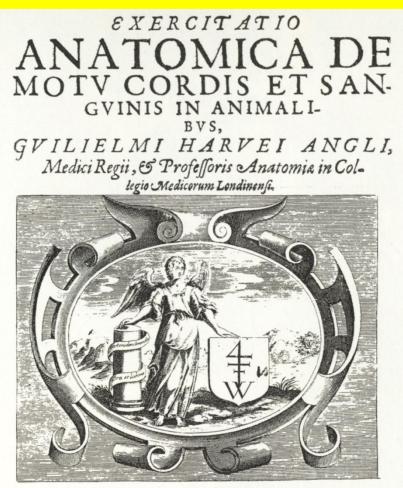
Lecture 5



FRANCOFVRTI, Sumptibus GVILIELMI FITZERI.

DISTURBANCES OF CIRCULATION

ACTIVE HYPEREMIA (ARTERIAL)

EXCESS OF BLOOD IN ARTERIES: <u>PHYSIOLOGICAL WHEN MOVING</u>, <u>RUNNING</u>





CONFIRMED IN PARALYSIS AND IRRITATION OF VESSEL-CONTROLLING NERVES BECAUSE OF INCREASE IN CONCENTRATION OF CO2 IN THE TISSUES

AND IN SOME STAGES OF INFLAMMATION

PASSIVE HYPEREMIA (VENOUS)

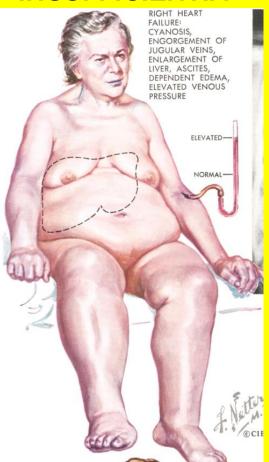
- **EXCESS OF BLOOD IN THE VEINS**
 - ALWAYS PATHOLOGICAL
- **MACROSCOPY OF ORGANS: HEAVY, DARK RED,**

GENERALIZED VENOUS HYPEREMIA

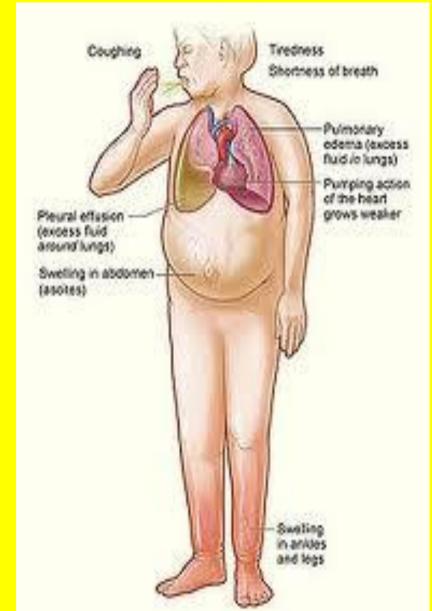
DEVELOPS WHEN THERE IS A RESTRICTION OF BLOOD FLOW IN THE CENTRAL POINTS OF CIRCULATION – HEART AND LUNGS

IT IS AN EQUIVALENT OF THE CLINICAL/MORPHOLOGICAL
CONDITION: CIRCULATION INSUFFICIENCY - INSUFFICIENTIA
CIRCULATORIA

- •GENERAL SYMPTOMS
- CYANOSIS
- DYSPNEA difficult breathing
- TRANSUDATE, EDEMA SWELLING



CIRCULATION INSUFFICIENCY



LOCAL VENOUS HYPEREMIA

E.G. THE RESTRICTION OF VENOUS FLOW THROUGH LIVER IN THE CASE OF CIRRHOSIS.

EARLY PERIOD –

CONGESTION IN THE VEINS,

EDEMA, DEGENERATION,

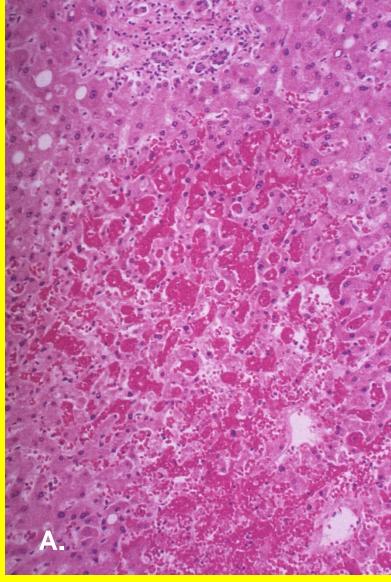
HYPOXIA

LATE PERIOD –

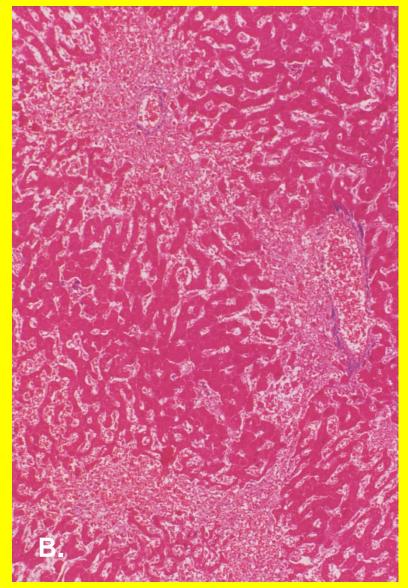
STIMULATION OF FIBROBLASTS -PRODUCTION OF COLLAGEN,

CYANOTIC INDURATION, ATROPHY OF PARENCHYMA, HEMOSIDERIN, NECROSIS, FUNCTIONAL DISORDERS, COLLATERAL CIRCULATION

LIVER VENOSTASIS

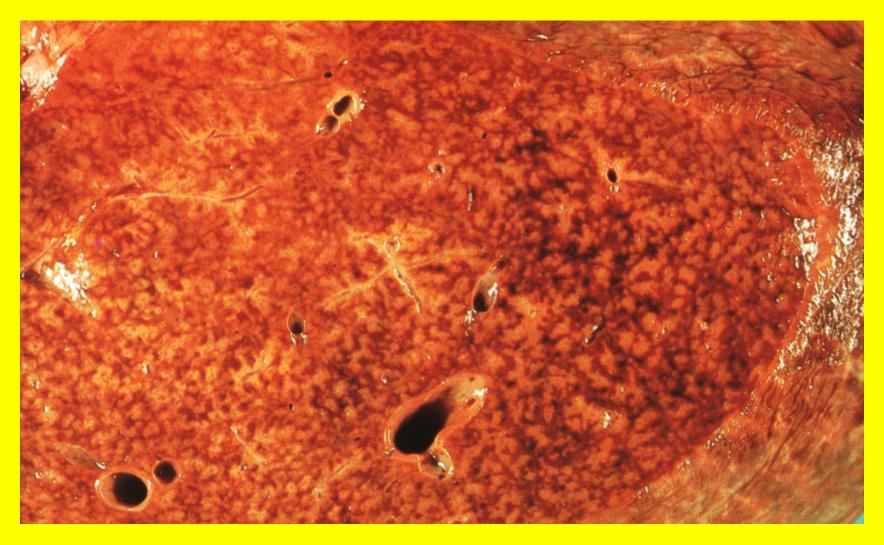


A. ERYTHROCYTES IN LIVER SINUSES



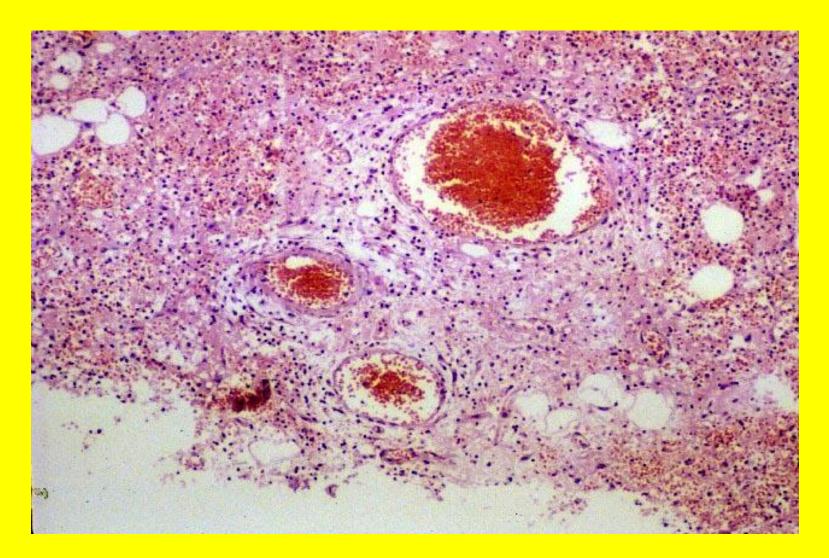
B. BLOOD IN THE AREA OF THE CENTRAL VEINS - FIBROSIS

VENOSTASIS (PASSIVE HYPEREMIA) IN LIVER

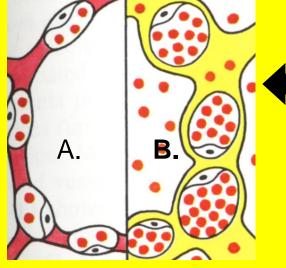




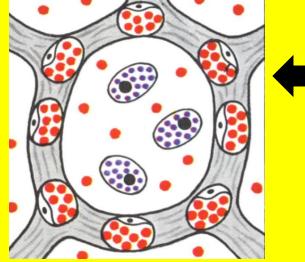
VENOSTASIS (PASSIVE HYPEREMIA)



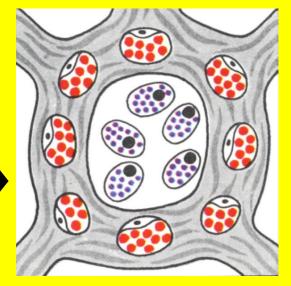
VENOUS HYPEREMIA IN LUNGS



A. NORMAL LUNG B. SEVERE BLOOD CONGESTION

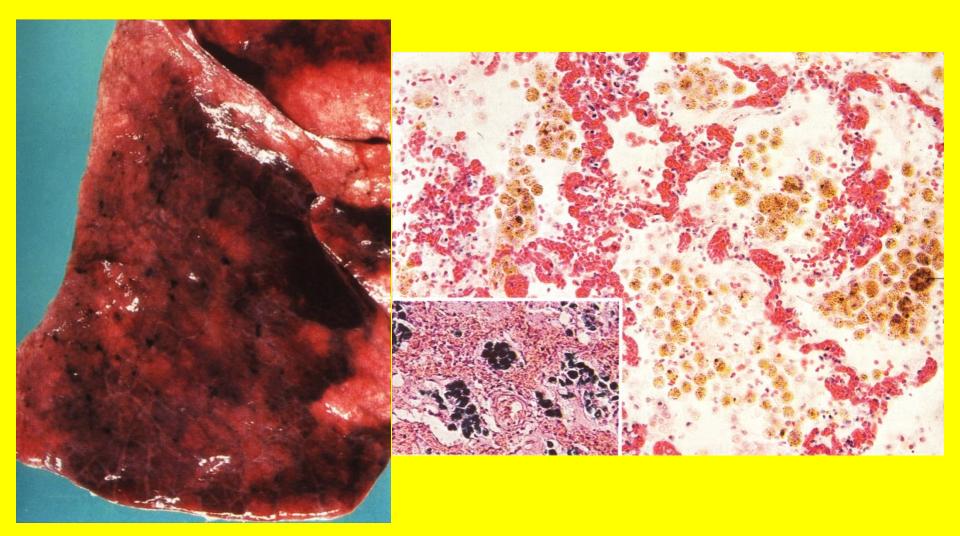


EARLY VENOSTASIS -MACROPHAGES

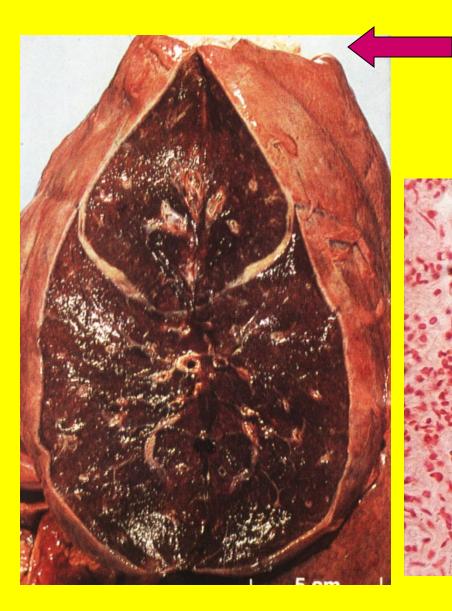


CHRONIC VENOSTASIS – FIBROSIS MACROPHAGES

VENOUS HYPEREMIA (PASSIVE) IN LUNGS EARLY STAGE OF VENOSTASIS IN THE LUNG

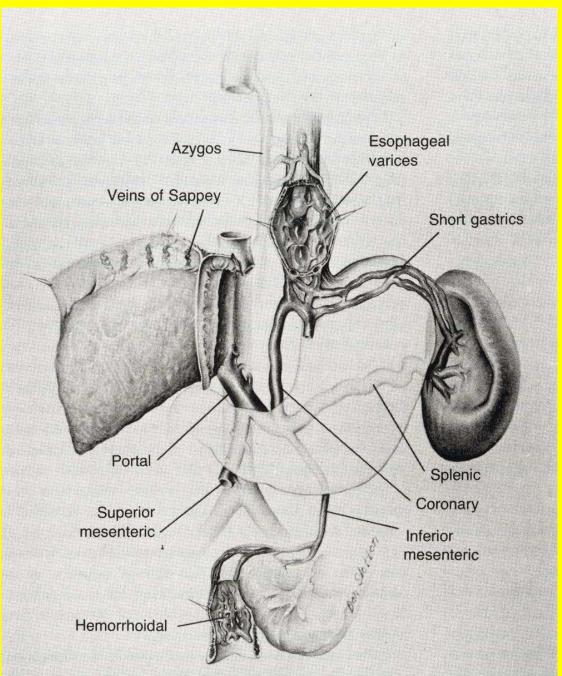


CHRONIC VENOSTASIS (HYPEREMIA) IN LUNGS

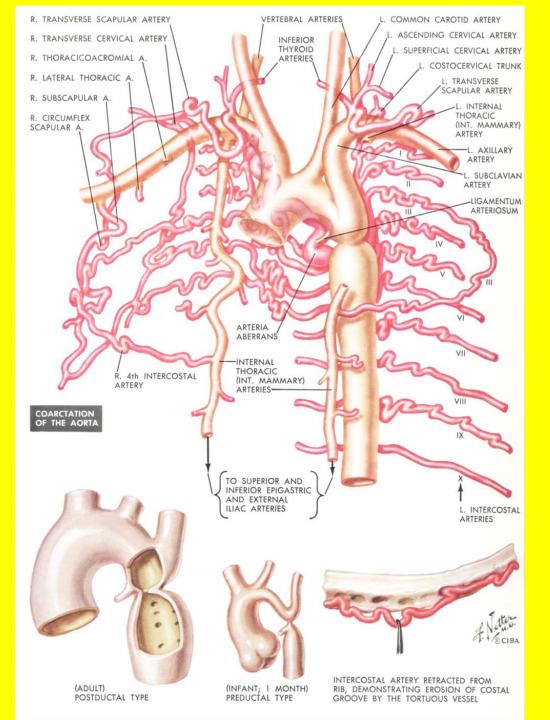


MACROSCOPIC AND MICROSCOPIC PICTURES OF VENOSTASIS IN THE LUNG

COLLATERAL CIRCULATION IN LIVER CIRRHOSIS



 VARICES (ESOPHAGUS)
VARICES (HEMORRHOIDAL)
MEDUSA HEAD (CAPUT MEDUSAE) IN CHILDREN COLLATERAL CIRCULATION DUE TO COARCTATION OF AORTA

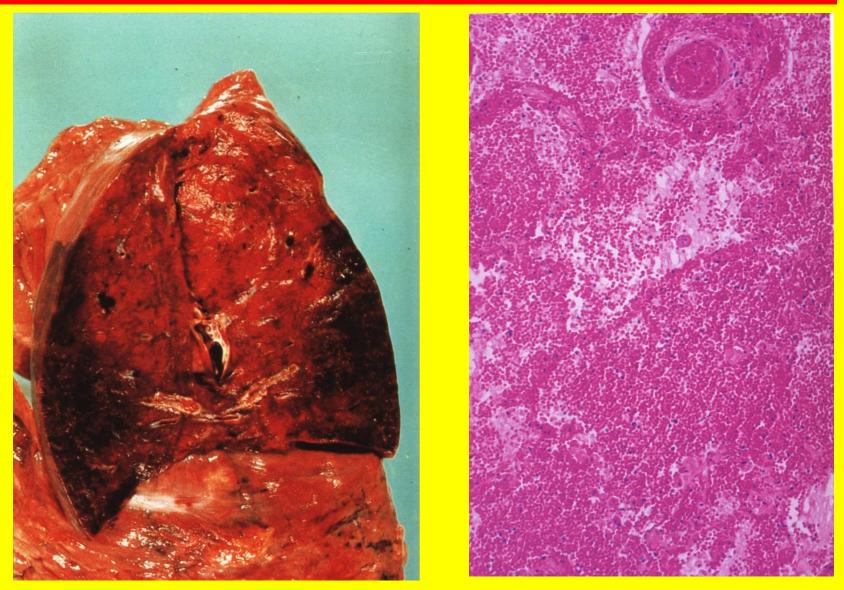


ANEMIC/PALE INFARCT - INFARCTUS PALLIDUS



INFARCTUS PALLIDUS – PALE INFARCT (LIVER) INFARCTUS PALLIDUS – PALE INFARCT (KIDNEY)

HEMORRHAGIC INFARCT [RED, RUBER, HAEMORRHAGICUS]



INFARCTUS HAEMORRHAGICUS (LUNG)

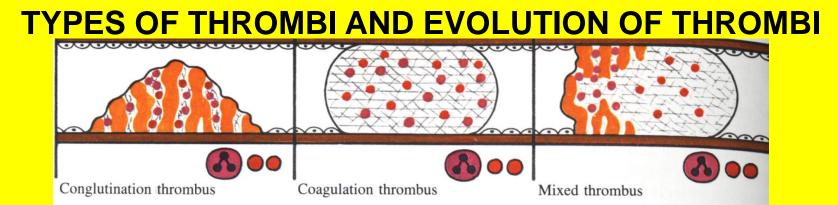
AFTER DEATH: IN HEART AND IN VESSELS = CRUOR, COAGULUM

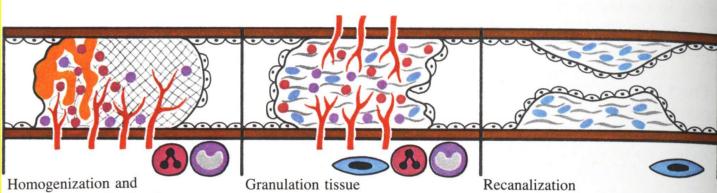
DURING LIFE: WHEN BLOOD SPILLS BEYOND A VESSEL = CRUOR, COAGULUM

DURING LIFE: WHEN BLOOD CLOTS INSIDE VESSELS = THROMBUS, CLOT

CRUOR - COAGULUM

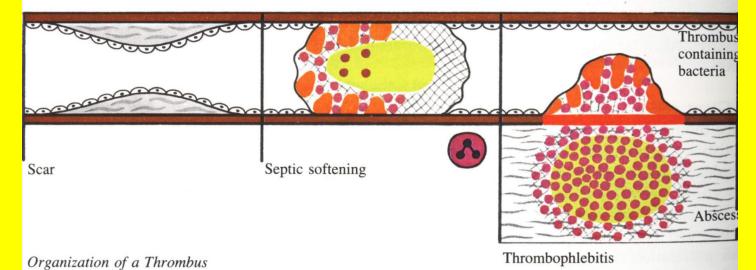






4-6 weeks

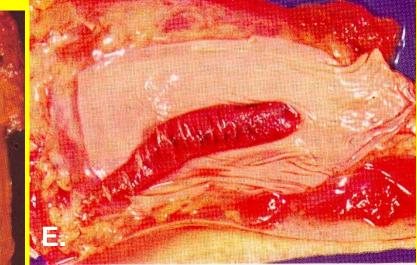
beginning organization by connective tissue, 2–5 days Granulation tissue (organization) 10 days-4 weeks

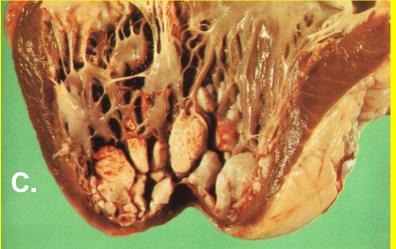




B.

FORMS OF THROMBI





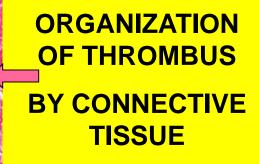
A. PARIETAL

5 cm

B. OBTURATING FEMORAL VEIN

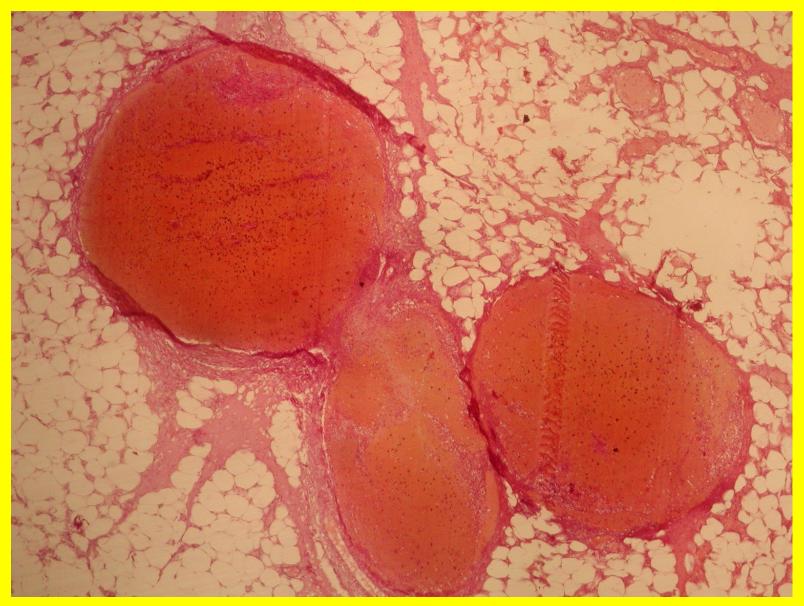
C. SPHERICAL THROMBUS IN HEART D. ELONGATED THROMBUS E. LAMINATED THROMBUS

RECENT THROMBUS

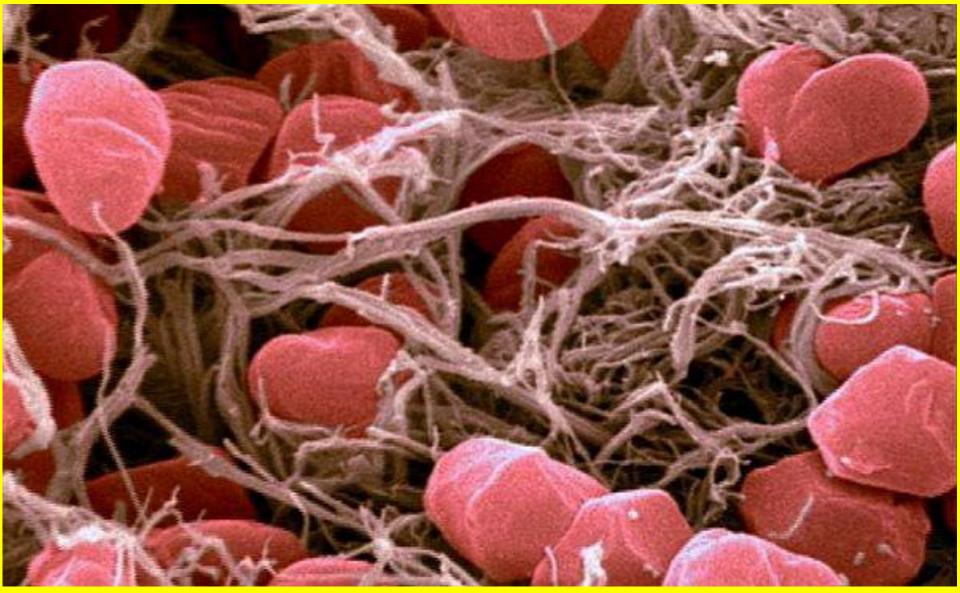


RECANALIZATION OF THROMBUS

THROMBI IN VESSELS



THROMBOSIS



THROMBOTIC EMBOLISM





A. THROMBOTIC EMBOLISM IN PULMONARY ARTERY

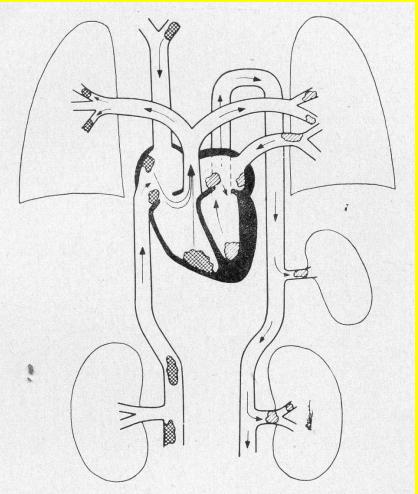
B. THROMBOTIC EMBOLISM IN PULMONARY ARTERY-RIDING, SADDLE, STRADDLING, PANTALOON

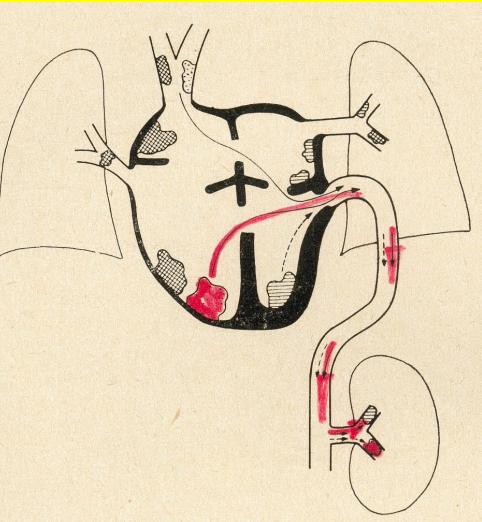
C. ORIGIN OF EMBOLISM – THROMBOSIS OF FEMORAL VEIN

Blood clot travels from leg to lung

EMBOLUS – EMBOLISM

ANY SUBSTANCE THAT HAS OBTURATED A VESSEL = EMBOLUS DEFINITION - OBTURATION OF THE LIGHT OF A VESSEL BY A SUBSTANCE THAT WAS PRESENT IN THE BLOOD STREAM





Ryc. III-10. Schemat wędrówki zakrzepów.

DIRECT EMBOLISM (ORTHOGRADE, SIMPLE) CROSSED EMBOLISM (PARADOXIC

FAT EMBOLISM



EMBOLIAE ADIPOSAE PULMONIS

REMEMBER : FAT EMBOLISM AND OTHER EMBOLISMS CAUSED BY FLUIDS OR MATERIAL WHICH ARE SMALL IN SIZE SHOULD BE SEARCHED IN PARENCHYMA OF THE LOWER LOBI.

FAT EMBOLISM IS A PART OF DECOMPRESSION DISEASE

FAT MAY ORIGINATE FROM "RESERVE" FAT TISSUE OR FAT PRESENT IN PLASMA

FAT EMBOLISM

 Usually presenting with a delay of 12-72 h, the classical triad consists of <u>respiratory</u> <u>distress, cerebral signs and petechiae</u>.
Fat embolism syndrome can go unnoticed clinically or may present as an acute fatal event within hours of the inciting injury.

FAT EMBOLISM SYNDROME

- Clinical symptoms
- A. Respiratory symptoms:

These are usually the first presenting features. Hypoxemia, tachypnoea, and dyspnoea are the initial findings. In some cases, the patients may progress to respiratory failure, requiring mechanical ventilation. In other cases, if no ongoing embolism or

infection occurs, the lung usually recovers by the third day.

FAT EMBOLISM SYNDROME

• B. Neurological symptoms:

The symptoms may appear within 10-120 h and are highly varying. Ranging from confusion, to seizures, and they

may include irritability, anxiety, agitation, confusion, delirium, and coma, which are described as progressive changes and as single manifestation in individual cases. These symptoms are usually non-lateralizing, transient and fully reversible

Localizing signs, such as aphasia, apraxia

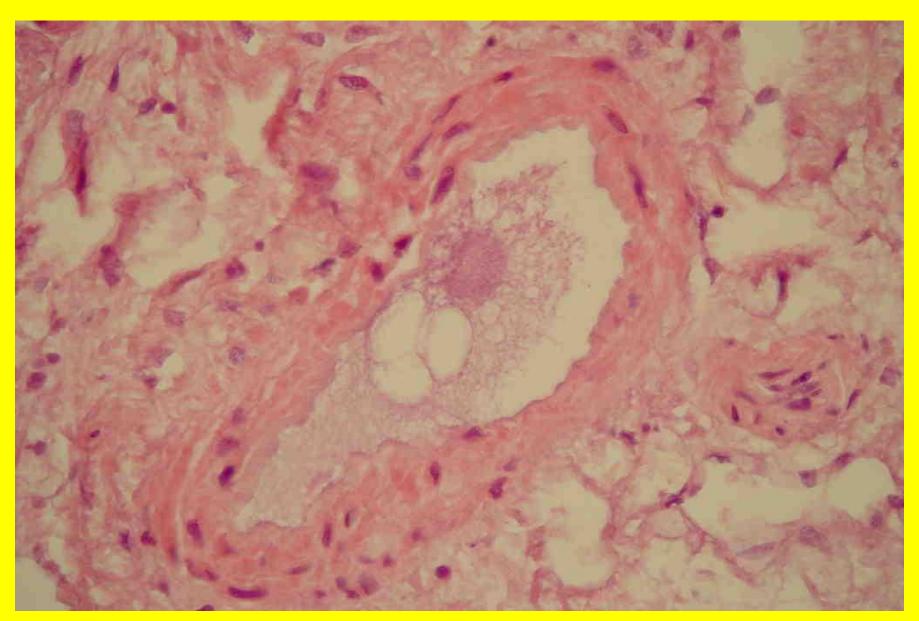
APHASIA is an inability to comprehend and formulate language because of damage to specific brain regions. This damage is typically caused by a cerebral vascular accident (stroke)

APRAXIA IS A MOTOR DISORDER CAUSED BY DAMAGE TO THE BRAIN IN WHICH THE INDIVIDUAL HAS DIFFICULTY WITH THE MOTOR PLANNING TO PERFORM TASKS OR MOVEMENTS WHEN ASKED, PROVIDED THAT THE REQUEST OR COMMAND IS UNDERSTOOD AND HE/SHE IS WILLING TO PERFORM THE TASK.

FAT EMBOLISM SYNDROME

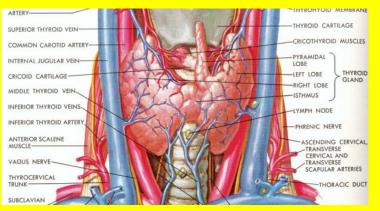
- C. Cutaneous manifestations:
- Petechial rash may be the last component to develop in FES. Appearing within 36 h, this is believed to be pathognomic feature of FES.
- It is usually self limiting, and disappears within a week. It can be easily missed in dark skinned persons, and is to be actively sought on the upper portions of the chest and axillae.
- **Other manifestations: Fever, tachycardia are non-specific but seen in almost all cases of FES**

FAT EMBOLISM



AIR EMBOLISM

300 ML OF OXYGEN MAY KILL WHEN ADMINISTERED INTO THE CIRCULATORY SYSTEM



SURGERY IN THE HEAD AND NECK AREA



DECOMPRESSION SICKNESS (DIVERS)



DELIVERY - ATONY OF UTERUS

Decompression Sickness

- decompression sickness (DCS), a complex resulting from changed barometric pressure, includes high-altitude-related and aerospace-related events,
- decompression associated also with the sudden decrease in pressures during underwater ascent, usually occurring during free or assisted dives.

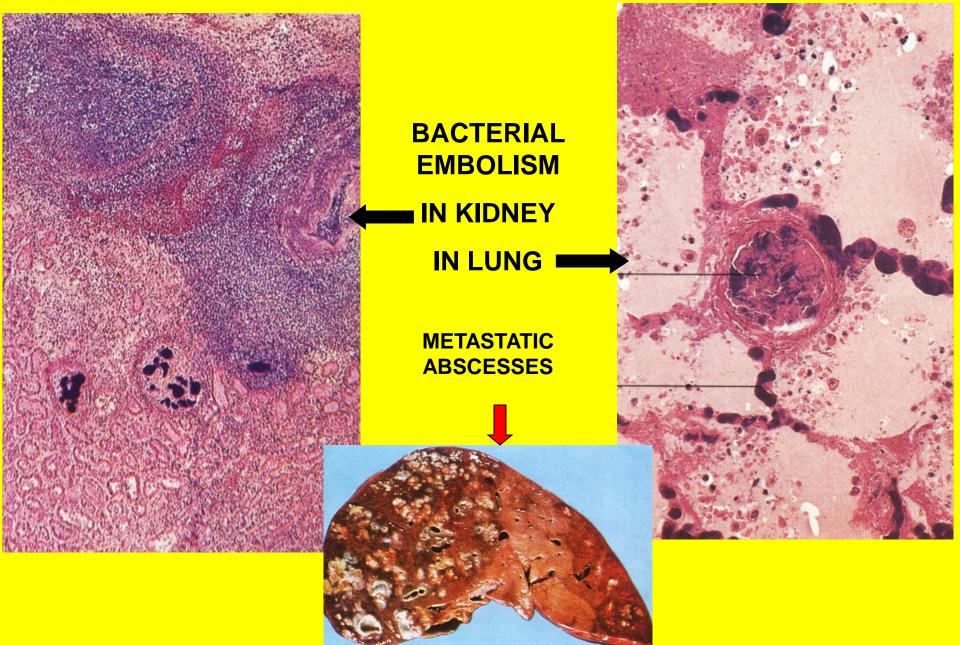
Decompression sickness

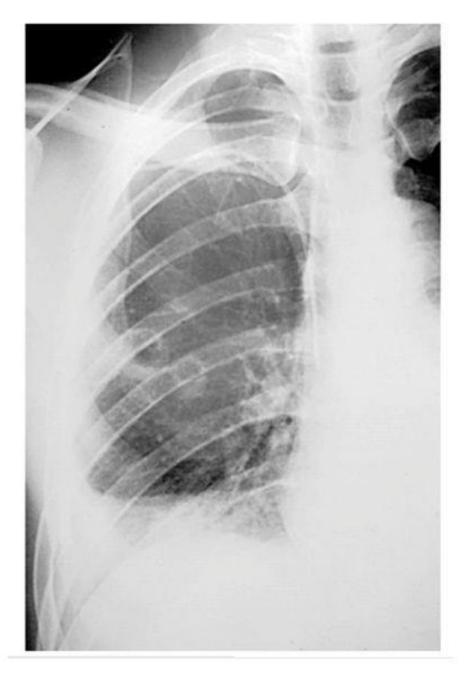
 also called generalized barotrauma or the bends, refers to injuries caused by a rapid decrease in the pressure that surrounds you, of either air or water. It occurs most commonly in scuba or deepsea divers, although it also can occur during high-altitude or unpressurized air travel. However, decompression sickness is rare in pressurized aircraft, such as those used for commercial flights.

Symptoms of decompression sickness

- include:
- joint pain, dizziness (vertigo), headache,
- difficulty thinking clearly
- extreme fatigue, ear tingling, numbness
- weakness in arms or legs
- a skin rash
- www.health.harvard.edu/diseases

MICROBIAL EMBOLISM

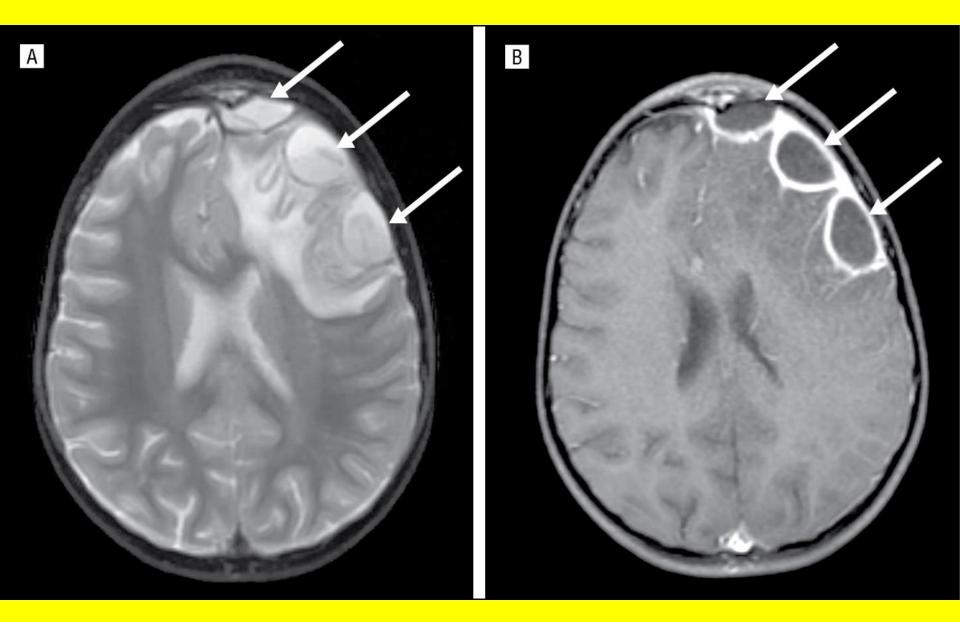




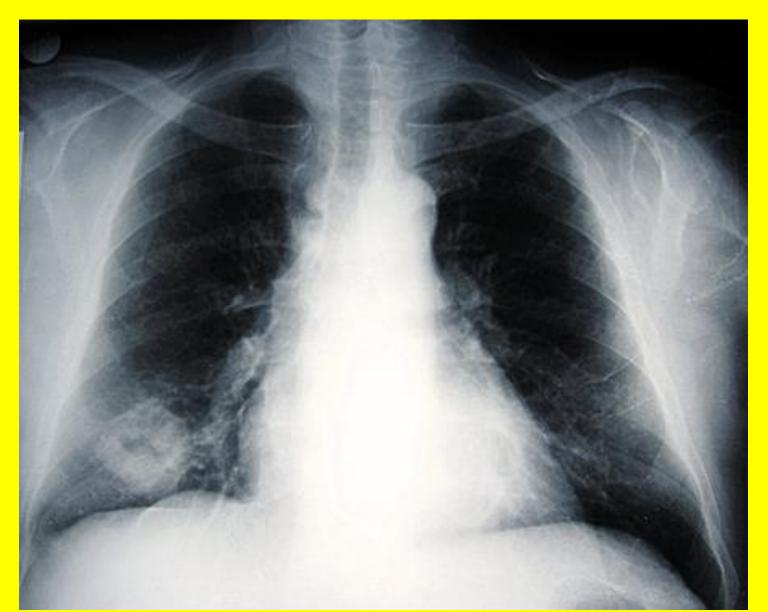


Metastatic Abscess

METASTATIC ABSCESSES - EPIDURAL



LUNG ABSCESS



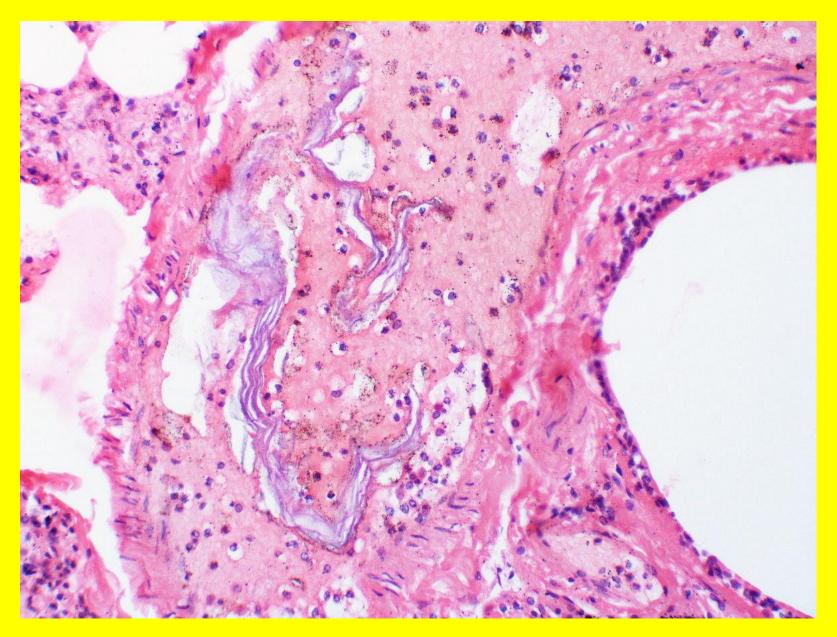
Viridans streptococci Intracranial Abscess



AMNIOTIC FLUID EMBOLISM

- Amniotic fluid embolism (AFE) is a life threatening obstetric emergency characterized by sudden cardiorespiratory collapse and disseminated intravascular coagulation.
- AFE occurs in 2-8 per 100,000 deliveries and is responsible for between 7.5% to 10% of maternal mortality in the United States.
- The diagnosis of AFE has traditionally been made at autopsy when fetal squamous cells are found in the maternal pulmonary circulation

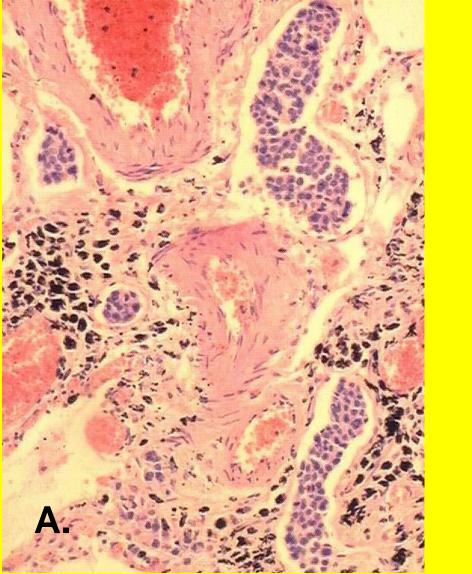
AMNIOTIC FLUID EMBOLISM

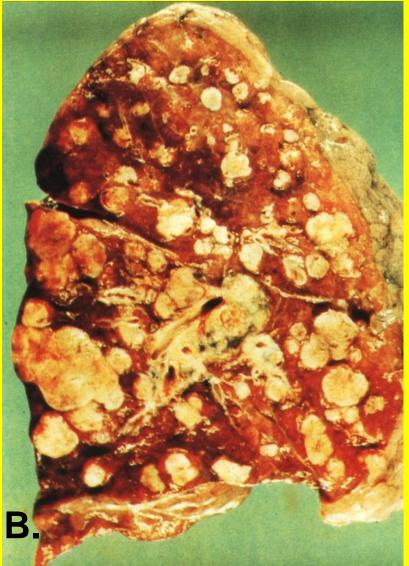


AMNIOTIC FLUID EMBOLISM



NEOPLASTIC (TUMOR, CANCER) EMBOLISM





A. MICROSCOPICAL PICTURE OF METASTATIC EMBOLISM IN LUNG B. METASTATIC EMBOLISM IN LUNG

TONSIL CANCER with Neck Metastasis

