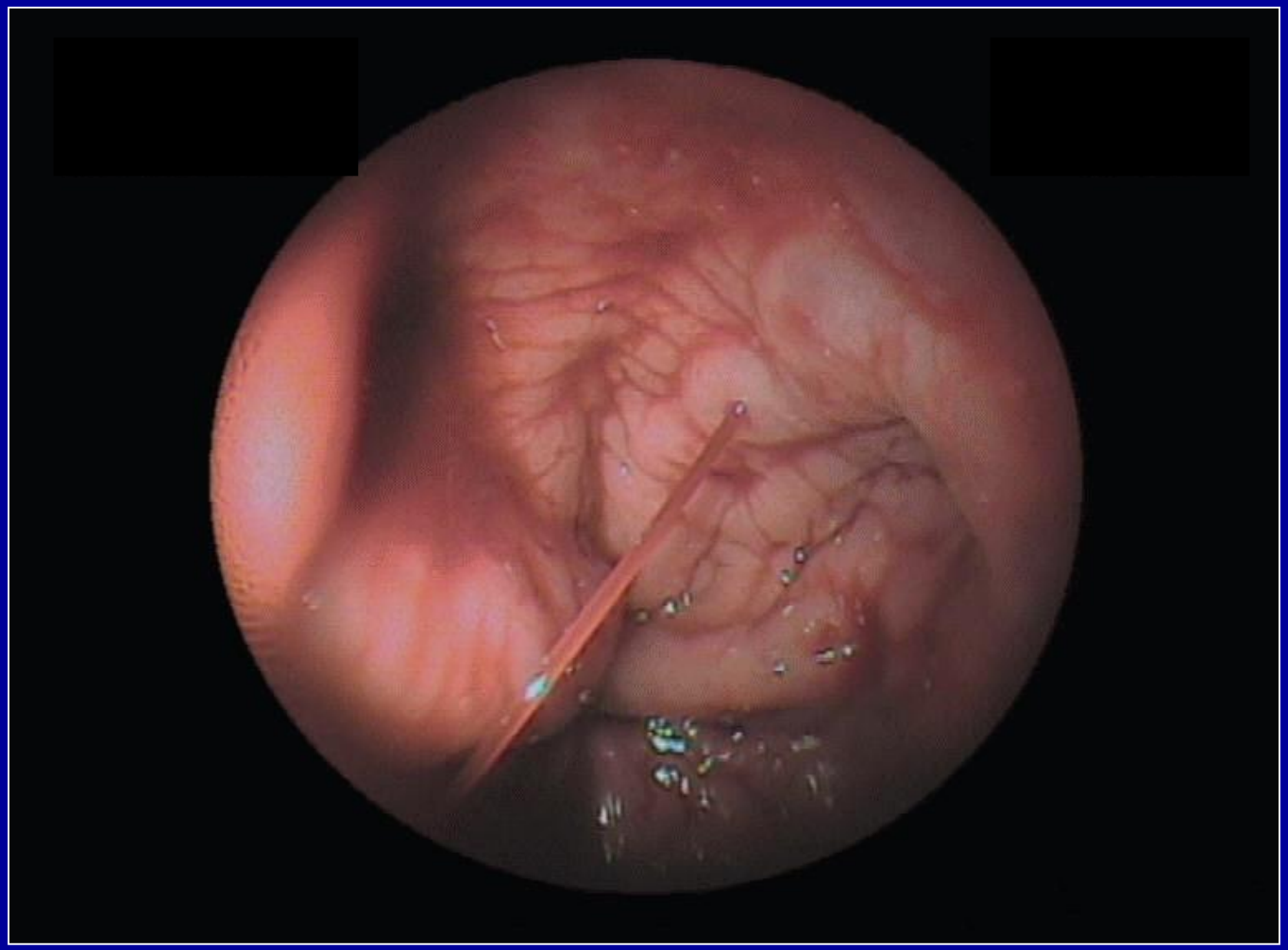
Distended (dilated) and engorged umbilical veins which are seen radiating from the umbilicus across the abdomen to join systemic veins. (on ant. abdominal wall.)



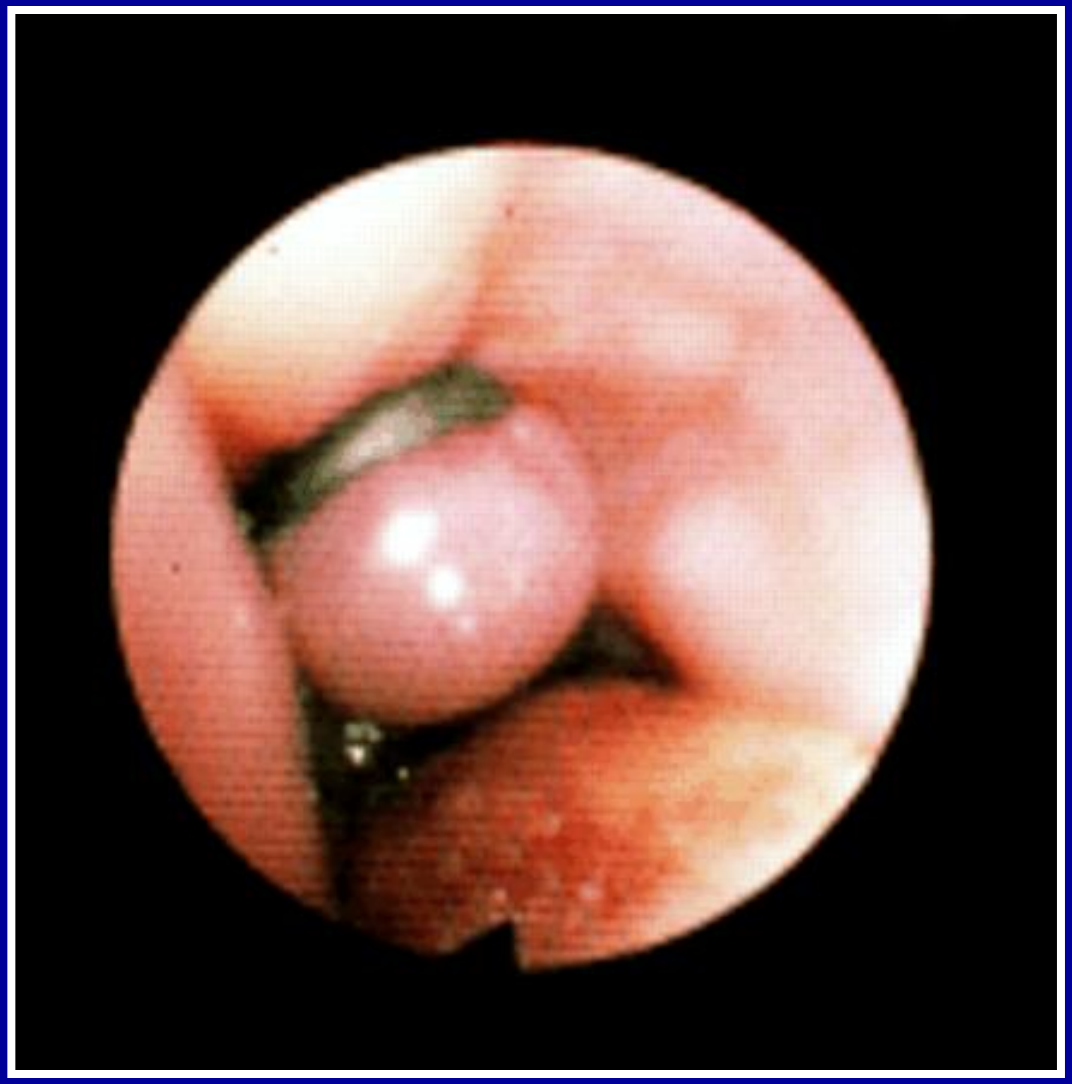




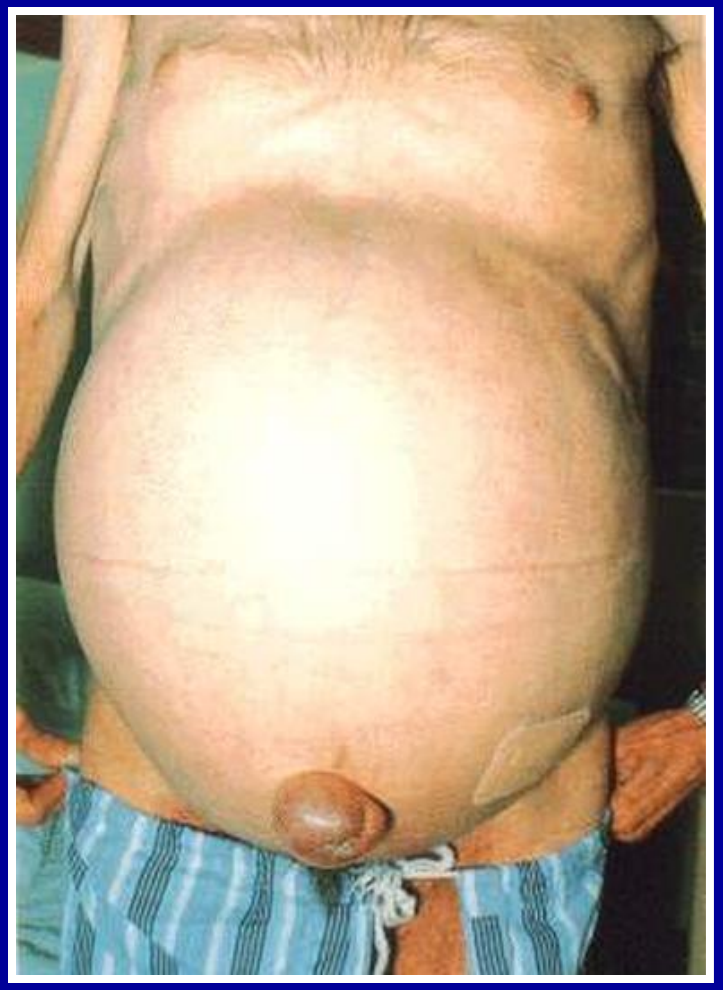
esophageal varices

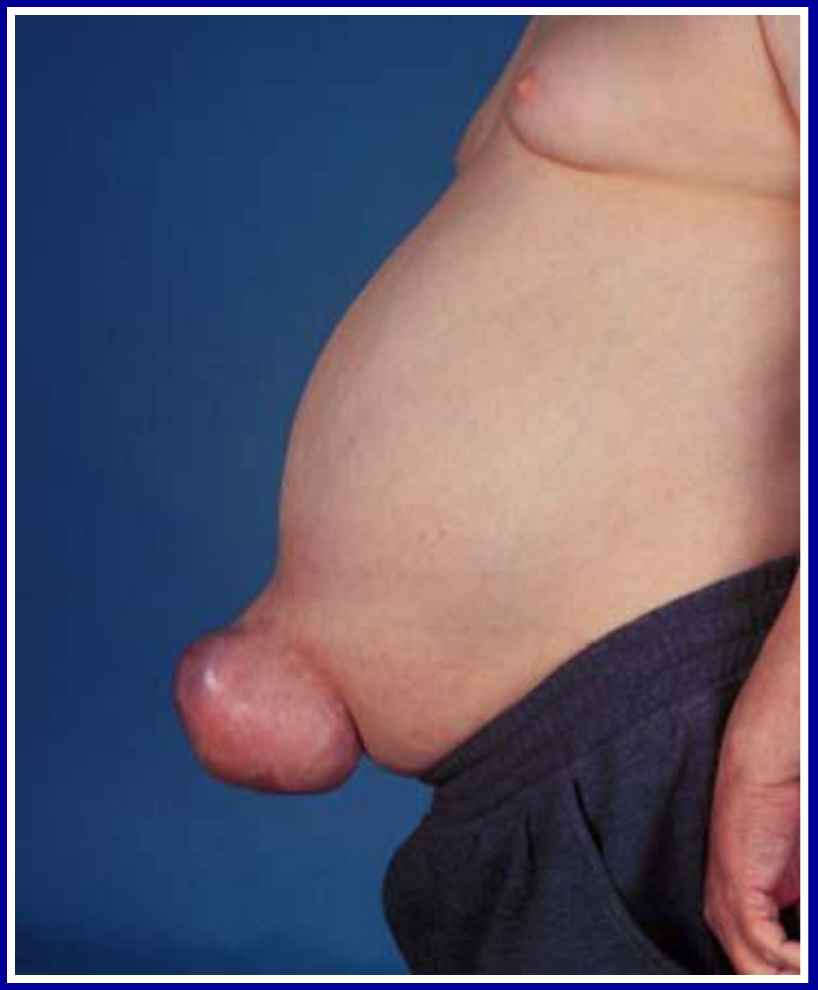


# esophageal band ligation

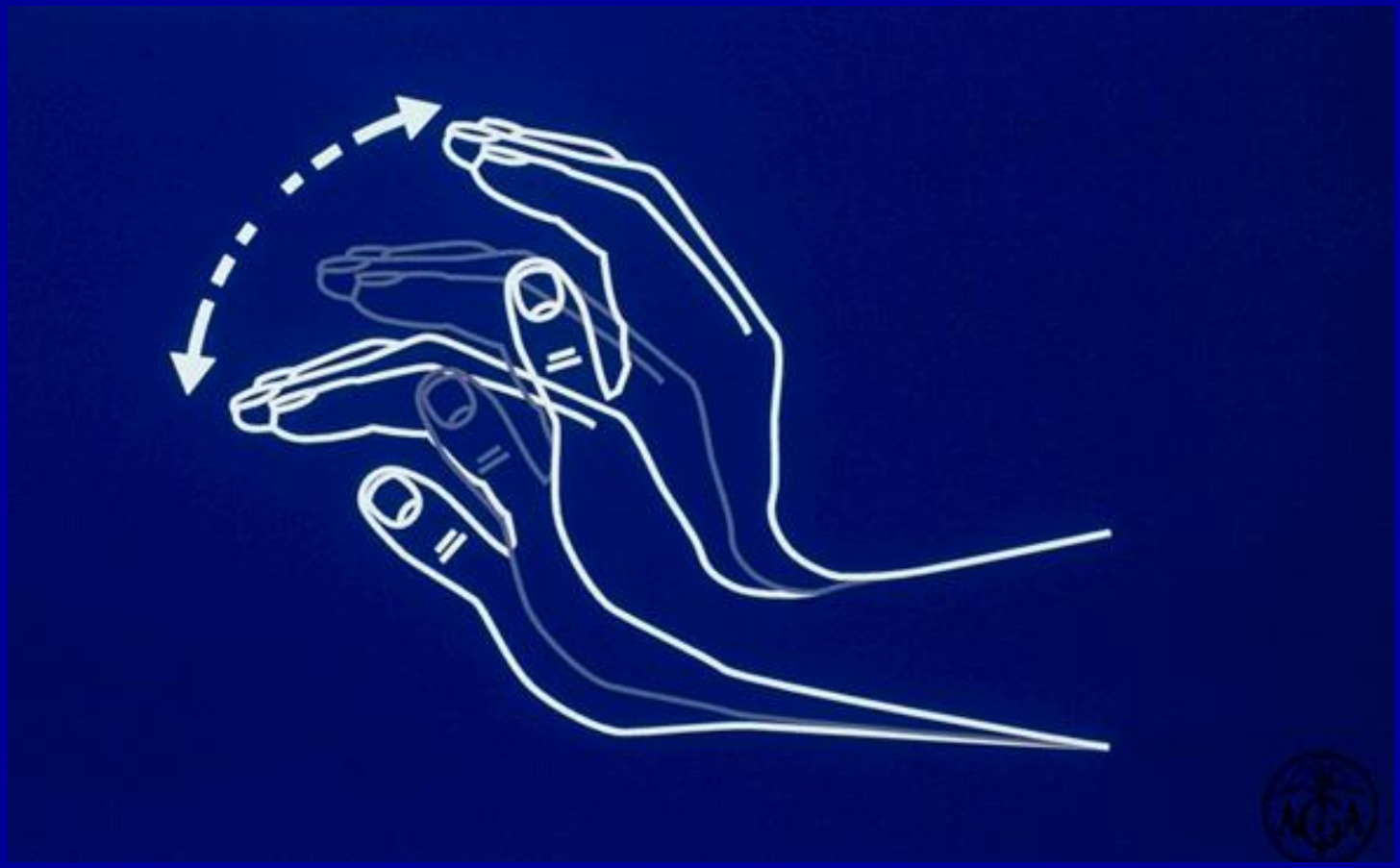


we apply during endoscopy as a trx option for patients with bleeding esophageal varices





massive ascites +umbilical hernia due to



## Astraxia

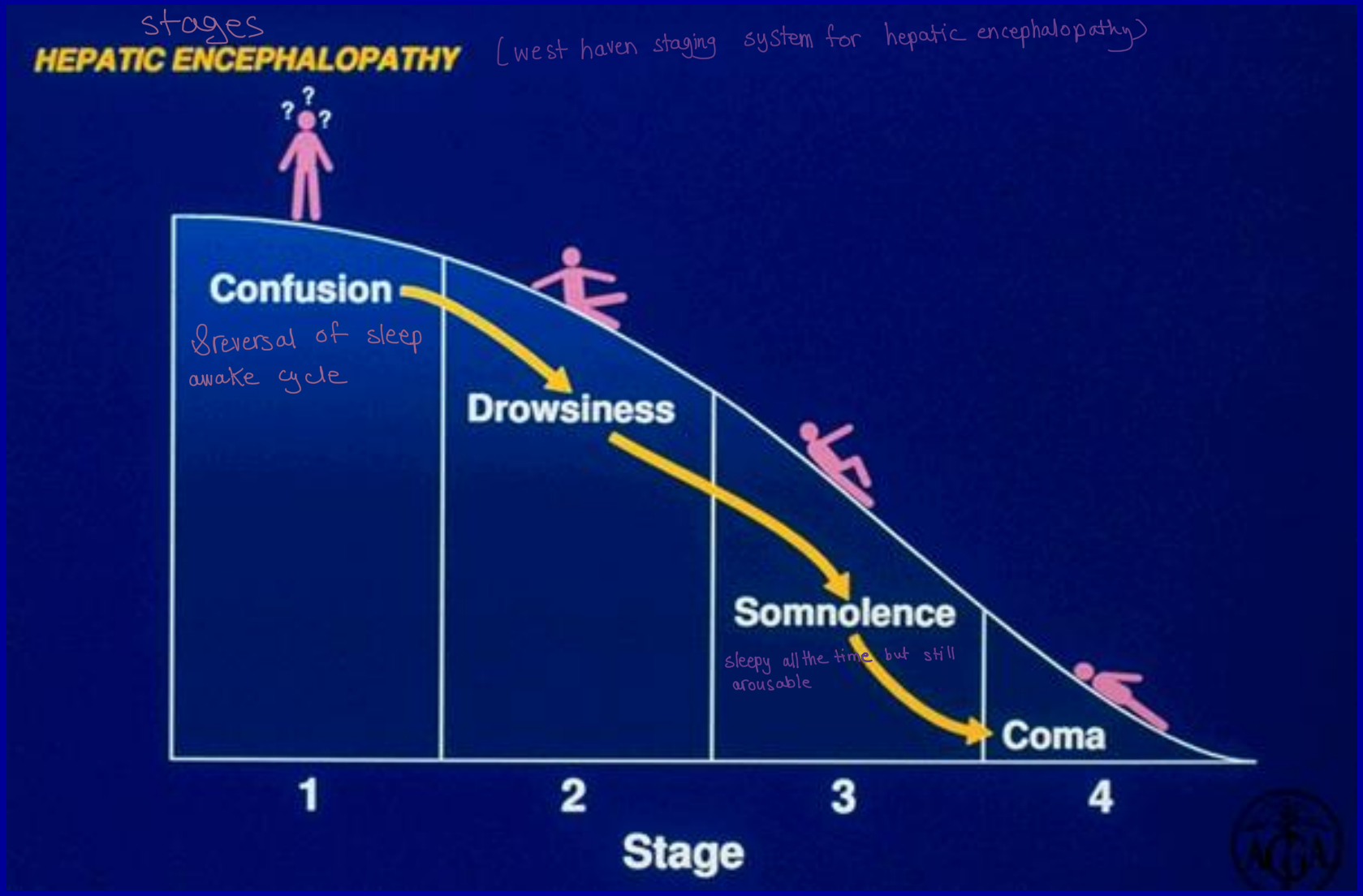
hepatic encephalopathy + ♡ failure + respiratory failure

Flapping tremors, quick arrythmic movement in back ground tonic muscle contracion

stages

# HEPATIC ENCEPHALOPATHY

(west haven staging system for hepatic encephalopathy)



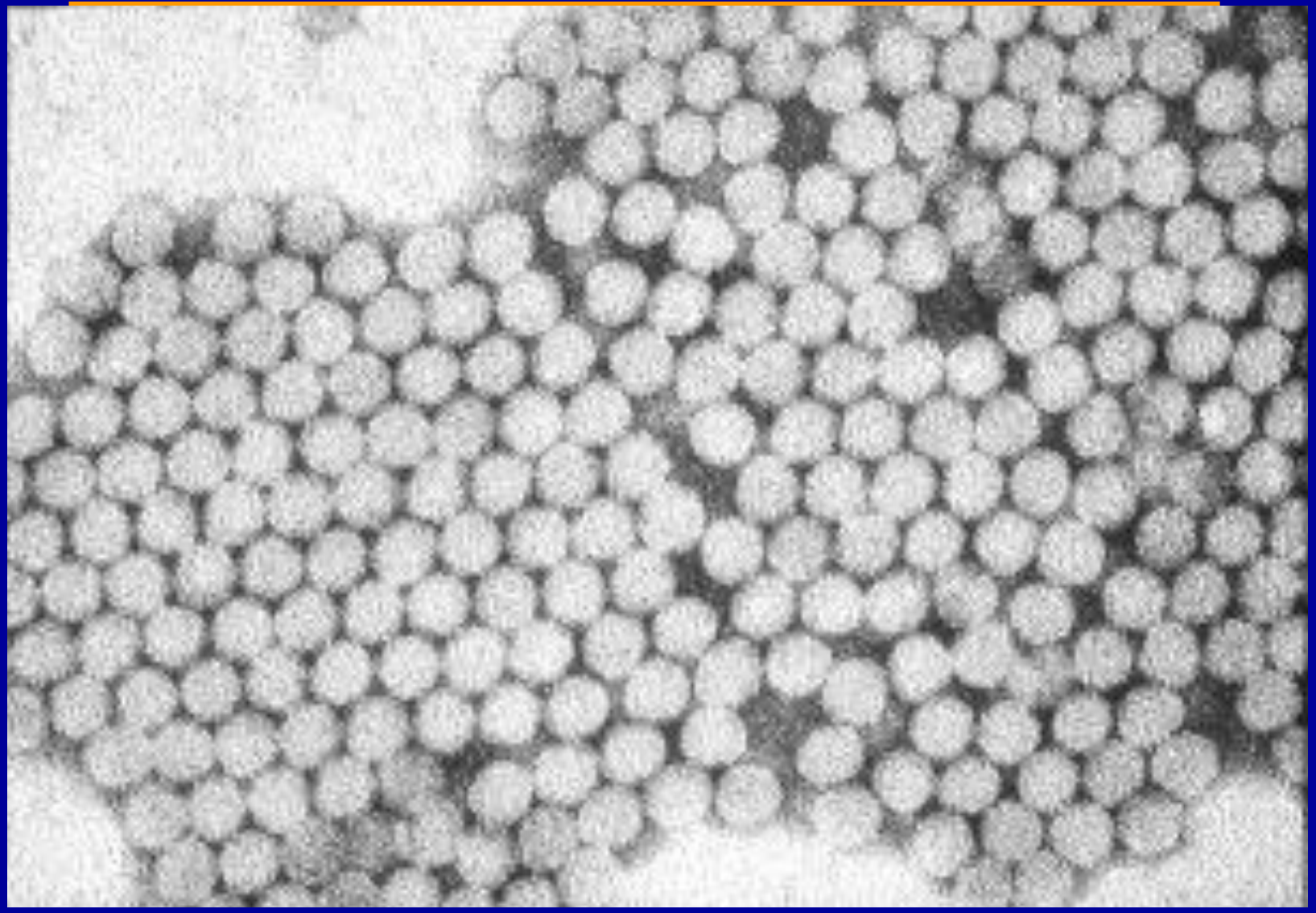
# Hepatitis A-E Viruses

## An Overview

# Type of Hepatitis

| | A | B | C | D | E |
|---|---|---|---|---|---|
| Source of virus | feces | blood/ blood-derived body fluids | blood/ blood-derived body fluids | blood/ blood-derived body fluids | feces |
| Route of transmission | fecal-oral | percutaneous permucosal | percutaneous permucosal | percutaneous permucosal | fecal-oral |
| Chronic infection | no | yes | yes | yes | no |
| Prevention | pre/post-exposure immunization | pre/post-exposure immunization | blood donor screening; risk behavior modification | pre/post-exposure immunization; risk behavior modification | ensure safe drinking water |

# Hepatitis A Virus



# Hepatitis A - Clinical Features

- Incubation period: Average 30 days
  Range 15-50 days
- Jaundice by age group: <6 yrs, <10%
  6-14 yrs, 40%-50%
  >14 yrs, 70%-80%
- Complications: Fulminant hepatitis
  Cholestatic hepatitis
  Relapsing hepatitis
- Chronic sequelae: None

- pediatric age group will NOT show the illness, it'll come just like any viral infx: slight fever, slight jaundice (not noticed)
- Adults → jaundice, abdominal pain, vomit

rare

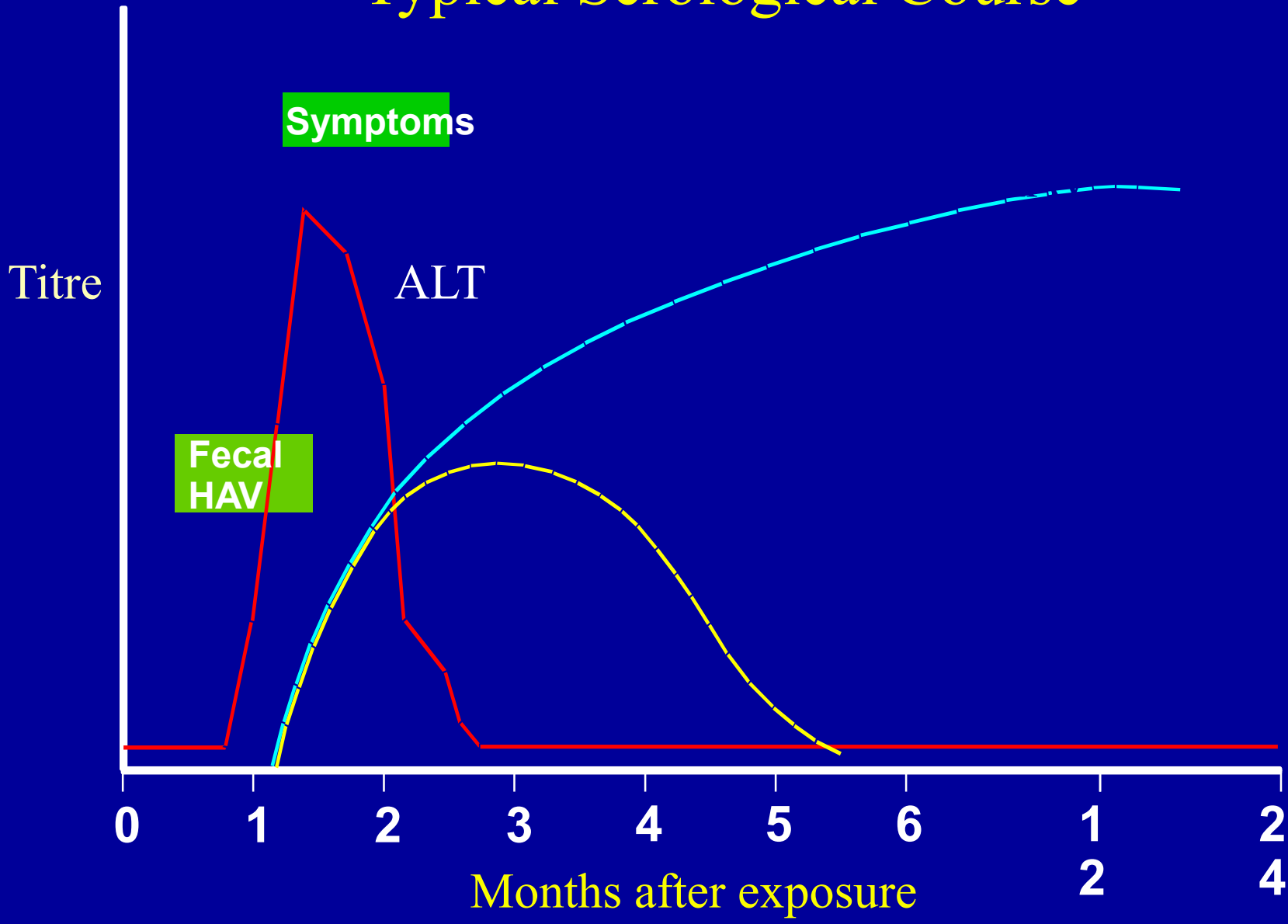
< 1% cases with acute liver failure → liver transplant

recover the virus (most patients)

< 1% fulminant hepatitis < die / liver transplant

# Hepatitis A Infection

## Typical Serological Course



# Hepatitis A Virus Transmission

feco-oral

- **Close personal contact**
  **(e.g., household contact, sex contact, child day care centers)**
- **Contaminated food, water**
  **(e.g., infected food handlers, raw shellfish)**
- **Blood exposure (rare)**
  **(e.g., injecting drug use, transfusion)**

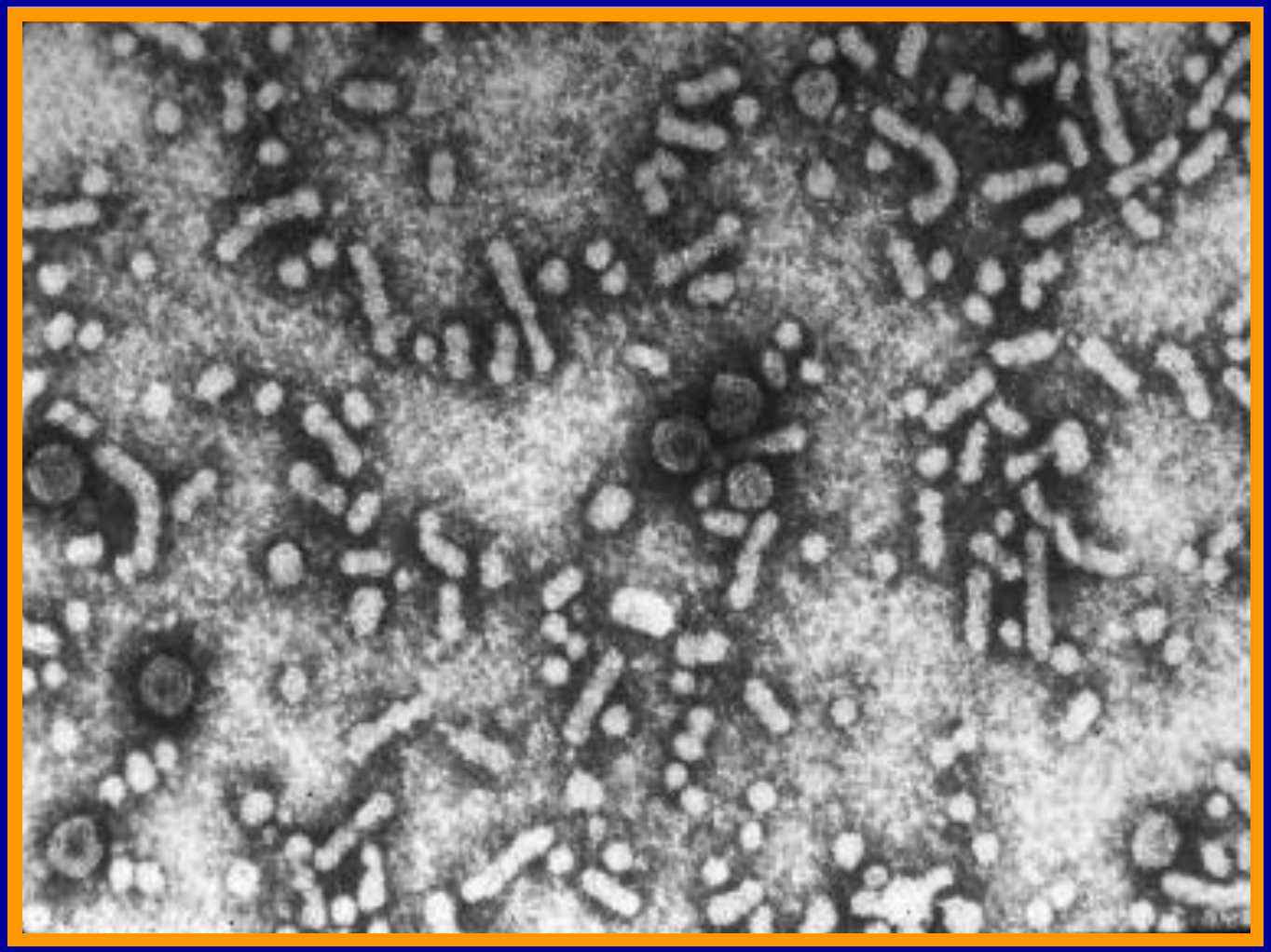
# Laboratory Diagnosis

- **Acute infection is diagnosed by the detection of HAV-IgM in serum by EIA.**
- **Past Infection i.e. immunity is determined by the detection of HAV-IgG by EIA.**

(remote infx)

*Vaccine → for people at risk (cirrhosis, chronic HBV)
Xnational program

# Hepatitis B Virus



# Hepatitis B - Clinical Features

- Incubation period: Average 60-90 days<br>Range 45-180 days
- Clinical illness (jaundice): <5 yrs, <10%<br>5 yrs, 30%-50%

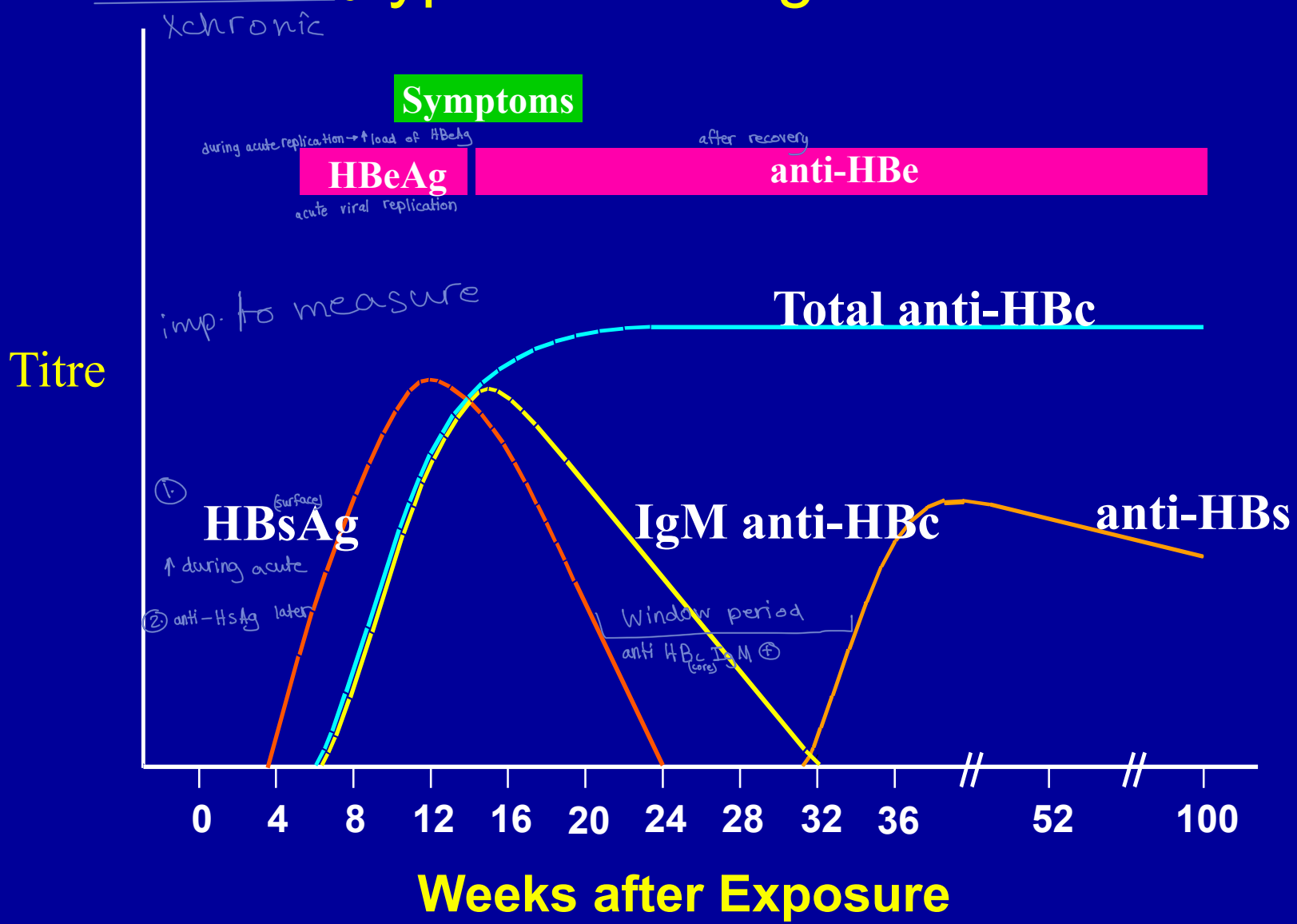
Adults showing symptoms is much more common than pediatric age group,
↳ can beat the virus & mostly recovery, they will show acute clinical illness: jaundice, abdominal pain, some SBS of acute HBV
↳ : most of them get chronic infx 90%.

- Acute case-fatality rate: 0.5%-1%
- Chronic infection: <5 yrs, 30%-90%<br>5 yrs, 2%-10%
- Premature mortality from chronic liver disease: 15%-25%

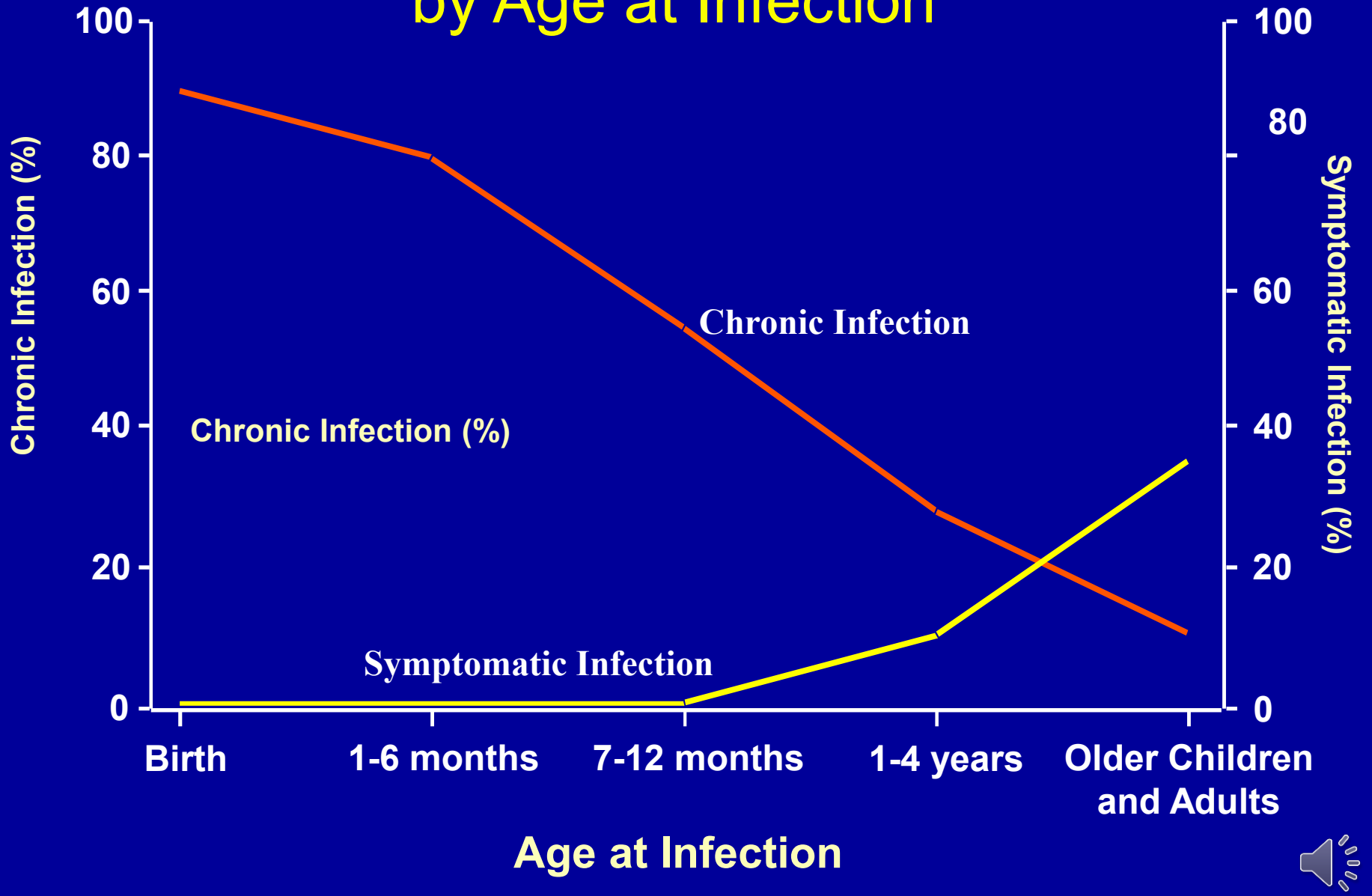
# Spectrum of Chronic Hepatitis B Diseases

1. Chronic Persistent Hepatitis - asymptomatic
2. Chronic Active Hepatitis - symptomatic exacerbations of hepatitis
3. Cirrhosis of Liver
4. Hepatocellular Carcinoma

# Acute Hepatitis B Virus Infection with Recovery: Typical Serologic Course



# Outcome of Hepatitis B Virus Infection by Age at Infection



# Concentration of Hepatitis B Virus in Various Body Fluids

| High | Moderate | Low/Not Detectable |
| --- | --- | --- |
| blood | semen | urine |
| serum | vaginal fluid | feces |
| wound exudates | saliva | sweat |
| | | tears |
| | | breastmilk |

# Hepatitis B Virus
# Modes of Transmission

- Sexual - sex workers and homosexuals are particular at risk.
- Parenteral - IVDA, Health Workers are at increased risk.
- Perinatal - Mothers who are HBeAg positive are much more likely to transmit to their offspring than those who are not. Perinatal transmission is the main means of transmission in high prevalence populations.

IV drug users

vertical pathway: mom → baby during delivery

we have some precautions, but if we don't use them the baby WILL most likely get the infx from his mom & will develop chronic infx

- the baby does NOT have enough immunity to even recognize the virus, so the virus lives within his hepatocytes friendly till at some point when he's 20-30 y.o when the body discovers that there's a foreign body in liver & starts acute phase of inflammation.
- during childhood → virus replicating happily in liver without any response from our body

# Diagnosis

NOT required

- A battery of serological tests are used for the diagnosis of acute and chronic hepatitis B infection.
- **HBsAg** - used as a general marker of infection.
- **HBsAb** - used to document recovery and/or immunity to HBV infection.
- **anti-HBc IgM** - marker of acute infection.
- **anti-HBcIgG** - past or chronic infection.
- **HBeAg** - indicates active replication of virus and therefore infectiveness.
- **Anti-Hbe** - virus no longer replicating. However, the patient can still be positive for HBsAg which is made by integrated HBV.
- **HBV-DNA** - indicates active replication of virus, more accurate than HBeAg especially in cases of escape mutants. Used mainly for monitoring response to therapy.

# Prevention

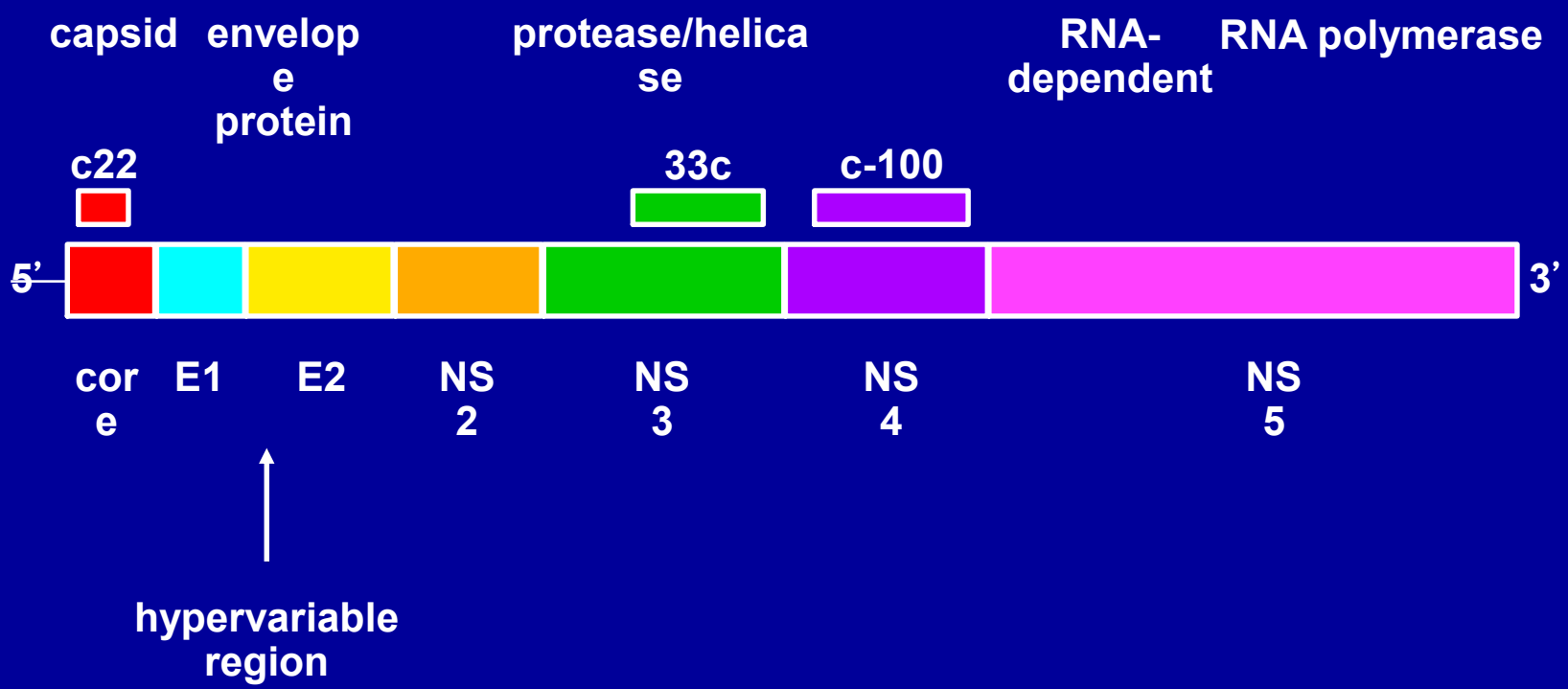
we're ALL vaccinated, national vaccination program in Jordan

- **Vaccination - highly effective recombinant vaccines are now available. Vaccine can be given to those who are at increased risk of HBV infection such as health care workers. It is also given routinely to neonates as universal vaccination in many countries.**
- **Hepatitis B Immunoglobulin - HBIG may be used to protect persons who are exposed to hepatitis B. It is particular efficacious within 48 hours of the incident. It may also be given to neonates who are at increased risk of contracting hepatitis B i.e. whose mothers are HBsAg and HBeAg positive.**
- **Other measures - screening of blood donors, blood and body fluid precautions.**

*pregnant → trx during 3rd trimester & soon after delivery the baby should get hepatitis B immunoglobulins and vaccinated at the same time asap.

# Hepatitis C Virus

RNA virus



# Hepatitis C - Clinical Features

| | |
|---|---|
| Incubation period: | Average 6-7 wks<br>Range 2-26 wks |
| Clinical illness (jaundice): | 30-40% (20-30%) |
| Chronic hepatitis: | 70% |
| Persistent infection: | 85-100% |
| Immunity: | No protective antibody response identified |

Xproven vaccine

# Chronic Hepatitis C Infection

- The spectrum of chronic hepatitis C infection is essentially the same as chronic hepatitis B infection.
- All the manifestations of chronic hepatitis B infection may be seen, albeit with a lower frequency i.e. chronic persistent hepatitis, chronic active hepatitis, cirrhosis, and hepatocellular carcinoma.

# Risk Factors Associated with Transmission of HCV

- Transfusion or transplant from infected donor
- Injecting drug use
- Hemodialysis (yrs on treatment)
- Accidental injuries with needles/sharps
- Sexual/household exposure to anti-HCV-positive contact
- Multiple sex partners
- Birth to HCV-infected mother

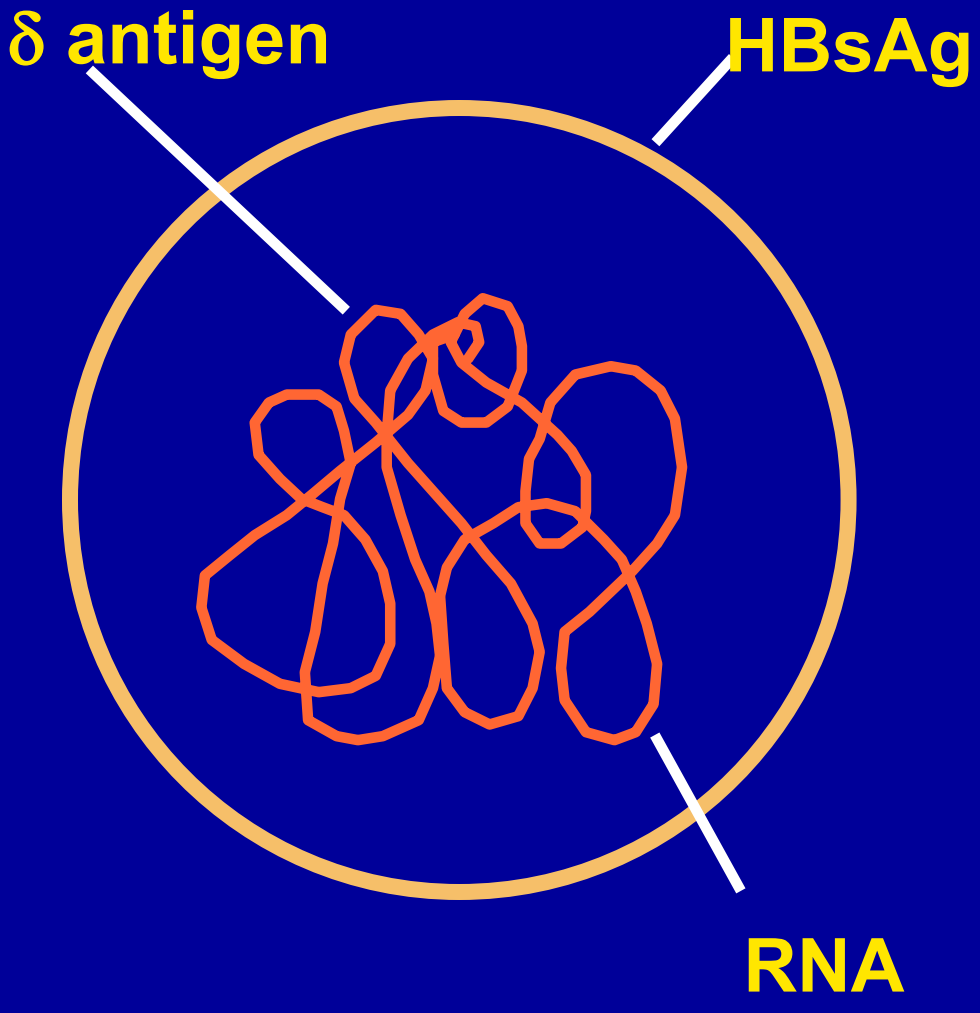
# Laboratory Diagnosis

- **HCV antibody** - generally used to diagnose hepatitis C infection. Not useful in the acute phase as it takes at least 4 weeks after infection before antibody appears.
- **HCV-RNA** - various techniques are available e.g. PCR and branched DNA. May be used to diagnose HCV infection in the acute phase. However, its main use is in monitoring the response to antiviral therapy.
- **HCV-antigen** - an EIA for HCV antigen is available. It is used in the same capacity as HCV-RNA tests but is much easier to carry out.

# Prevention of Hepatitis C

- Screening of blood, organ, tissue donors
- High-risk behavior modification
- Blood and body fluid precautions

# Hepatitis D (Delta) Virus




# Hepatitis D - Clinical Features

can NOT infect the patient alone

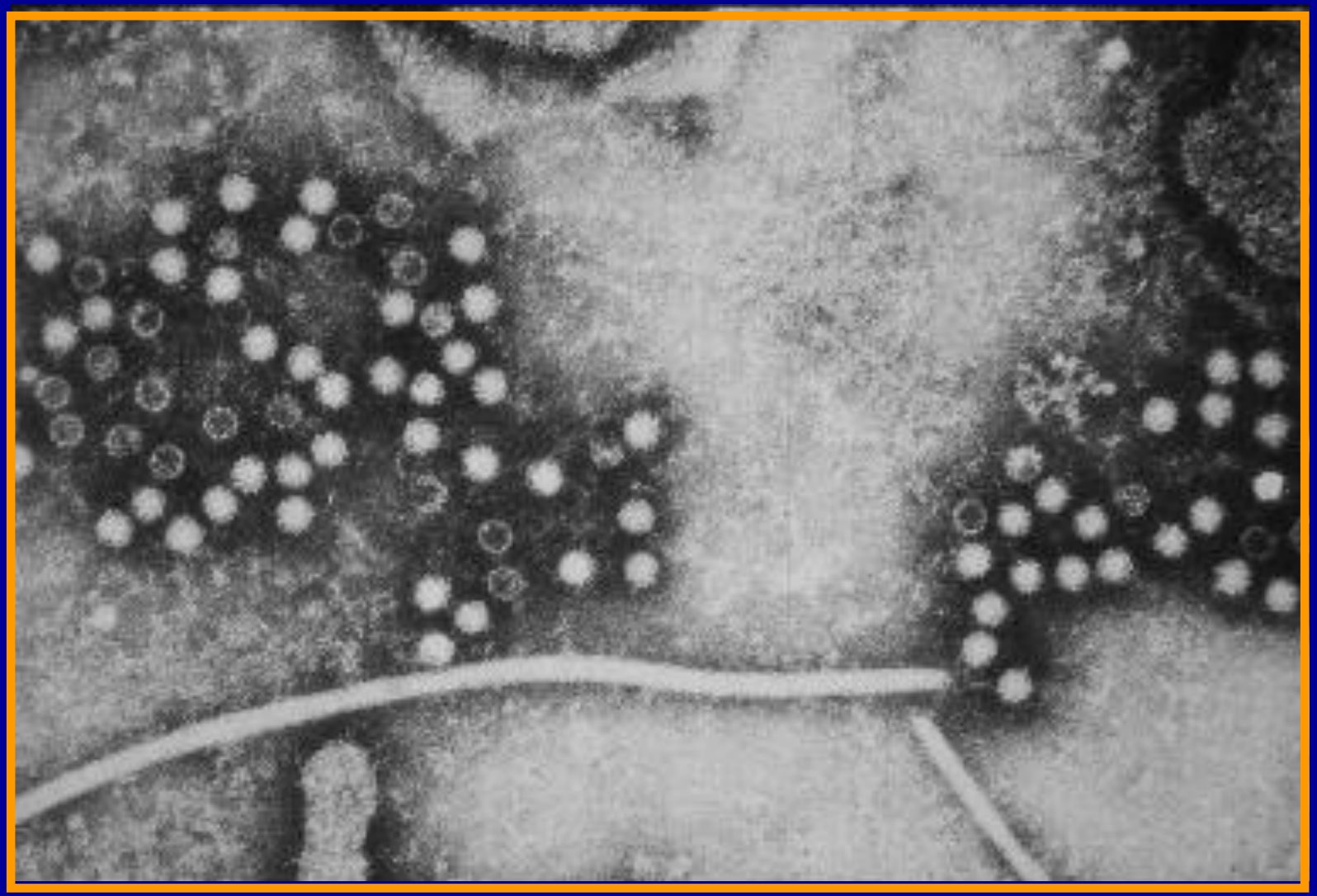
- Coinfection — both hepatitis B+D virus from the same source
  - severe acute disease.
  - low risk of chronic infection.
- Superinfection — already have chronic HBV and now he gets HDV from another source
  - usually develop chronic HDV infection. ↑ risk
  - high risk of severe chronic liver disease. > coinfection
  - may present as an acute hepatitis.

# Hepatitis D Virus Modes of Transmission

just like HBV, blood borne transmission

- Percutanous exposures
  - injecting drug use
- Permucosal exposures
  - sex contact

# Hepatitis E Virus



# Hepatitis E - Clinical Features

-just like HAV in modes of transmission: feco-oral route through infected food

- ↑mortality in pregnant females
-in most of the patients it causes self-limiting illness, acute infx ONLY; Xchronic hepatitis as B&C

- Incubation period: Average 40 days
  Range 15-60 days
- Case-fatality rate: Overall, 1%-3%
  Pregnant women, 15%-25%
- Illness severity: Increased with age
- Chronic sequelae: None identified