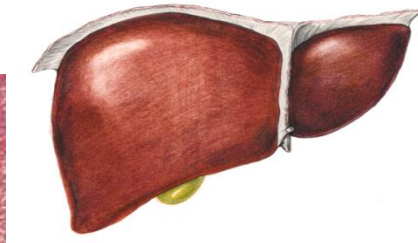
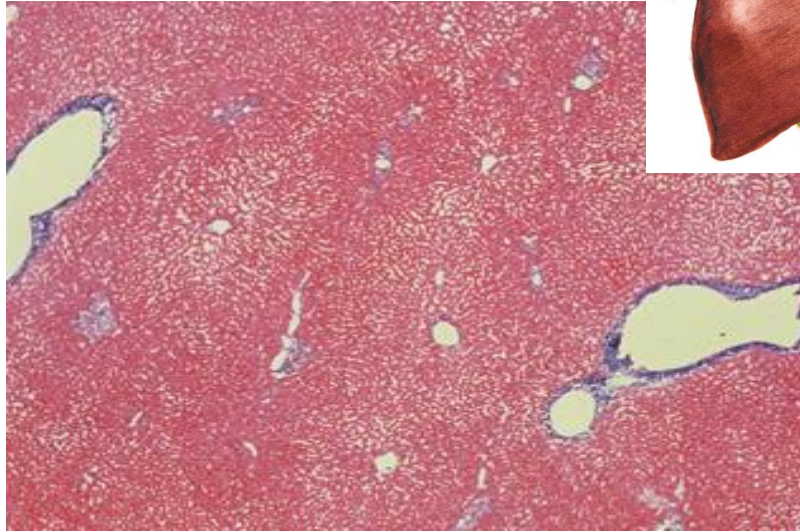
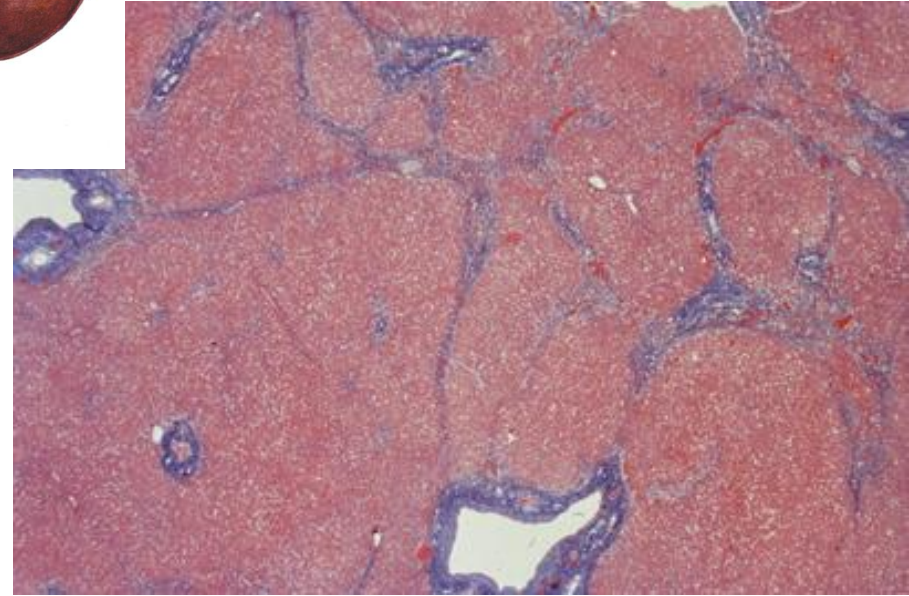


# Cirrhosis, Portal hypertension and Viral Hepatitis

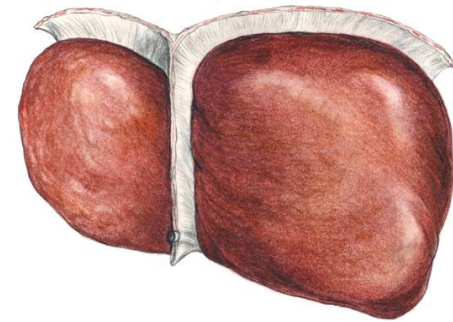
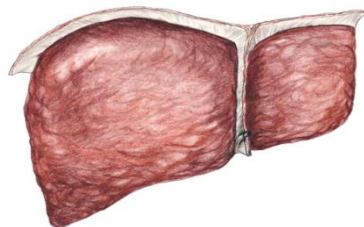
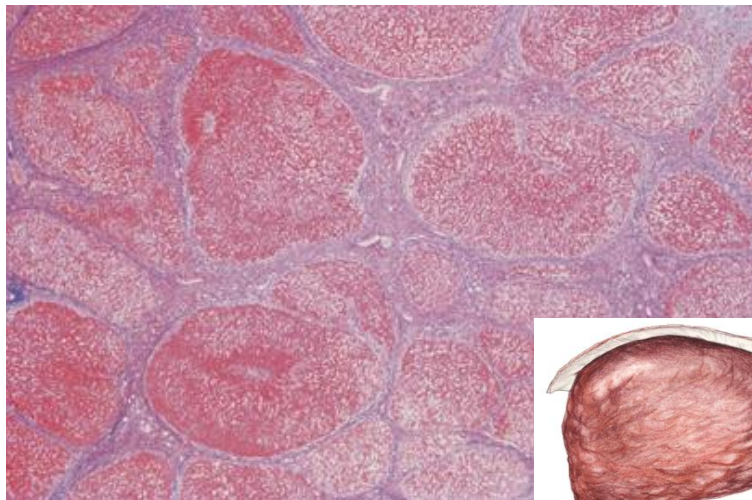
## Healthy Liver



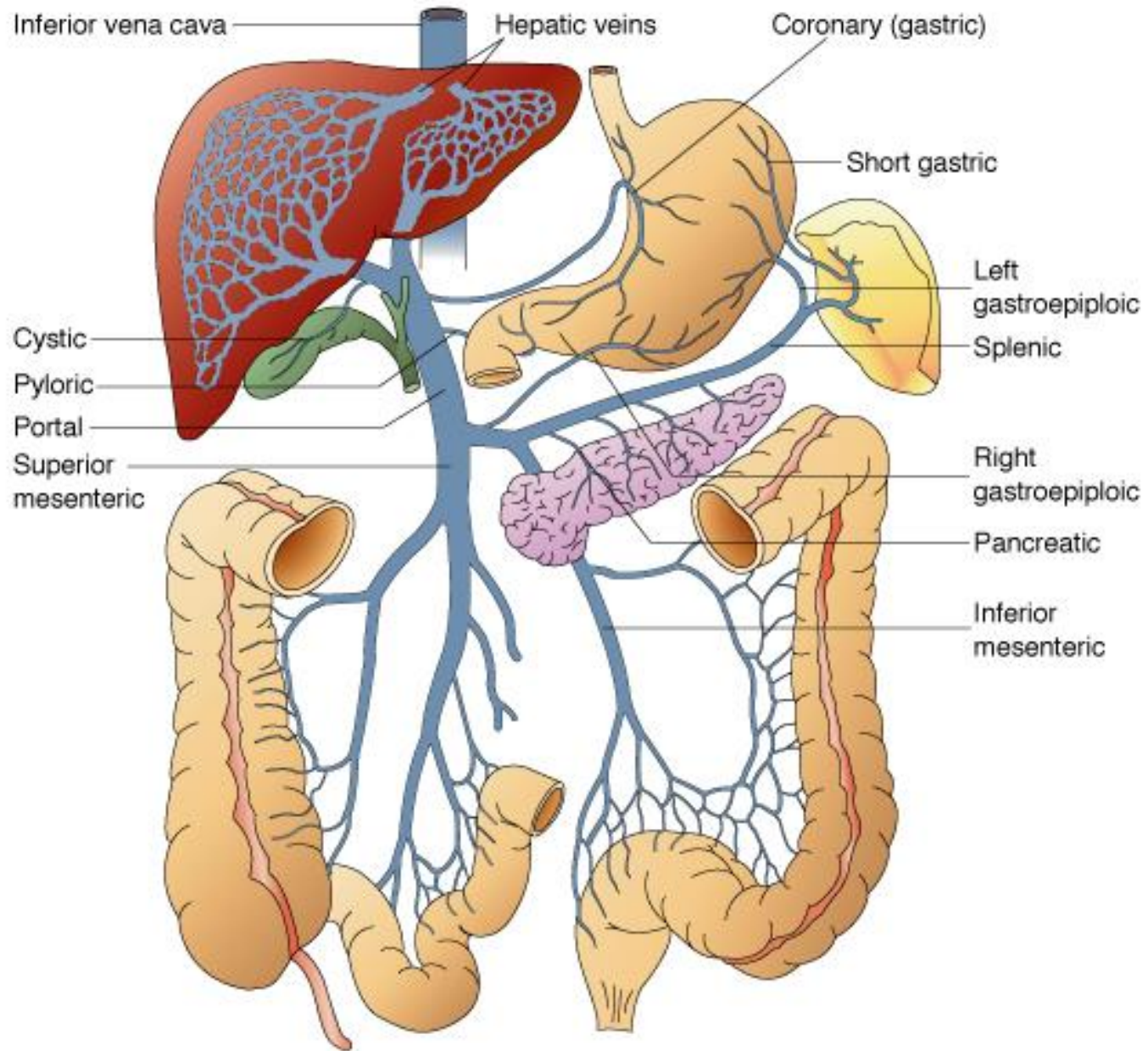
## Liver Fibrosis



## Cirrhosis



These four liver histology sections clearly show how the liver condition worsens through the stages of hepatitis C infection, and we can see how the liver tissue architecture has changed.



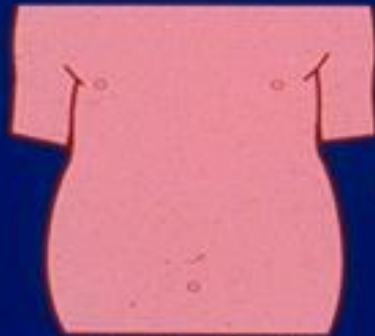
# Complications of Cirrhosis



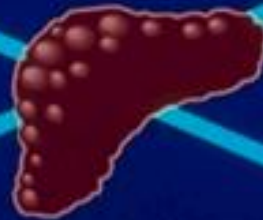
**Hepatic encephalopathy**



**Portal hypertension**



**Ascites/SBP**



**Hepatorenal syndrome**





## Jaundice

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



## 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.



This picture shows spider angioma indicating hyperdynamic circulation seen in advanced liver disease. Other indication of hyperdynamic status include wide pulse pressure, warm extremities and capillary pulsations in the nail beds.

## 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







## Gynecomastia

Breast development in men



## Dupuytren's Contractures

Joint contractures

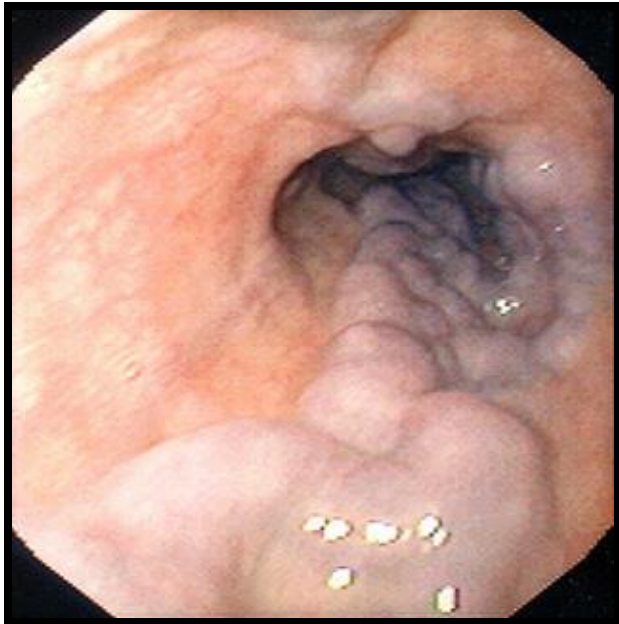


## **Caput Medusae**

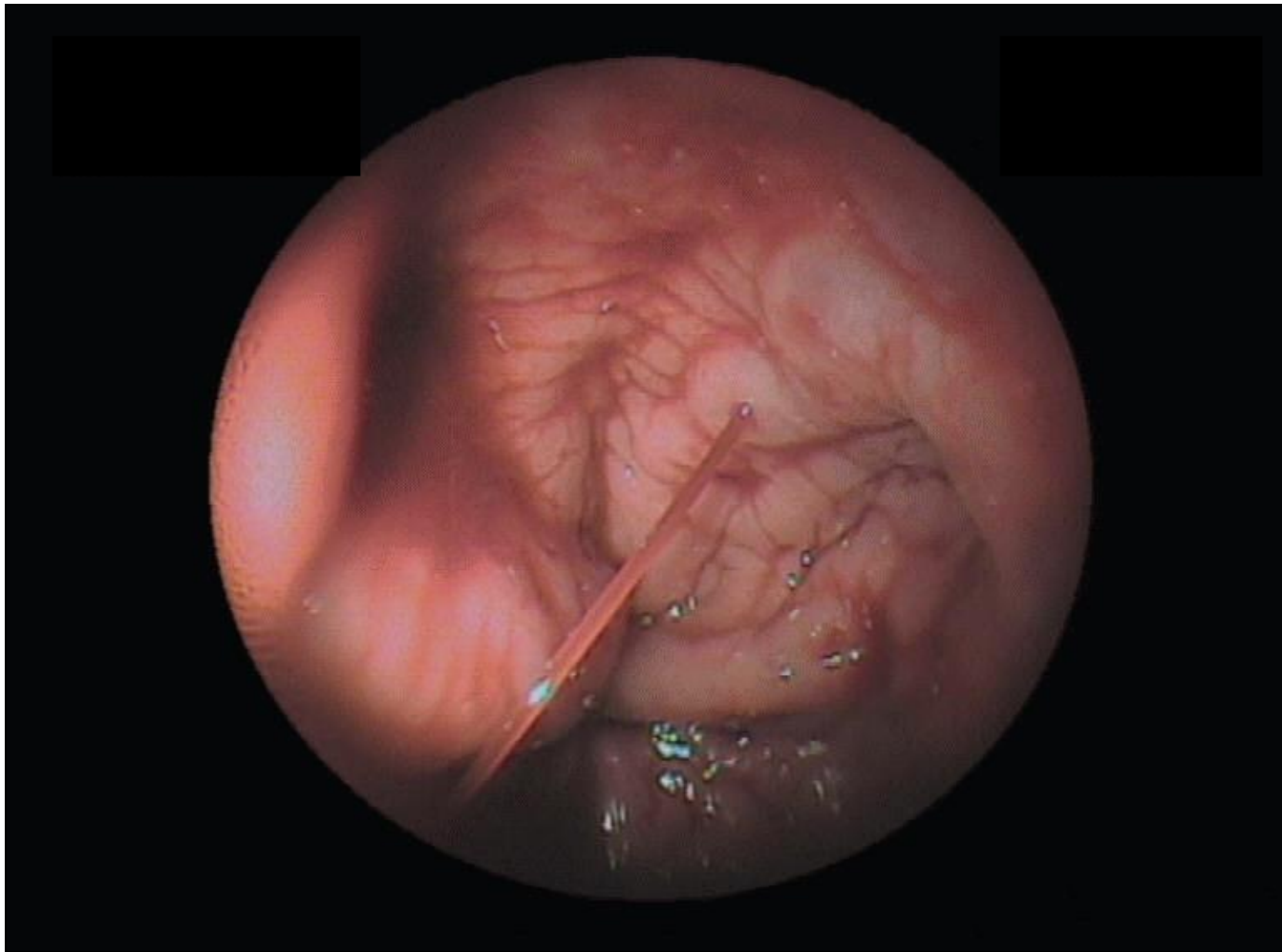
Distended and engorged umbilical veins which are seen radiating from the umbilicus across the abdomen to join systemic veins.

This is a picture of one of my patients, he is a 35 year old male patient, diagnosed as a case of budd chiari syndrome 5 years ago and despite proper anticoagulation his liver disease progressed to decompensated cirrhosis, he comes for follow up every couple of weeks for paracentesis,

this is a picture taken after his last paracentesis showing **Caput Medusae** which describes the appearance of distended and engorged umbilical veins which are seen radiating from the umbilicus across the abdomen to join systemic veins, indicating advanced portosystemic shunting seen in decompensated cirrhosis.



In general we see other areas with porto-systemic communications, like Gastro-Esophageal junction where Left Gastric vein and Middle Gastric vein communicate with systemic blood, lower rectum causing rectal varices resembling hemorrhoids, and retroperitoneal veins. The most significant clinically are **Esophageal Varices**, which are an almost unavoidable complication of portal hypertension. They are found in 40% of patients with compensated cirrhosis at the time of diagnosis and in 90% in all patients after long follow up



Rupture of esophageal varices is the main complication of portal hypertension and frequent cause of death. Despite the advancement in medical and endoscopic therapy, mortality is still high averaging between 25-35%.



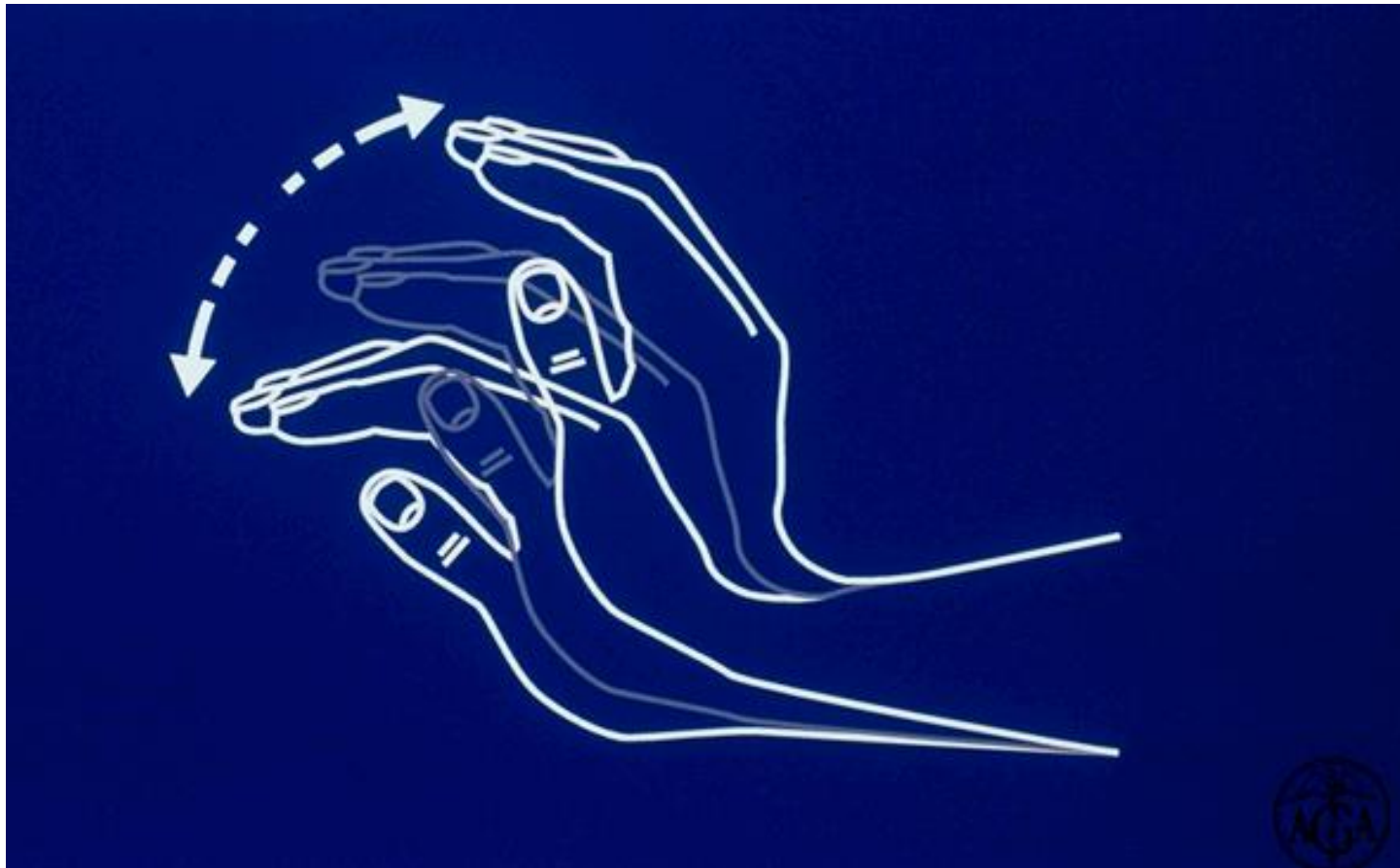


So how does ascites occur?

Ascites indicates advanced portal hypertension and a bad prognosis with median survival of 2 years after the onset.

In ascites patients there is sodium retention and impairment of free water excretion, leading to dilutional hyponatremia

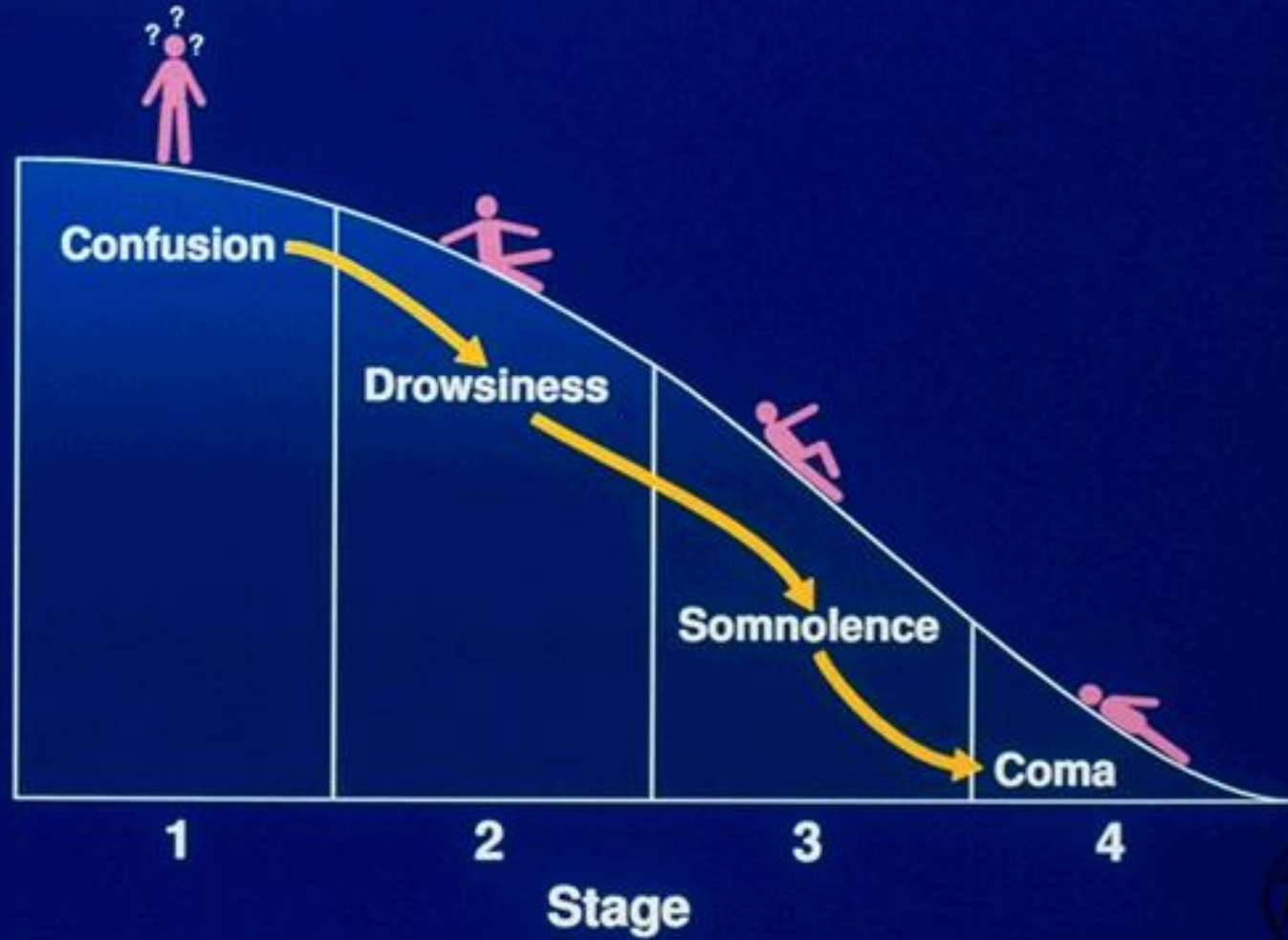




## Astraxia

Flapping tremors, quick arrhythmic movement in background tonic muscle contraction

# HEPATIC ENCEPHALOPATHY



# Hepatitis A-E Viruses

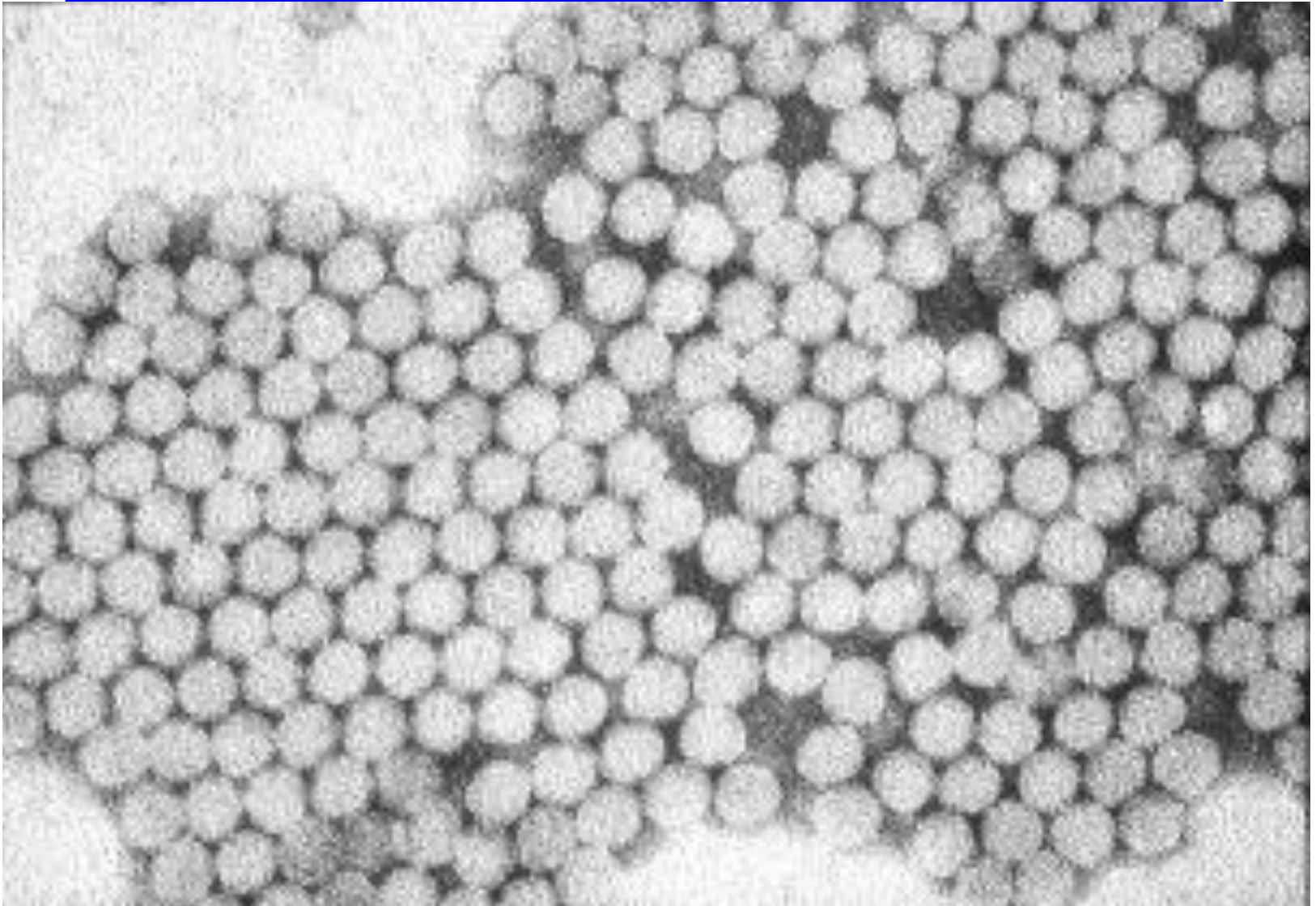
## An Overview

# Type of Hepatitis

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	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

# Hepatitis A Virus Transmission

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)

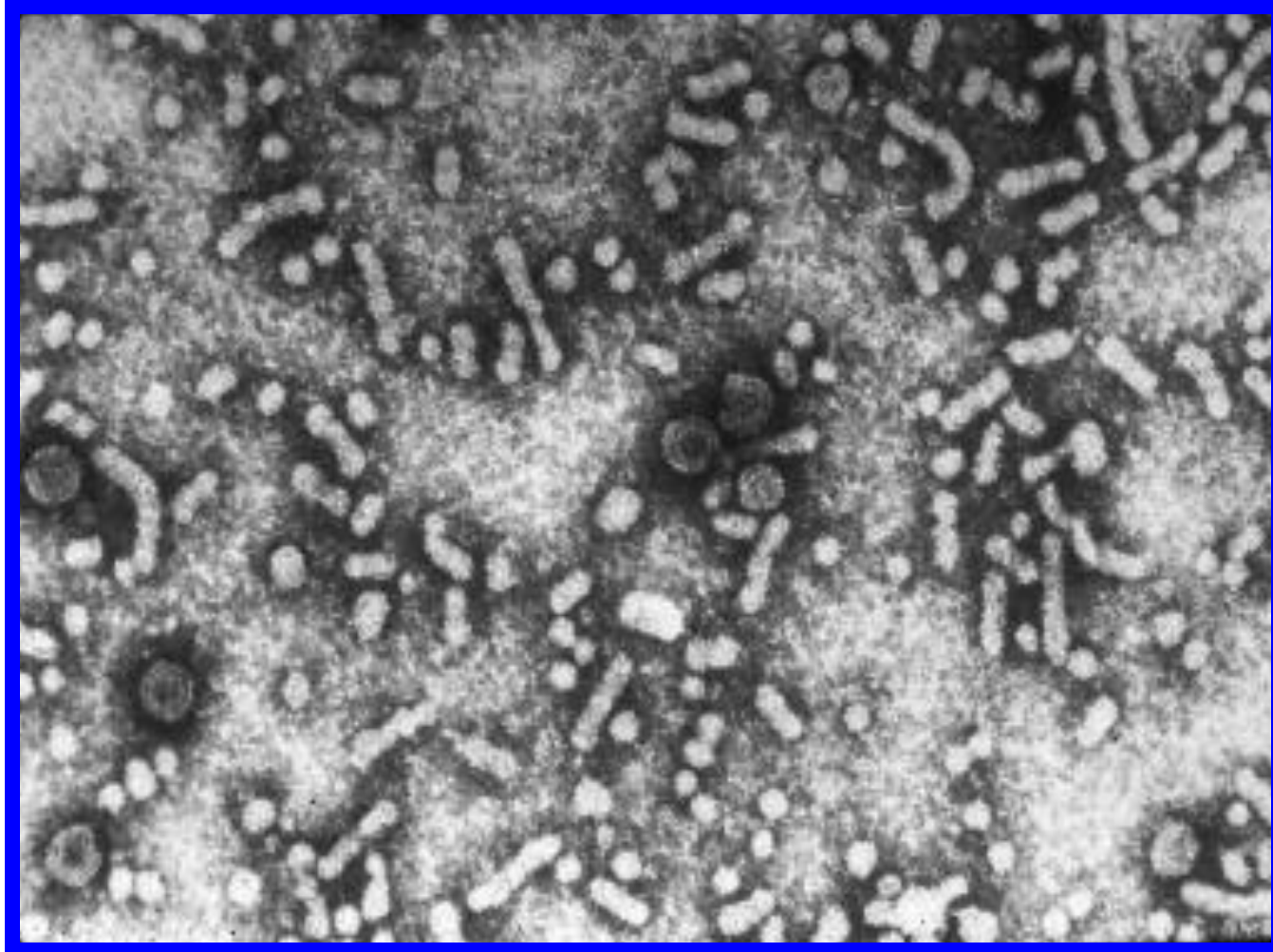
# Laboratory Diagnosis

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.



# Hepatitis B Virus



# Hepatitis B - Clinical Features

- **Incubation period:** Average 60-90 days  
Range 45-180 days
- **Clinical illness (jaundice):** <5 yrs, <10%  
5 yrs, 30%-50%
- **Acute case-fatality rate:** 0.5%-1%
- **Chronic infection:** <5 yrs, 30%-90%  
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

# Concentration of Hepatitis B Virus in Various Body Fluids

<b>High</b>	<b>Moderate</b>	<b>Low/Not Detectable</b>
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.

# Diagnosis

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.

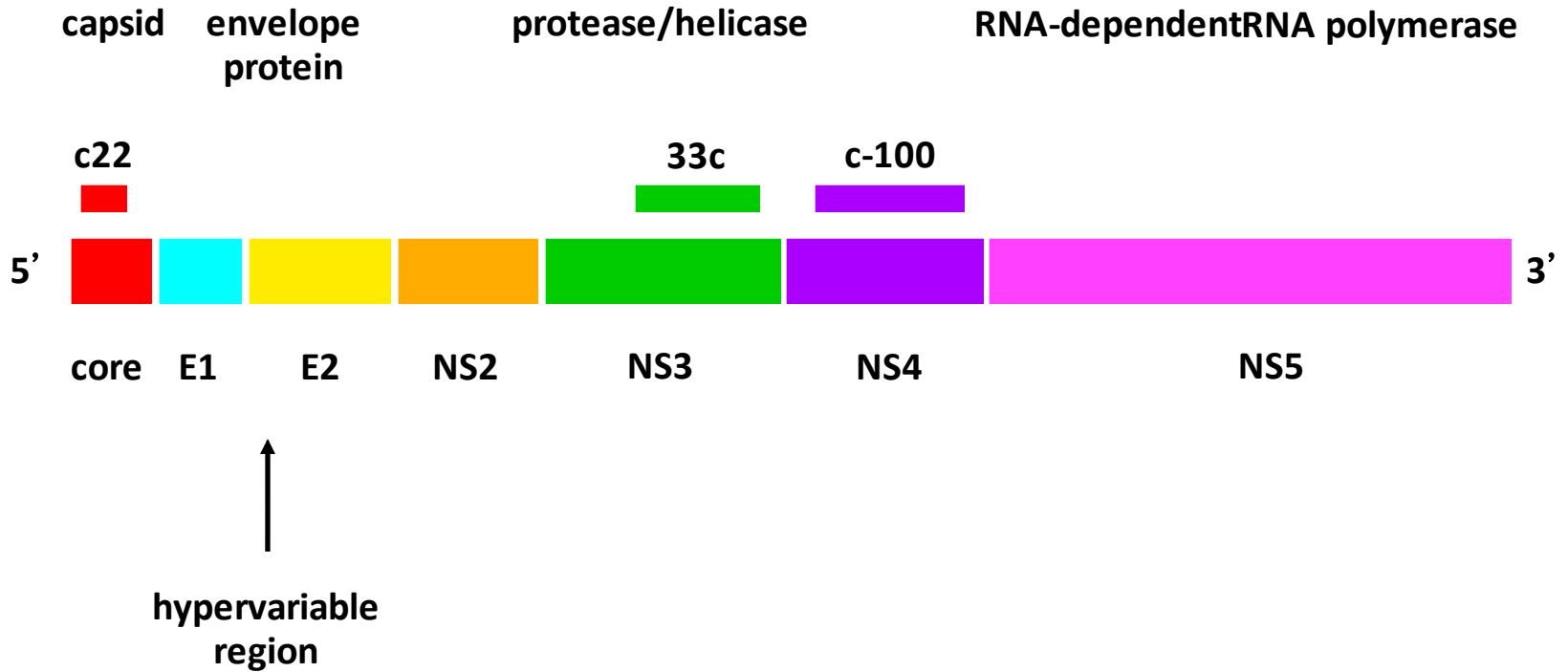
# Prevention

**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 particularly 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.

# Hepatitis C Virus





# Hepatitis C - Clinical Features

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

# 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.

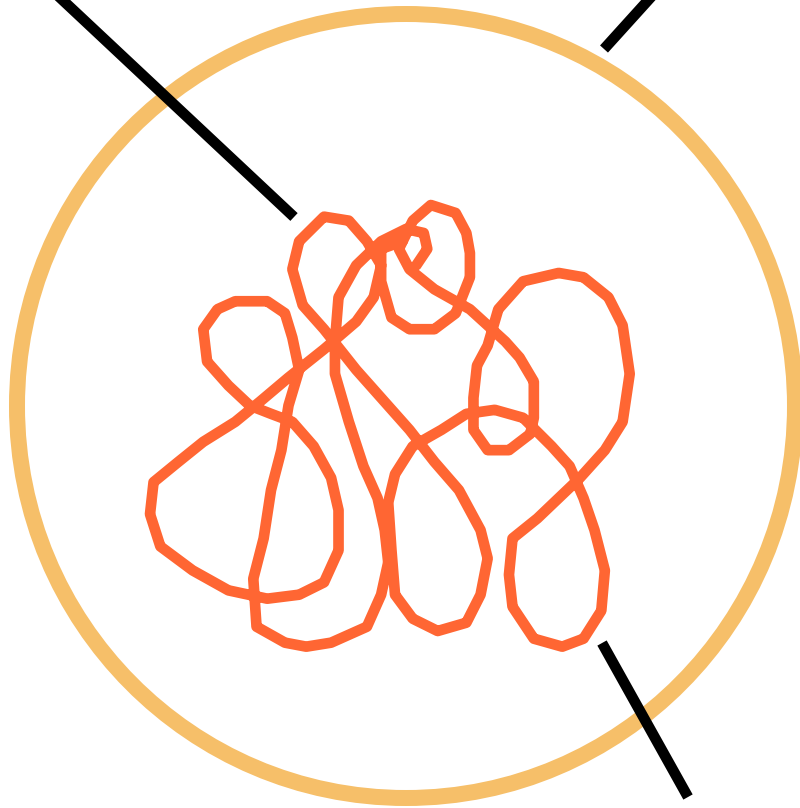
# Prevention of Hepatitis C

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

# Hepatitis D (Delta) Virus

$\delta$  antigen

HBsAg



RNA



# Hepatitis D - Clinical Features

- **Coinfection**

  - severe acute disease.

  - low risk of chronic infection.

- **Superinfection**

  - usually develop chronic HDV infection.

  - high risk of severe chronic liver disease.

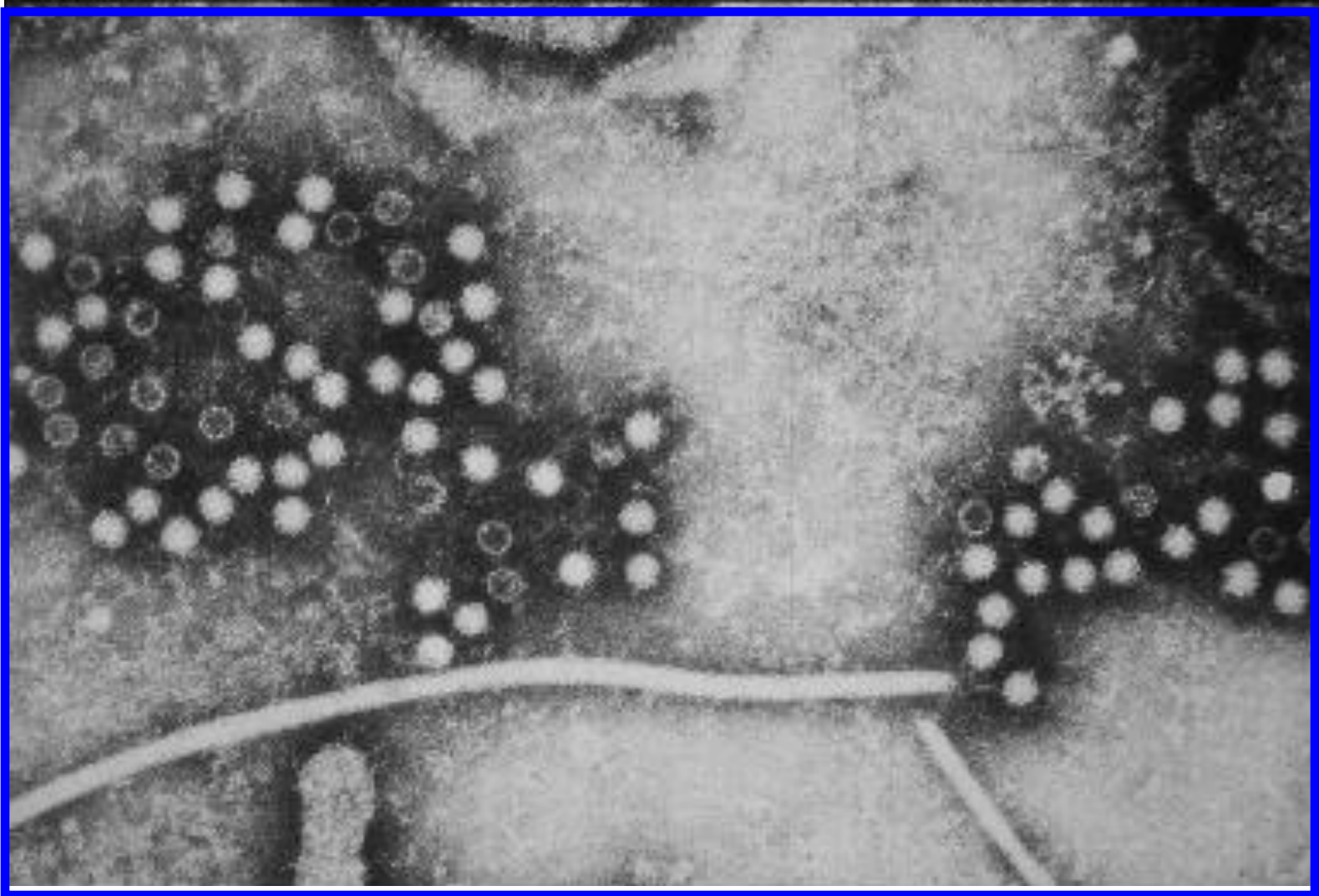
  - may present as an acute hepatitis.

# Hepatitis D Virus Modes of Transmission

- Percutaneous exposures
  - injecting drug use
- Per mucosal exposures
  - sex contact



# Hepatitis E Virus



# Hepatitis E - Clinical Features

- 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