Sudden natural death

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Objectives

- To define the sudden natural death in forensic context.
- Enumerate the different causes of sudden natural death
- Identify the autopsy findings specific to each sudden natural death

The definitions of "sudden" and "unexpected" death

- (WHO)

  Sudden death is death occurring within 24 hours of the onset of symptoms when the death is not instantaneous and not due to sudden cardiac death or sudden infant death syndrome (SIDS)

Definition of sudden cardiac death

Unexpected loss of heart function within minutes/ seconds of onset of symptoms

Most commonly death within 1 hour of the onset of symptoms (ICD-10 code I46.1).

Still other authors - "sudden" death to be synonymous with "instantaneous" death

From the forensic pathologist's prospective

generally viewed in a much broader sense

includes **all** deaths

where an **adequate diagnosis** of a lethal medical disease **has not been reached before death**

regardless of the duration of the illness

The definition of "unexpected"

- Based more on the probability that a decedent would have died at that particular time given the individual's medical history, or lack thereof.
- May occur in previously healthy individuals
- Those with known natural disease when the severity of the person's condition does not explain death.

Sudden unexplained death syndrome (SUDS)

Sudden death in an otherwise healthy individual with no cause identified following a complete and detailed autopsy and death investigation;

also known as "sudden adult death syndrome" or

"sudden arrhythmogenic death syndrome" (SADS).

Sudden unexplained nocturnal death syndrome (SUNDS)

Sudden death during sleep in a young, previously healthy male, typically of Southeast Asian descent

first noted in 1977 in US

Commonly reported among Filipinos.

Sudden unexpected death in epilepsy (SUDEP):

The sudden, unexpected, and nontraumatic death of persons with epilepsy

the postmortem examination fails to reveal a structural or toxicologic cause of death

the event may be witnessed or unwitnessed

can occur with or without evidence of a seizure (excluding documented status epilepticus

Role of the forensic Pathologist

Obtain and review a decedent's past medical history

A complete and thorough autopsy, including external and internal examinations,

Ancillary tests for histopathology, microbiology and toxicology

Saving material for molecular genetic testing.

Consultation with specialists in forensic neuropathology and cardiovascular if necessary

challenges faced by the forensic pathologist

Inability to determine the cause of death

5–10% are still unexplained after a gross autopsy

1– 5% remain unclear (negative) after completion of the gross and histological examination and other laboratory tests

Role of autopsy

Autopsy and particularly autopsy histology still the most accurate

Causes of instantaneous or sudden death

- Dysfunction/abnormalities of the

  Heart and its vessels

  Noncardiac vessels

  Pulmonary system

  Central nervous system (CNS).

Heart and coronary arteries

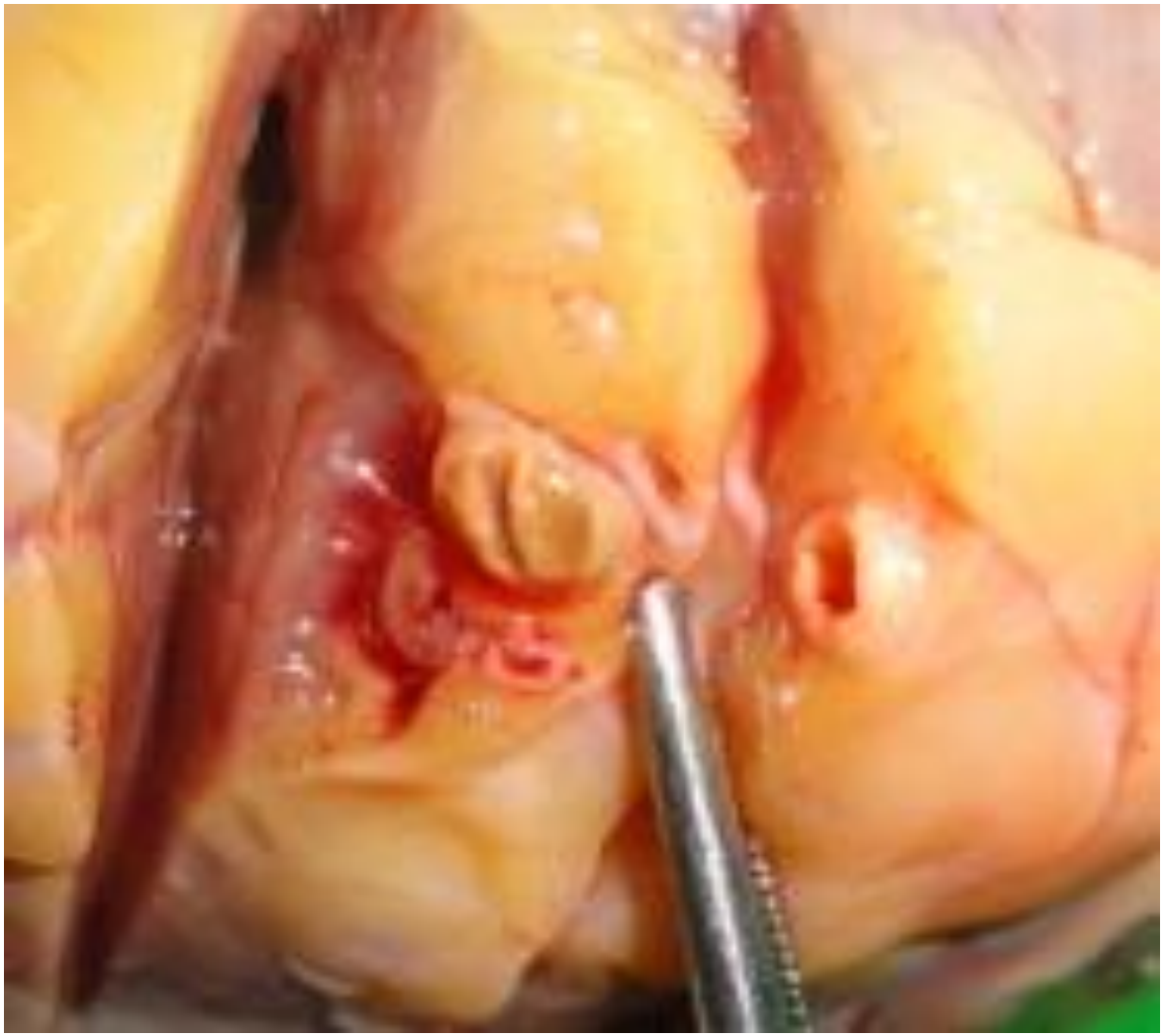
Death is instantaneous/sudden with ventricular fibrillation

Coronary arteries:

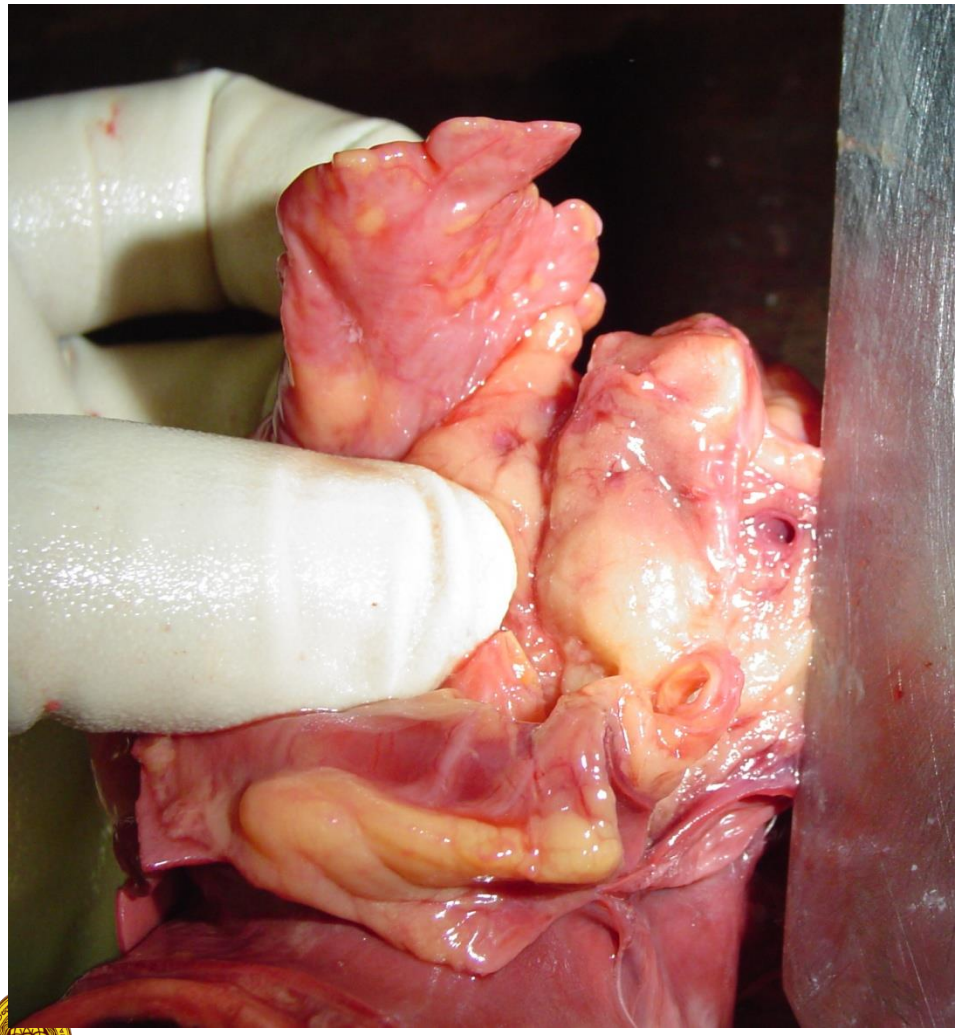
- Occlusion (atherosclerosis, thrombosis, embolus)
- Structural abnormalities of the epicardial arteries
- Coronary artery dissection or aneurysm

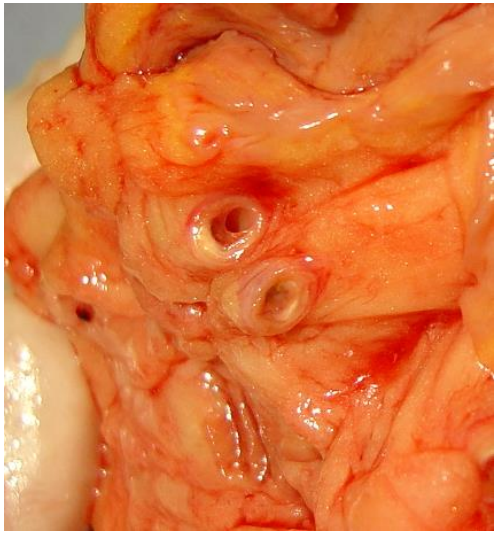
CORONARY ARTERY DISSECTION

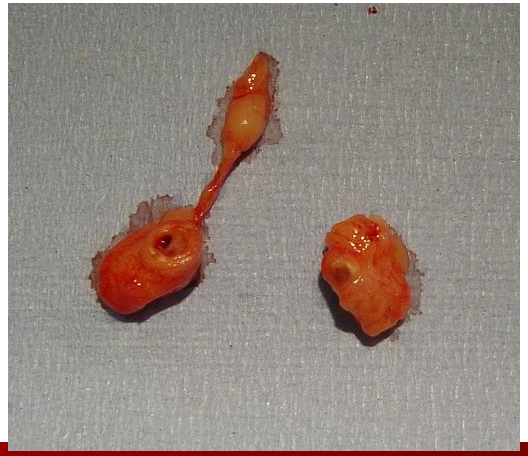
Narrowing of coronary lumen



Narrowing of coronary lumen







Sectioned at 2-3 mm intervals to identify any significant stenosis or thrombotic occlusion.

Acute thrombotic occlusion may be very focal

The degree of luminal stenosis, should be recorded.

75% is more likely to be significant

COD-coronary artery disease (CAD) with less stenosis if other signs present

Pathologic lesions of the epicardial vessels

- Coronary artery occlusion
- Coronary artery aneurysm/rupture: (Kawasaki disease, vasculitis, connective tissue disease)
- Coronary artery dissection: associated with increased eosinophils
- Arteritis: (Multiple vasculitides, rheumatic conditions, connective tissue disorders, and drugs (eg, cocaine)
- Coronary artery spasm
- Congenital anomalies of coronary vessels: (hypoplasia, tunneling)

Myocardial infarction

12 -24 hrs	• First 12 hrs no naked eye changes • 24 hours pale area
1-3 days	• Area better demarcated and yellow • Tigroid appearance due to interfiber haemorrhage
3-5days	• Yellow area with red margin, soft • Myomalasia cordis (rupture of the heart)
7d-3 weeks	• Center gelatinous and some hemorrhage inside

Myocardial infarction microscopy

- 1 and 4 hours - Contraction bands and wavy fibers
- up to 12 hours - Progressive necrosis, edema, and hemorrhage
- Between 12 and 24 hours –
- neutrophilic inflammation becomes detectable, myocytes become Hypereosinophilic,
- the nuclei become pyknotic
- distinct contraction band necrosis become apparent
- From 1 to 3 days- acute inflammation and coagulative necrosis are predominant (myocyte nuclei and striations are absent)

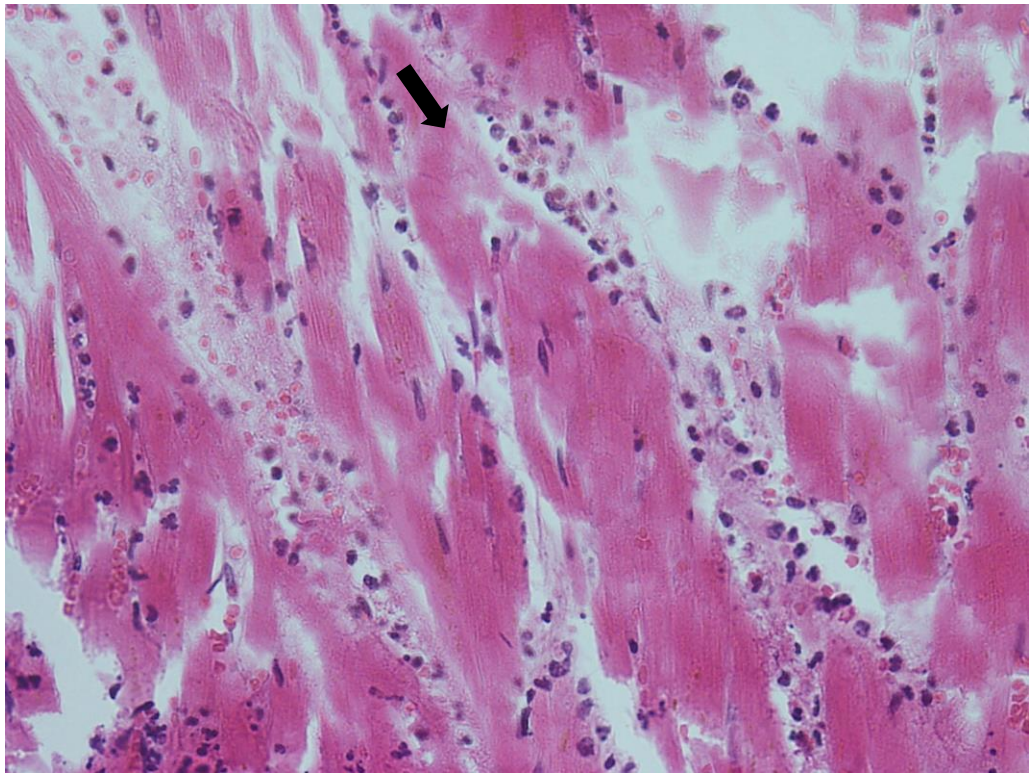
Disintegration of the dead cells and replacement

- Neutrophilic inflammation replaced by macrophages, particularly at the edge of the infarct occurs from 3 to 7 days, and becomes well-developed within 10 days

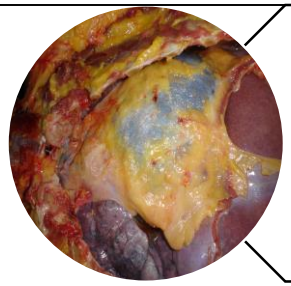
- Granulation tissue with collagen deposition - approximately 2 weeks, and by 2 months

- A scar is formed, with progressive loss of cellularity from there onward

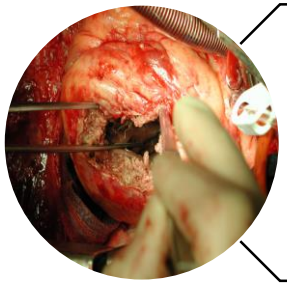
Mi – Microscopy



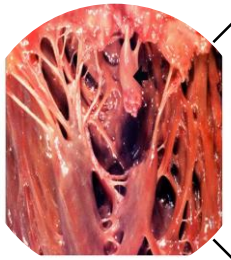
Complications of MI



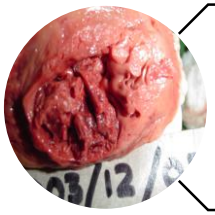
Myocardial rupture: haemopericardum



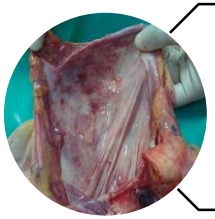
Intraventricular



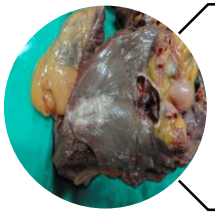
Papillary muscle rupture



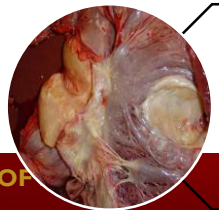
Mural thrombus: systemic embolism



Pericarditis



Myocardial fibrosis

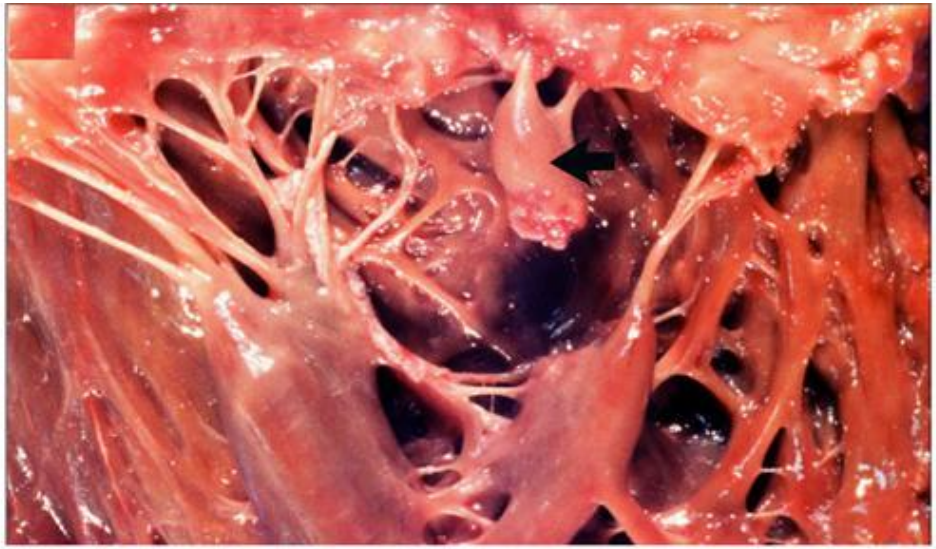


Myocardial Aneurysm

Myocardial rupture

- Causes sudden death from haemopericardium and cardiac tamponade
- Common in old women- soft senile myocardium
- Tends to take place 2-3 days after onset of the infarct when necrotic muscle is becoming soft

Rupture of the papillary muscles



- Rupture occur due to infarction and necrosis
- Allows mitral valve to prolapse
- Valve insufficiency
- Sudden death

Other cardiac causes of sudden death

Hypertensive heart disease: Fibrosis

Inflammatory: Myocarditis, pericarditis (bacterial, viral, Dressler syndrome)

Cardiomyopathies

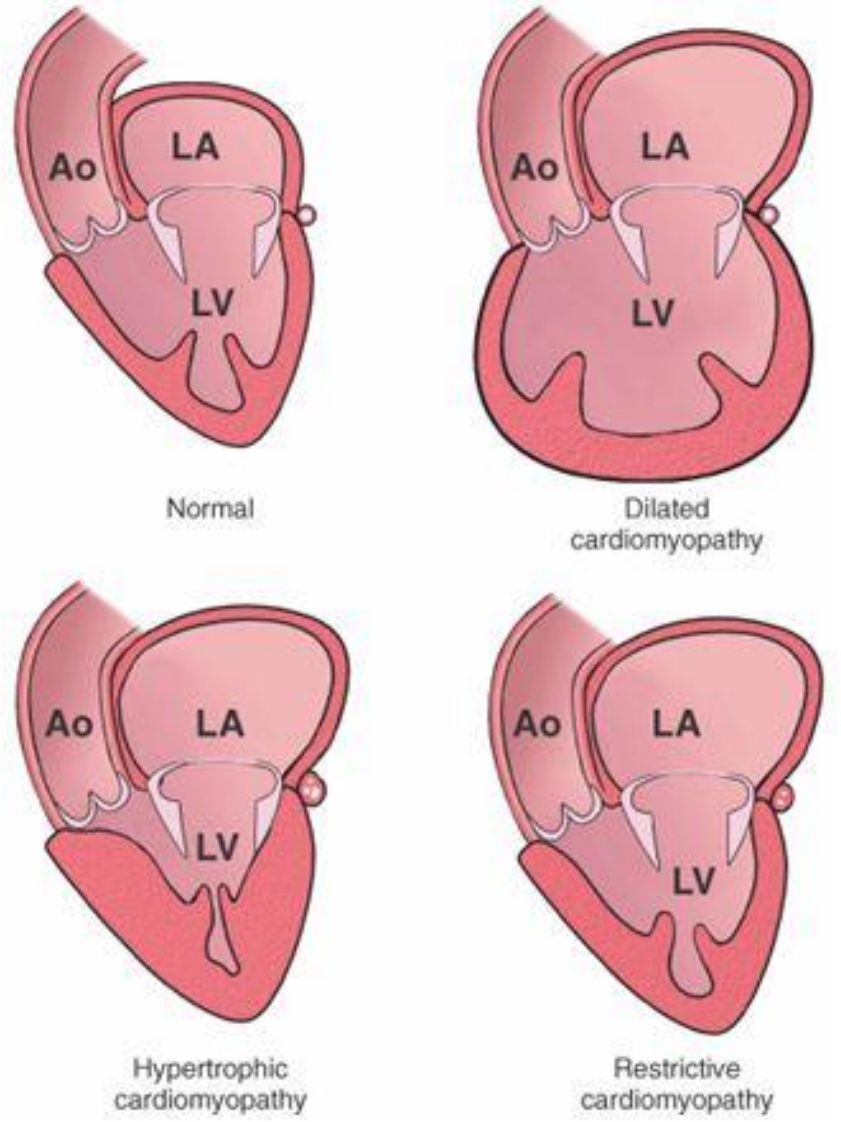
Cardiomyopathies

Hypertrophic cardiomyopathy,

Dilated cardiomyopathy (genetic, myocarditis, toxic, peripartum, idiopathic)

Restrictive cardiomyopathy (idiopathic, amyloidosis, sarcoidosis, radiation fibrosis, metastasis, deposition disease/inborn errors of metabolism)

Cardiomyopathies



Hypertrophic cardiomyopathy (HCM)

Asymmetric with a greater degree of thickening of the interventricular septum than the left ventricular free wall

Fiber disarray is characteristic microscopy but may be focal

Can develop into an end-stage dilated cardiomyopathy

Dilated cardiomyopathy

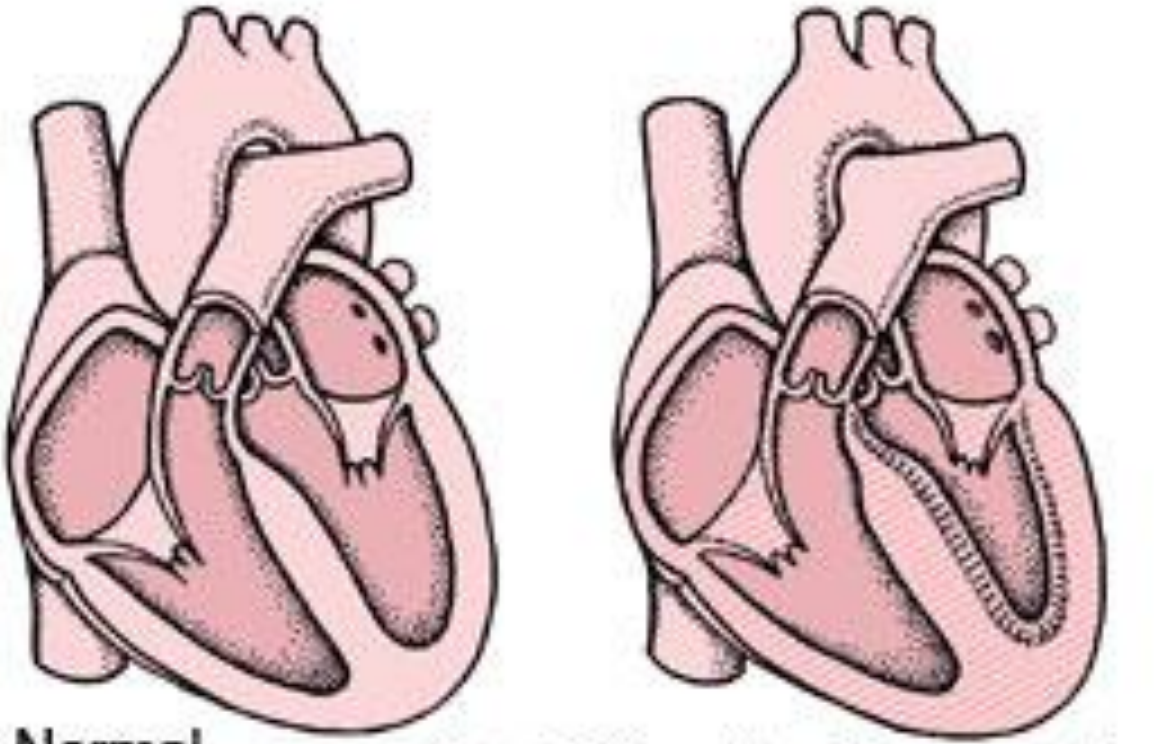
- Recognized by an enlarged, flabby heart with a dilated left ventricle

- The degree of left ventricular dysfunction is the best predictor of mortality

Restrictive cardiomyopathy

- Results from decreased myocardial compliance
- Typically secondary to fibrosis,
- Can also be due to infiltrative processes such as amyloidosis or sarcoidosis, in addition to various deposition diseases;

Restrictive cardiomyopathy



Normal

Restrictive Cardiomyopathy

In restrictive cardiomyopathy, the walls of the ventricles become stiff, but not necessarily thickened.

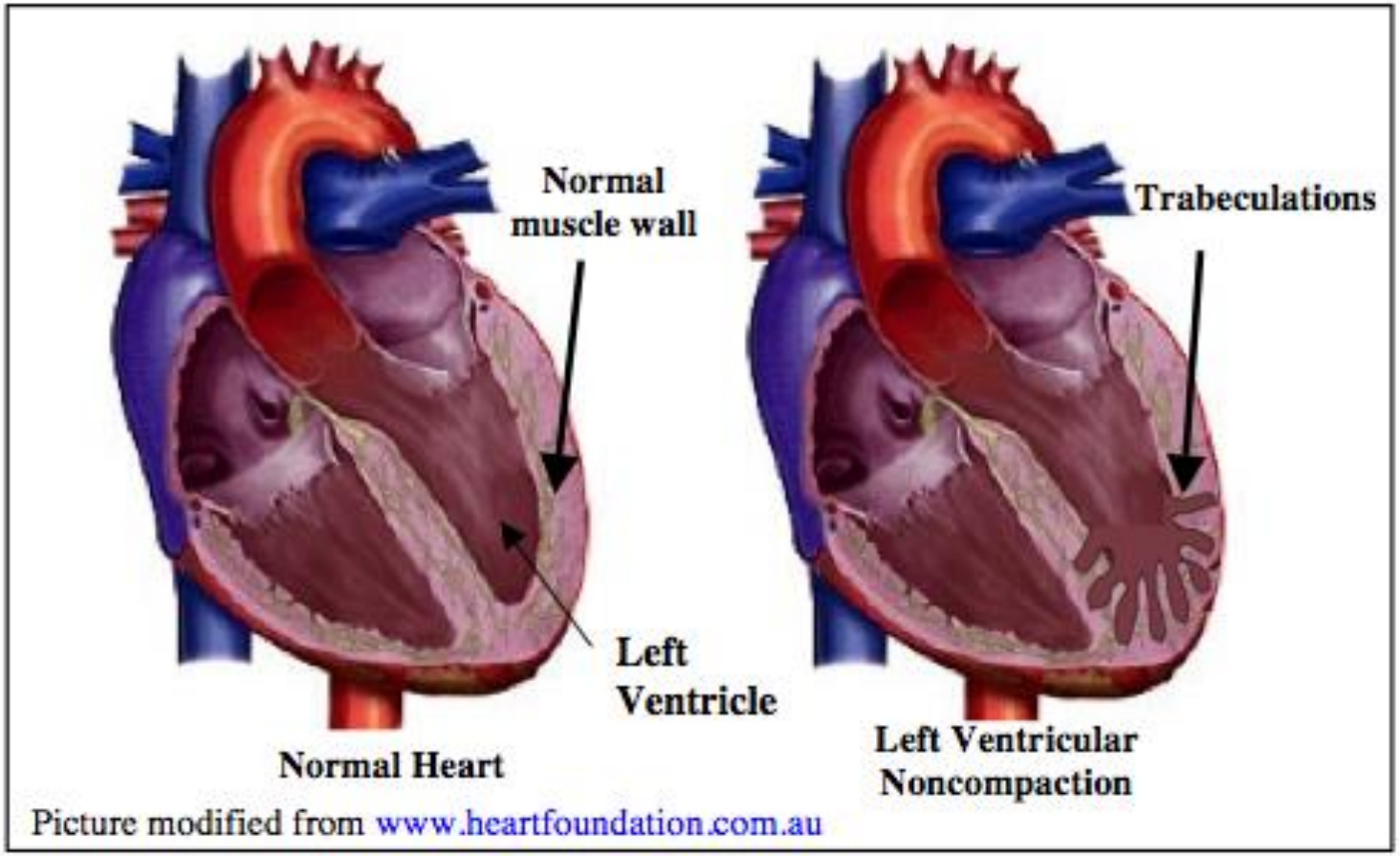
Physiologic hypertrophy

- Typically seen in high level athletes
- Not associated with sudden death

rare cardiomyopathies

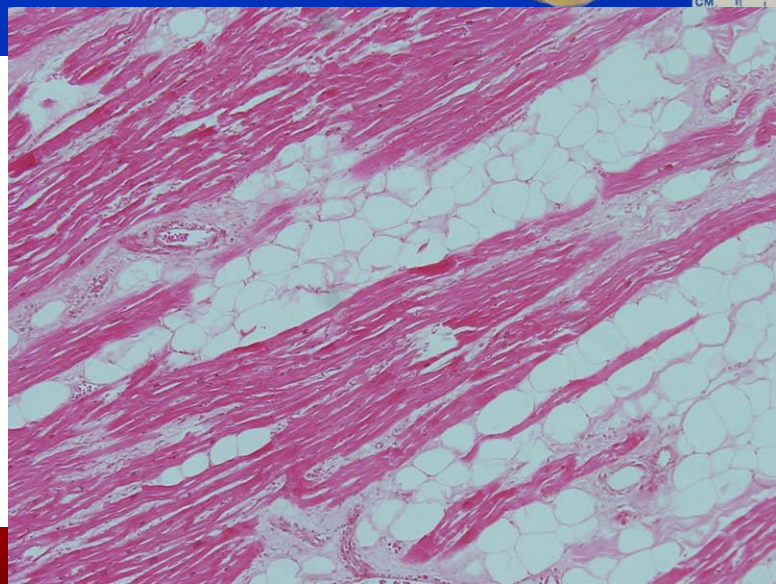
- Left ventricular noncompaction
- Arrhythmogenic right ventricular dysplasia (ARVD)

Left ventricular non compaction cardiomyopathy



Arrhythmogenic Right Ventricular Dysplasia (ARVD)

- Inherited form of right ventricular cardiomyopathy characterized by fibrofatty infiltration
- Common causes of sudden cardiac death, particularly in athletes.
- Caused by desmosome mutations.



Other cardiomyopathies:

Arrhythmogenic left ventricular dysplasia,

restrictive conditions

- endomyocardial fibrosis
- Loeffler endomyocarditis
- endocardial fibroelastosis

histologic features

- Restrictive cardiomyopathy
  - Mostly nonspecific patchy fibrosis
- Endomyocardial fibrosis
  - Fibrosis of the endocardium as well as subendocardium
- Endocardial fibroelastosis,
  - fibroelastic thickening of the endocardium
- Loeffler endomyocarditis
  - endocardial fibrosis with focal necrosis and eosinophilic infiltrates
- In dilated cardiomyopathy,
  - nonspecific myocyte hypertrophy and interstitial fibrosis.

HYPERTENSIVE HEART DISEASE

- Myocardium work against increasing systemic arterial pressure
- Hypertrophy of the heart. Upper limit of normal heart wt 400g
- As etiologies of HT and atheroma are similar atherosclerosis and HTsive heart disease co-exist
- Hypoxic and ischemic damage to muscle is unstable and irritable – arrhythmias and fibrillation.
- Death from hypertensive Heart disease shows signs of CCF (pulmonary oedema)

Cor pulmonale

- Right ventricular hypertrophy secondary to pulmonary pathology
- Death -from arrhythmia exacerbated by hypoxia (risk of death during sleep)
- Lung/lungs inflated and fixed before sectioning

Right ventricular hypertrophy

Determined by the right ventricular weight

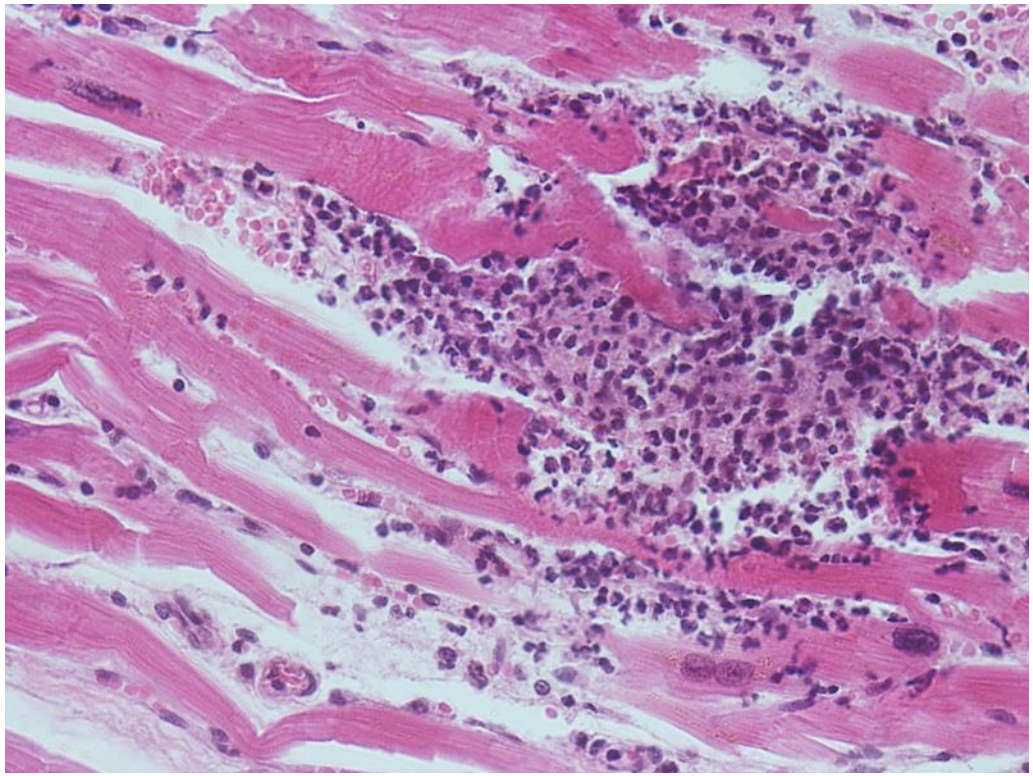
- Due to pulmonary disorders
  - pulmonary hypertension
  - chronic obstructive pulmonary disease [COPD]
  - interstitial fibrosis
  - bronchopulmonary shunts
  - chronic pulmonary emboli
  - bronchitis

Myocarditis

- Diffuse or focal infiltrates composed mostly of lymphocytes but also some neutrophils, macrophages, and plasma cells

- Myocyte necrosis is focal and associated with inflammatory infiltrates

Viral myocarditis



valvular dysfunction

Commonly due to

- Degenerative processes due to aging,
- Hypertension
- Structurally abnormal valves (bicuspid valves)
- Postinflammatory scarring (rheumatic heart disease)
- Infective endocarditis
- Mitral valve prolapse

(ballooning of thick, redundant valve leaflets; the chordae tendineae are typically thinned or ruptured)

- Rheumatologic and connective tissue diseases
- Marfan syndrome

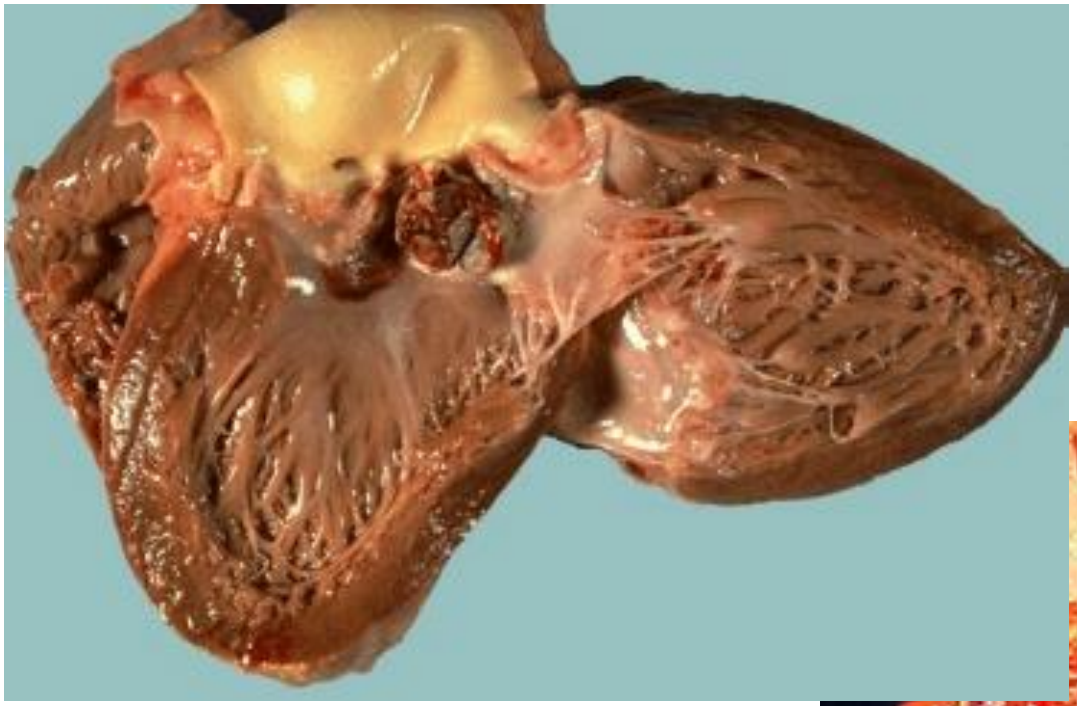
AORTIC STENOSIS

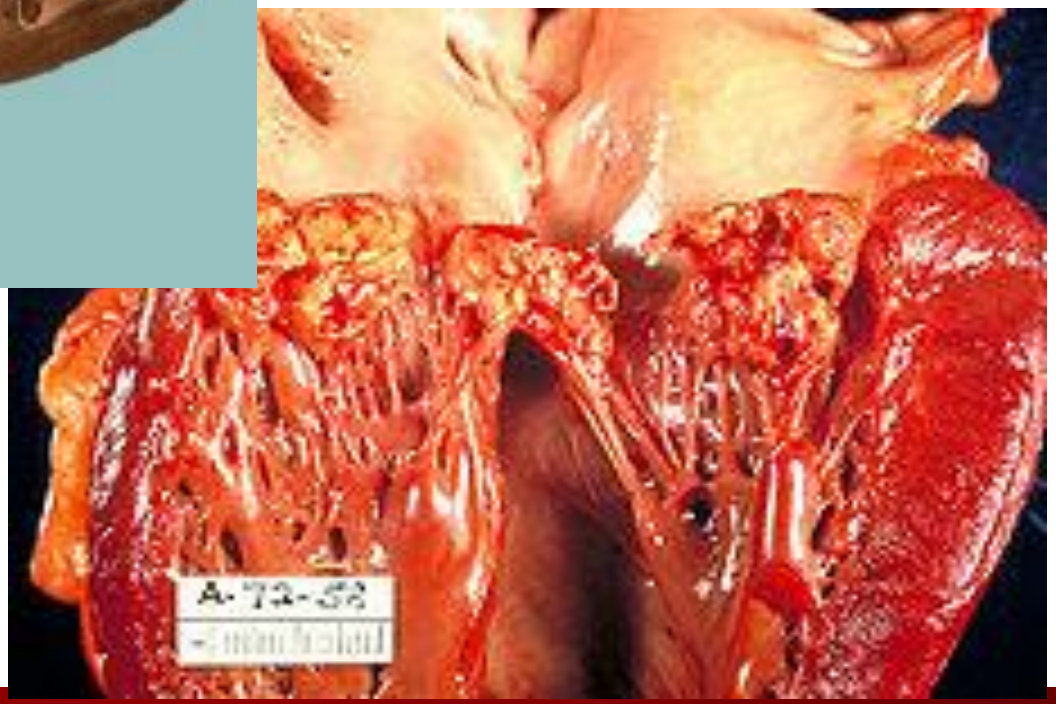
- Leads to left ventricular hypertrophy
- More marked than in hypertension
- Cause is calcific stenosis of aortic valve
- Affects males over 60 years
- Seen in younger people who have congenital bicuspid aortic valve
- Low perfusion pressure in coronary ostia

Endocarditis

- can be either septic or aseptic;
- large, friable and destructive vegetations involving previously normal valves
- caused by virulent organisms such as Staphylococcus aureus and have a rapid progression
- septic infarcts are identified in the spleen, kidney, and brain
- Subacute bacterial endocarditis is typically less destructive and show more fibrosis and granulation

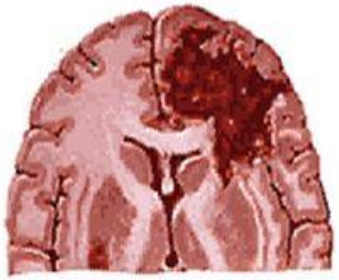
INFECTIVE ENDOCARDITIS





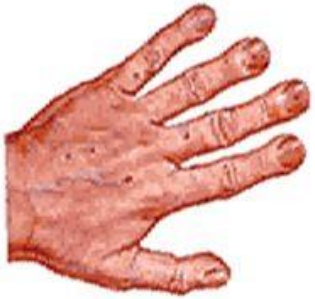
Bacterial Endocarditis Remote Embolic Effects

Endocarditis: Extracardiac Complications



Infarct of brain with secondary hemorrhage from embolism to r. ant. cerebral artery

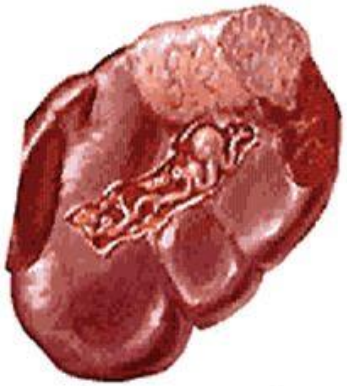
Embolis of vessel of occular fundus with retinal infarction



Multiple peticheae of skin and clubbing of fingers

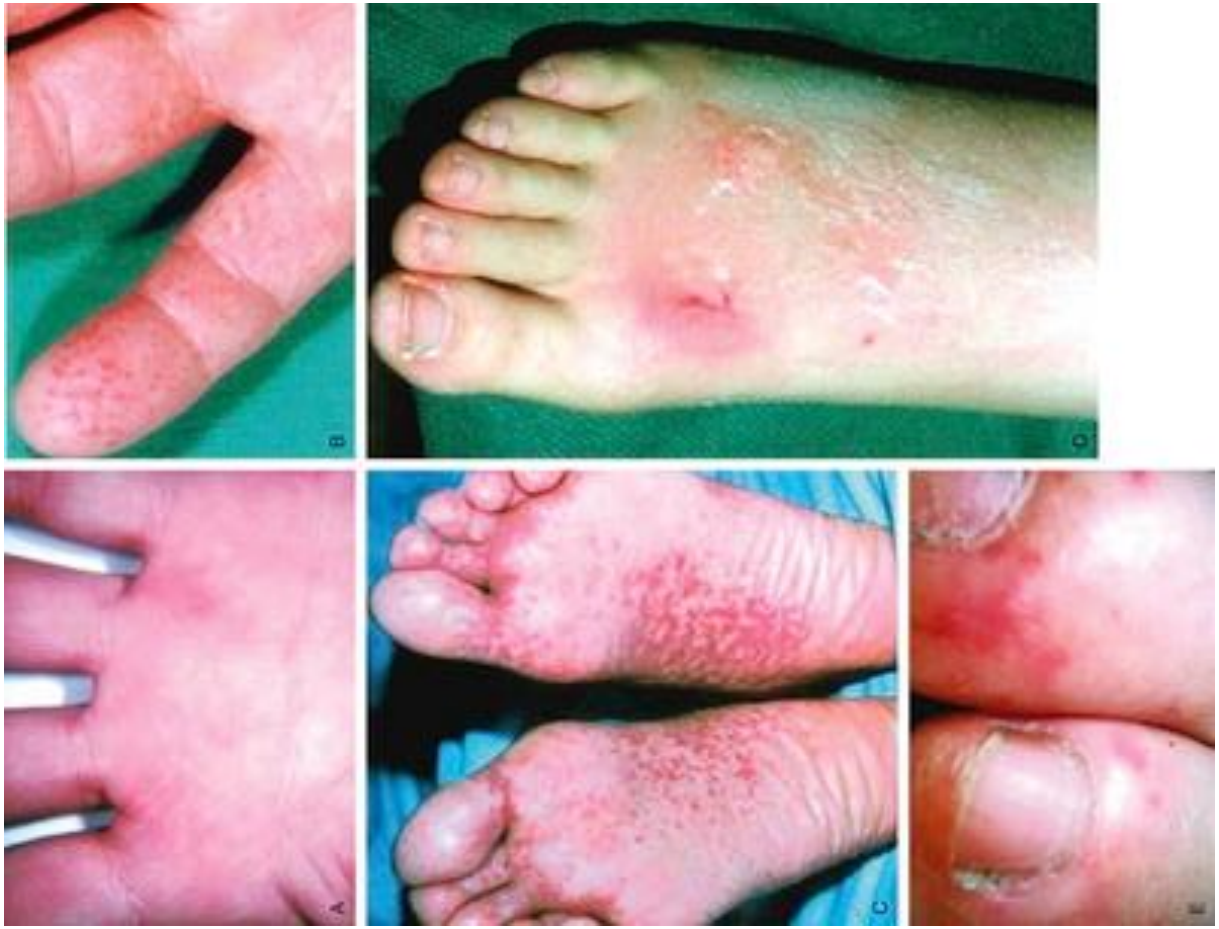
Peticheae of muccous membranes

Peticheae and gross infarcts of kidney



Mycotic aneurysms of splenic arteries and infarct of spleen, splenomegaly

Vascular skin lesions of endocarditis



Functional abnormalities:

Long QT syndrome (LQTS)

Short QT syndrome (SQTS)

Catecholaminergic polymorphic ventricular tachycardia (CPVT)

episodic syncope, with exertion/emotion

no structural cardiac abnormalities

Brugada syndrome

abnormal ECG

dies of VF

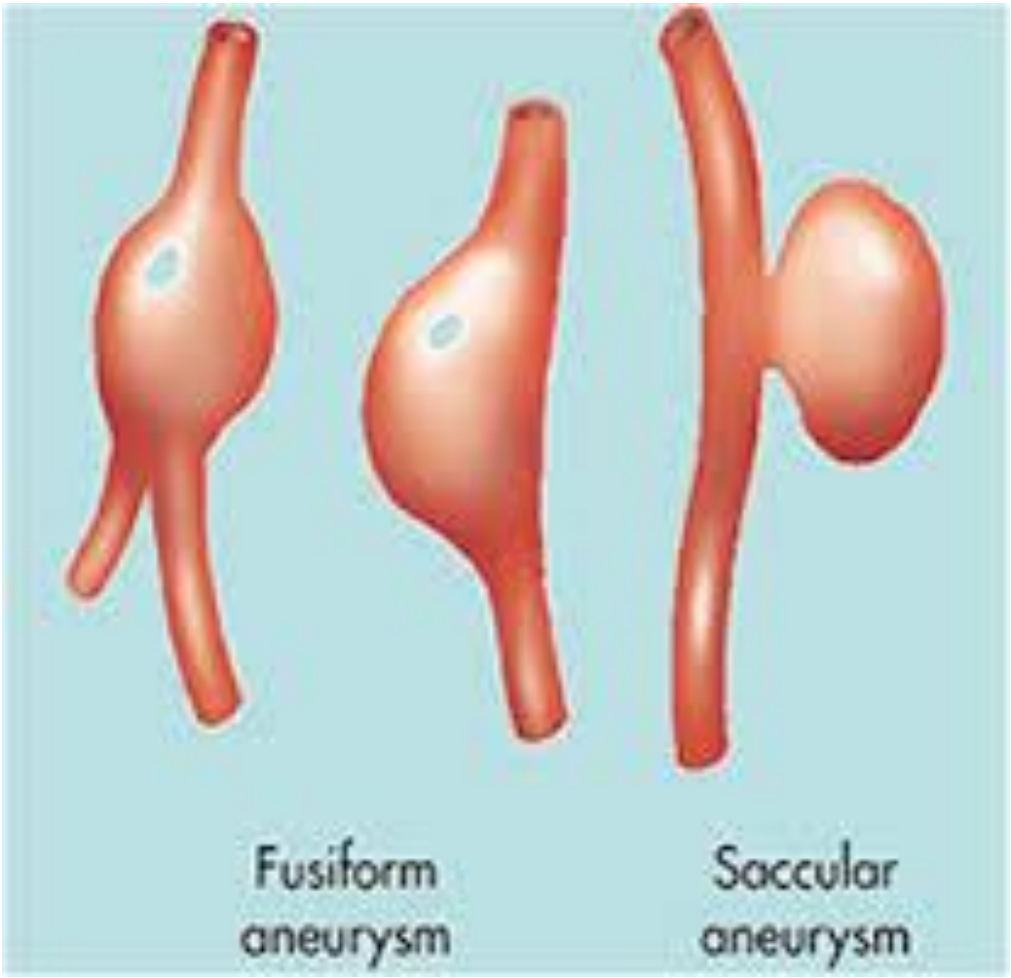
Asian sudden unexplained nocturnal death syndrome (SUNDS)

Aorta and noncardiac vessels

- Aneurysmal rupture (atherosclerotic, mycotic, traumatic aneurysm):
  - causing cardiac tamponade, intrapleural or retroperitoneal hemorrhage
- Aortic dissection:
  - Coronary ostia occlusion due to retrograde dissection,
  - infarction of abdominothoracic organs

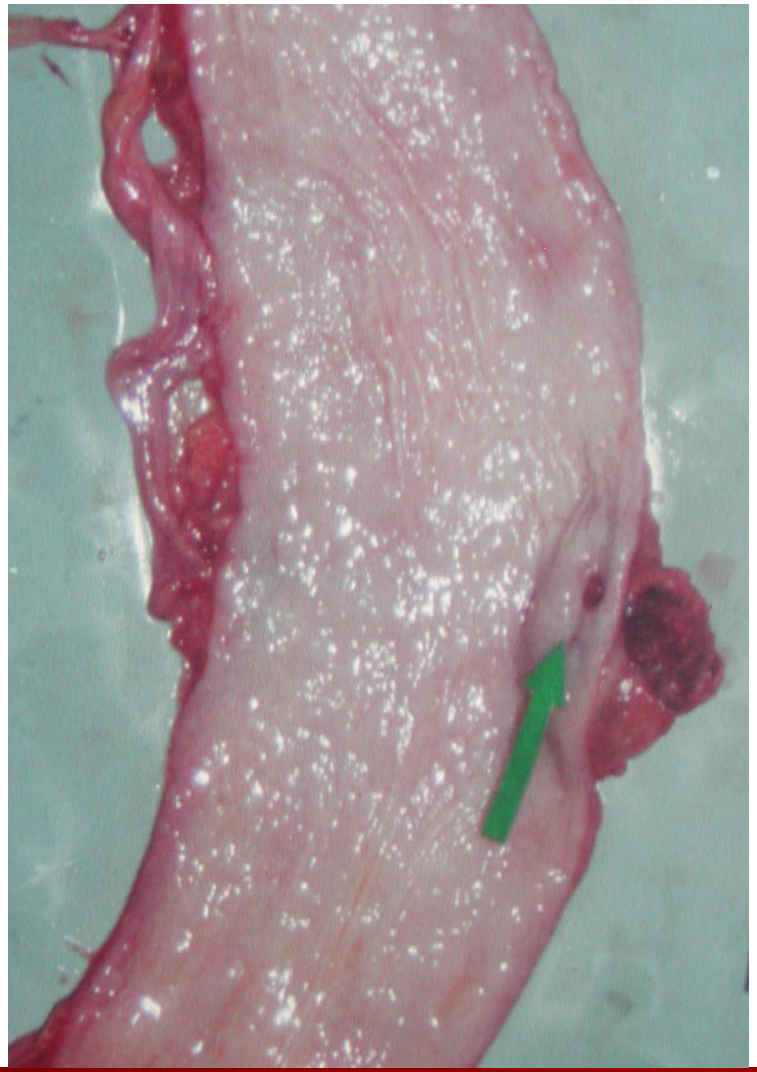
ATHEROMATOUS ANEURYSM

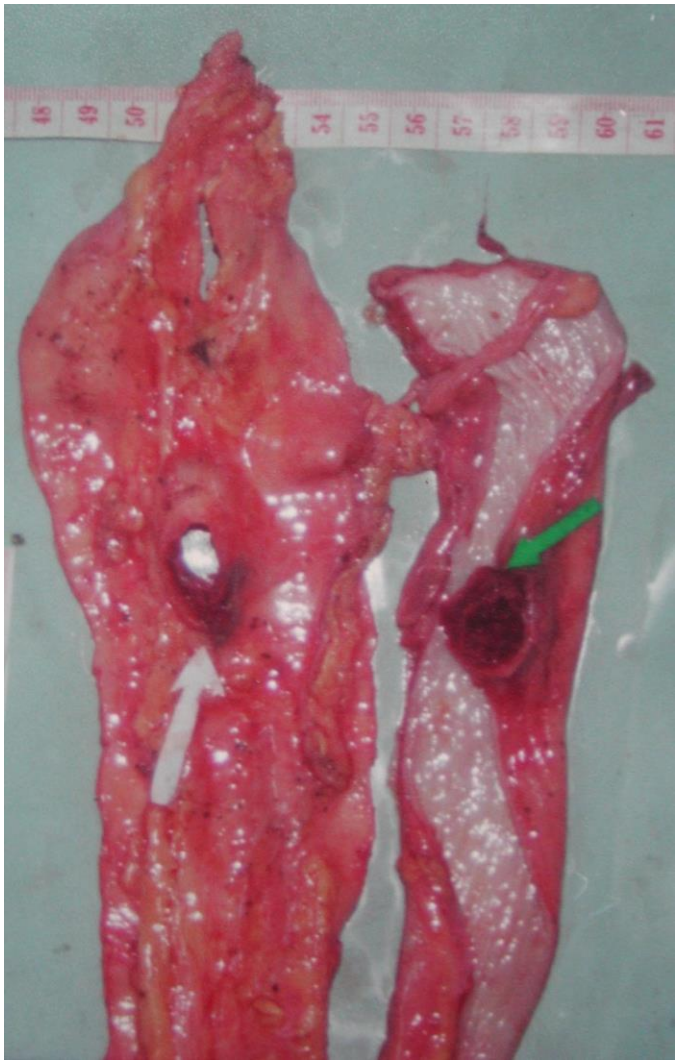
- Most often in abdominal aorta
- Damage to the wall by atherosclerosis
- Elderly and male
- Saccular – expanding to one side or fusiform – cylindrical


Fusiform
aneurysm
Saccular
aneurysm

- Vessel wall thinned out
- Destruction of elastic tissue
- Interior ulcerated
- May see a thrombus intact
- Rupture- massive haemorrhage

Atheromatus Thoracic aneurysm

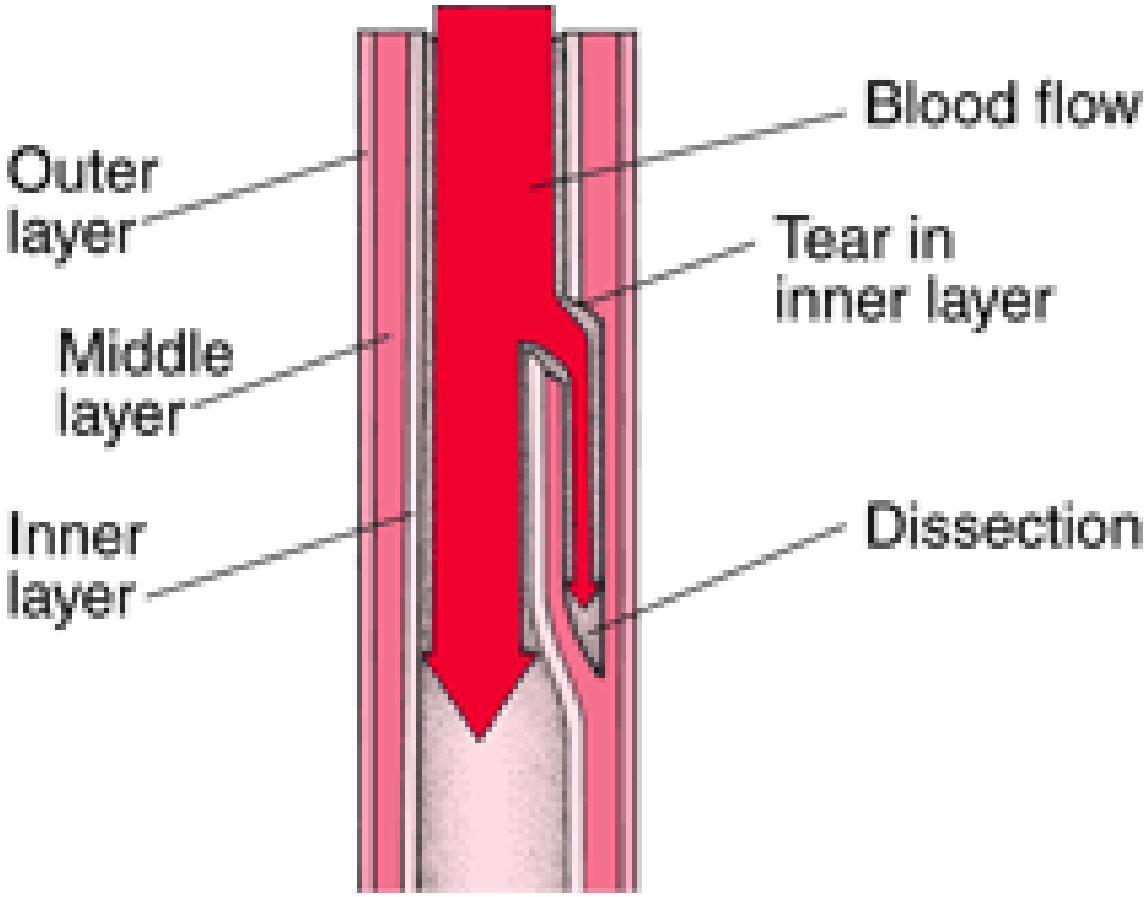




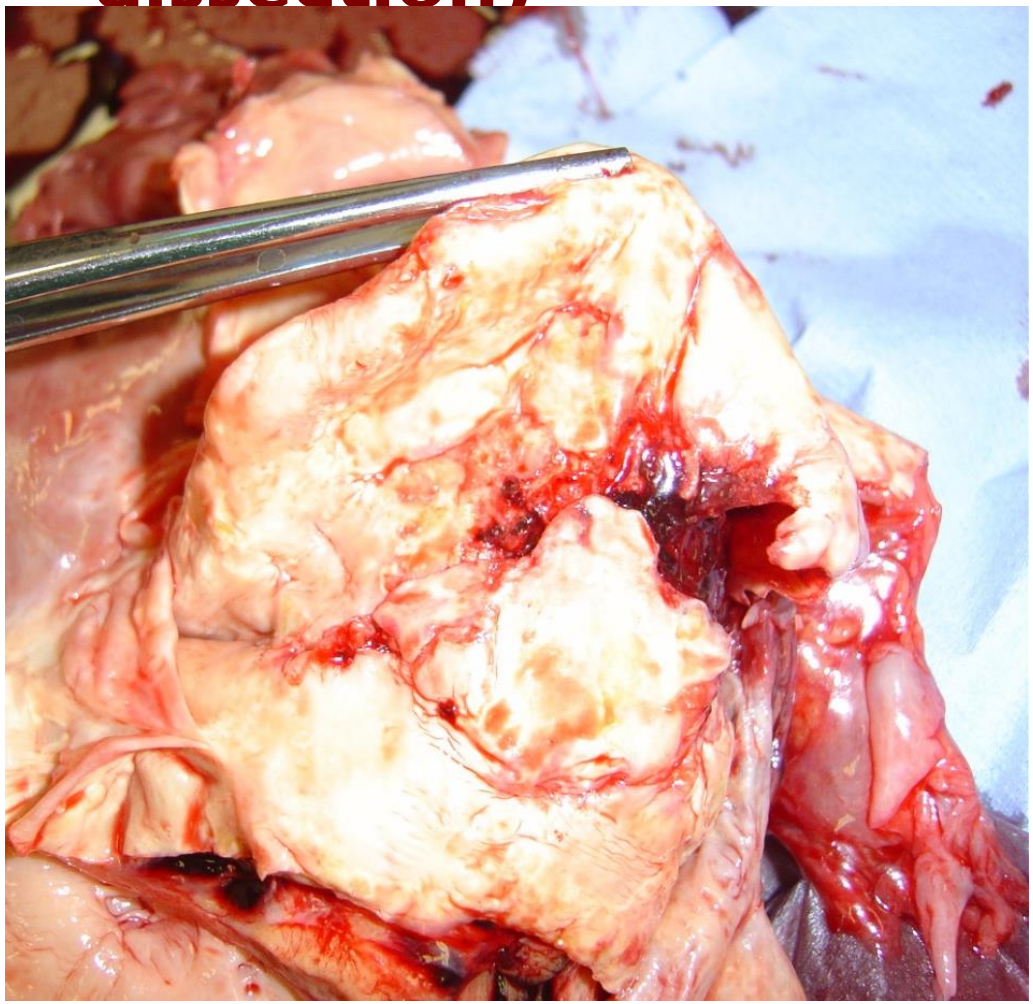
Dissecting aneurysm of aorta (Aortic dissection)

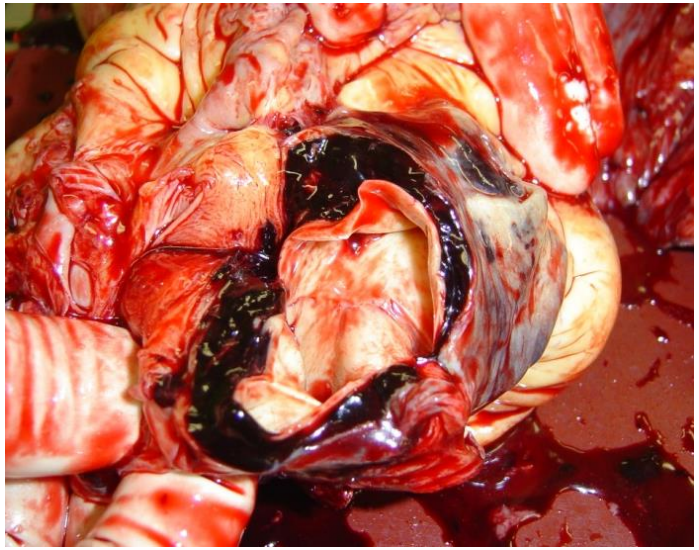
- Very rare
- Medio-necrosis of the wall of the aorta
- Weakening of the central layers of the wall
- Lumen rupture through atheromatus plaque
- Influx blood dissect the aortic wall downwards into iliac or femoral branches
- Tearing of dissecting aneurysm can produce worst pain possible

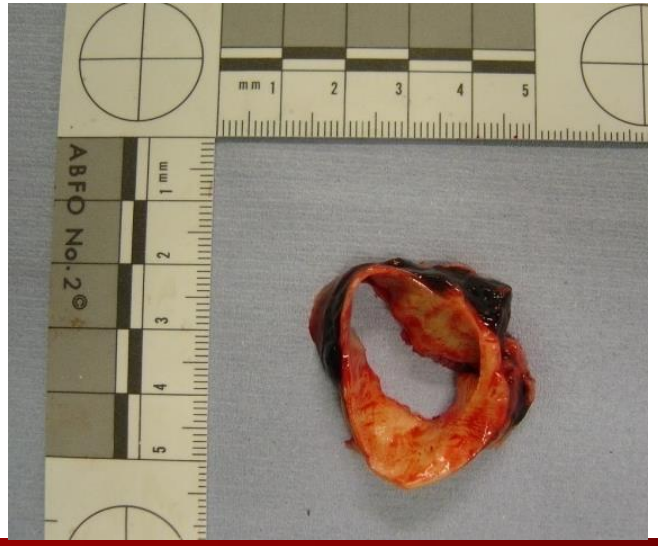
Dissection of aorta



Dissecting aneurysm of aorta (Aortic dissection)



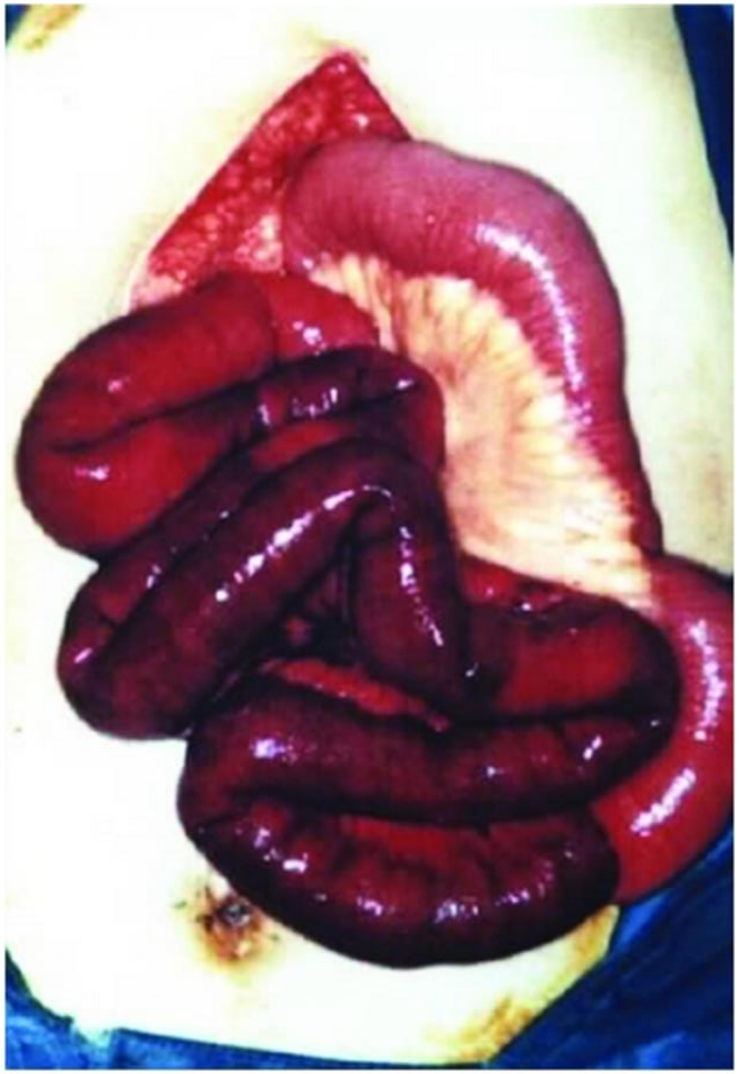




Other vessels

Mesenteric artery thrombosis:

Bowel infarction and septic shock



Pulmonary system

Laryngeal edema from infection (epiglottitis)

Anaphylaxis

Neoplasm of larynx

Sleep apnea (Ondine's curse, Pickwickian syndrome)

Pneumothorax

Bronchial asthma

Pulmonary embolism:

- Embolization from pelvic or deep veins of the lower extremities, fat/bone marrow emboli, amniotic fluid embolus, air embolus

Massive acute hemorrhage:

- Neoplasms, pulmonary tuberculosis (Rasmussen aneurysm), lung abscess

Lungs are examined for areas of

Consolidation

Infarcts

Parenchymal loss

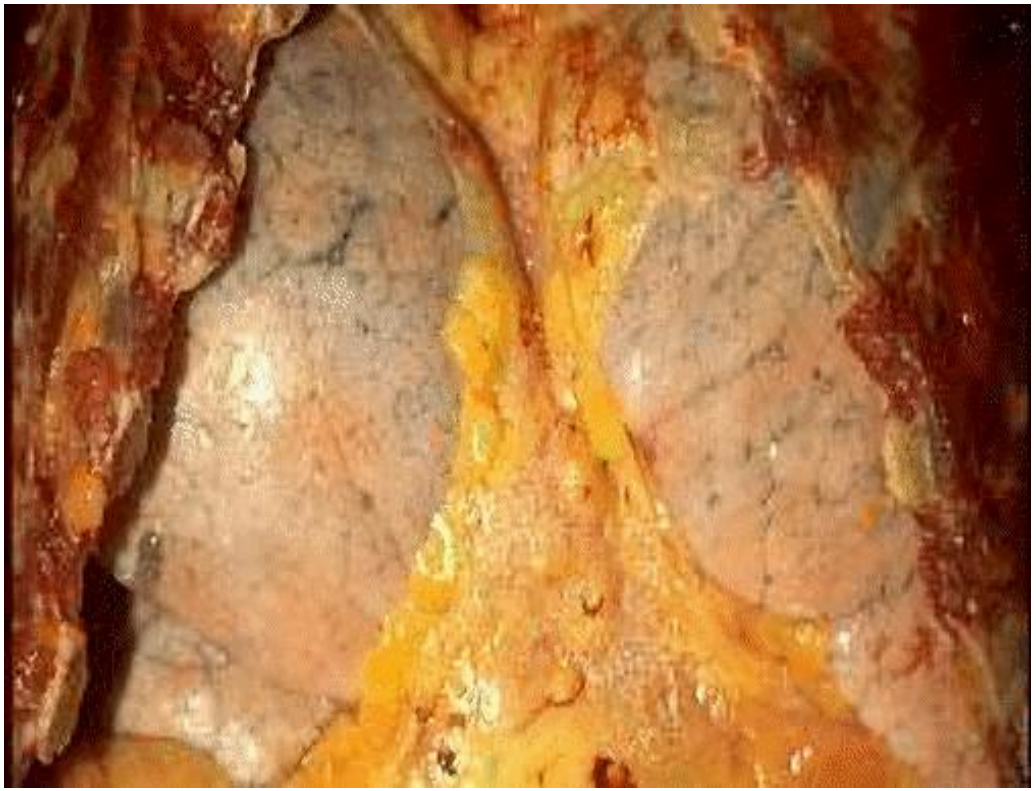
Fibrosis

Oedema

Small- to medium-sized vessels -small pulmonary emboli (confirm with histology)

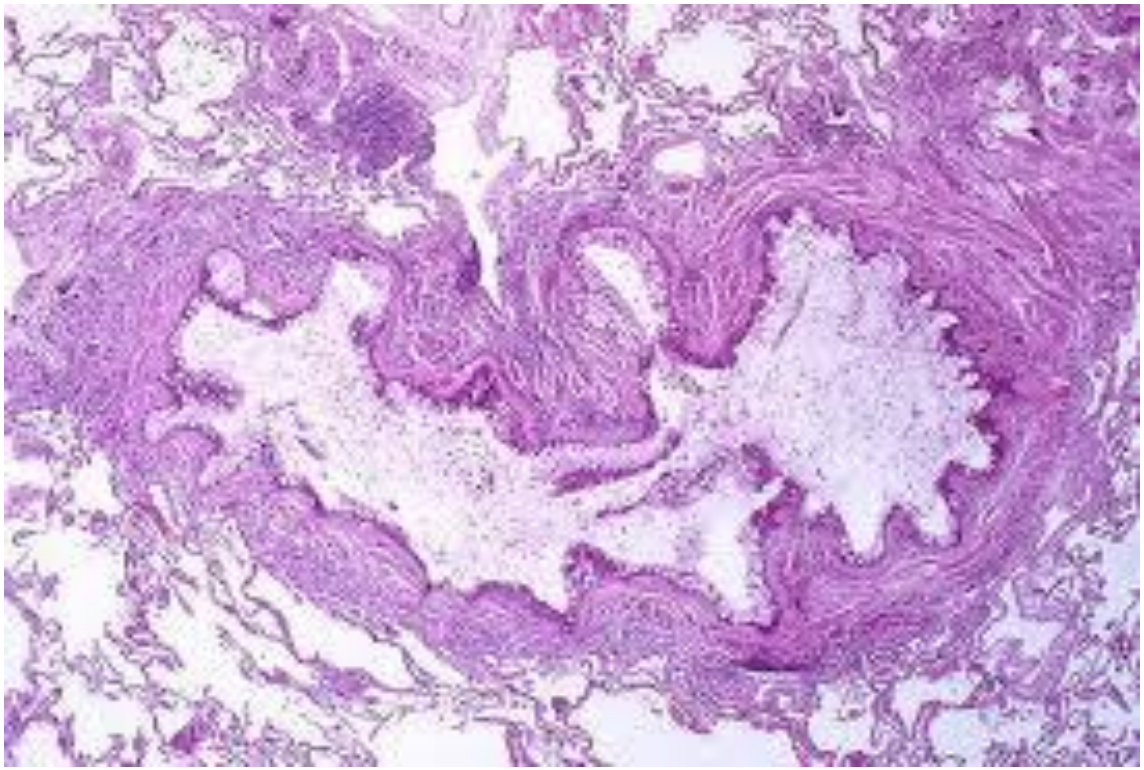
asthma-related deaths

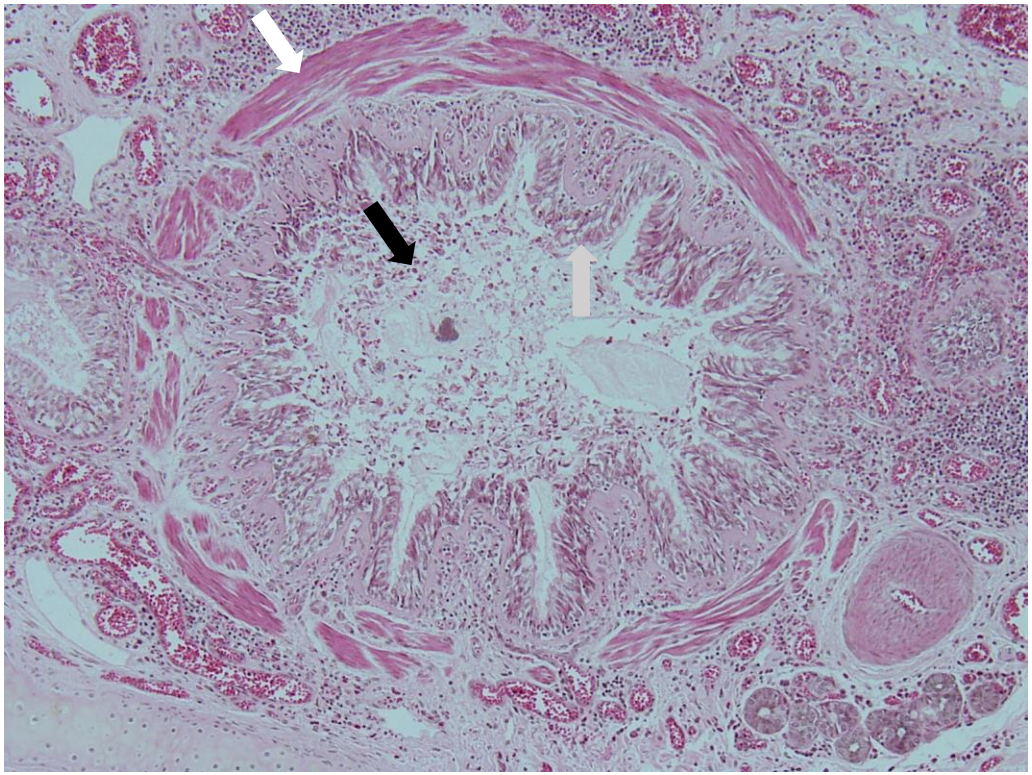
- Lungs may appear hyperinflated
- Rib indentations



Histologic features characteristic of status asthmaticus

- acute airway edema
- thick, cellular mucus plugs blocking small bronchi and bronchioles
- the mucus contains eosinophils, Charcot-Leiden crystals, and sloughed epithelial cells;
- chronic changes in longstanding asthma include airway remodeling with fibrosis, muscular hypertrophy of bronchial wall
- Mucus gland hyperplasia

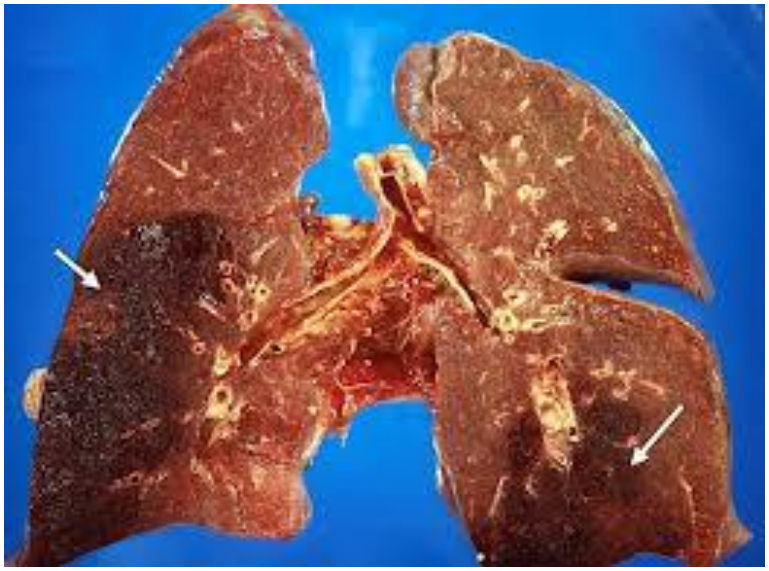


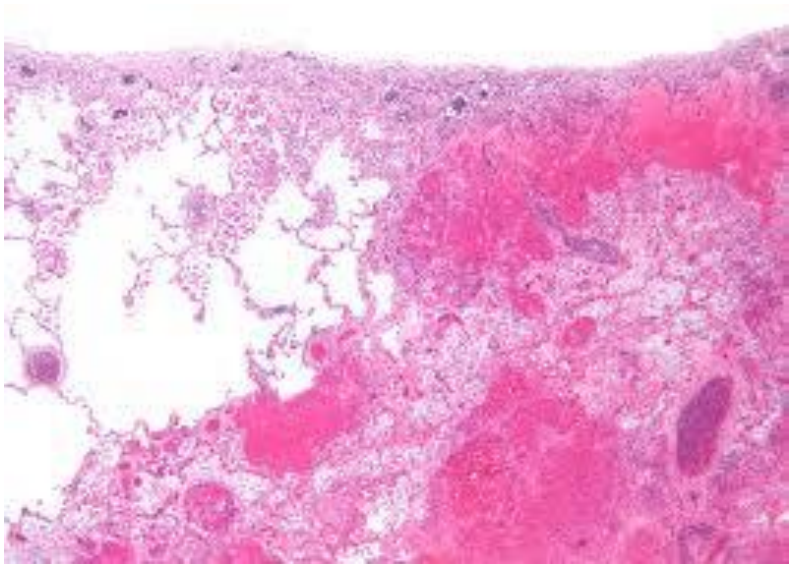


Pulmonary infarcts

- Usually due to systemic emboli,
- Commonly seen in the lower lobes and in older individuals
- Appear as raised, hyperemic, wedge- shaped areas arising at the periphery of the lung.

PULMONARY INFARCTION





Pneumonia

- Bronchopneumonia

  Patchy areas of consolidation

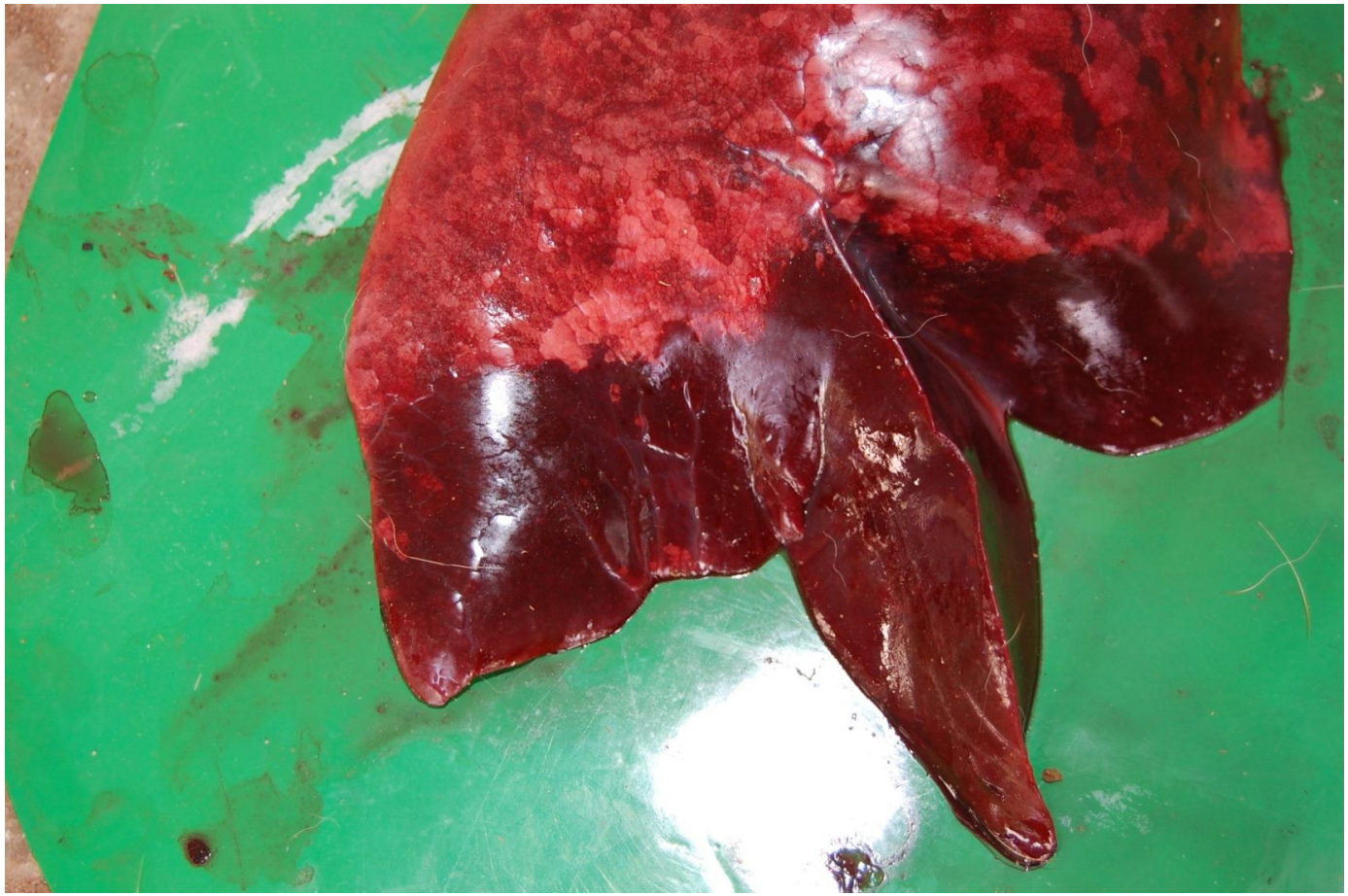
- Lobar pneumonia

  typically involves an entire lobe with variable gross appearance (congestion, red hepatization, gray hepatization, and resolution) with or without fibrinous pleural exudates;

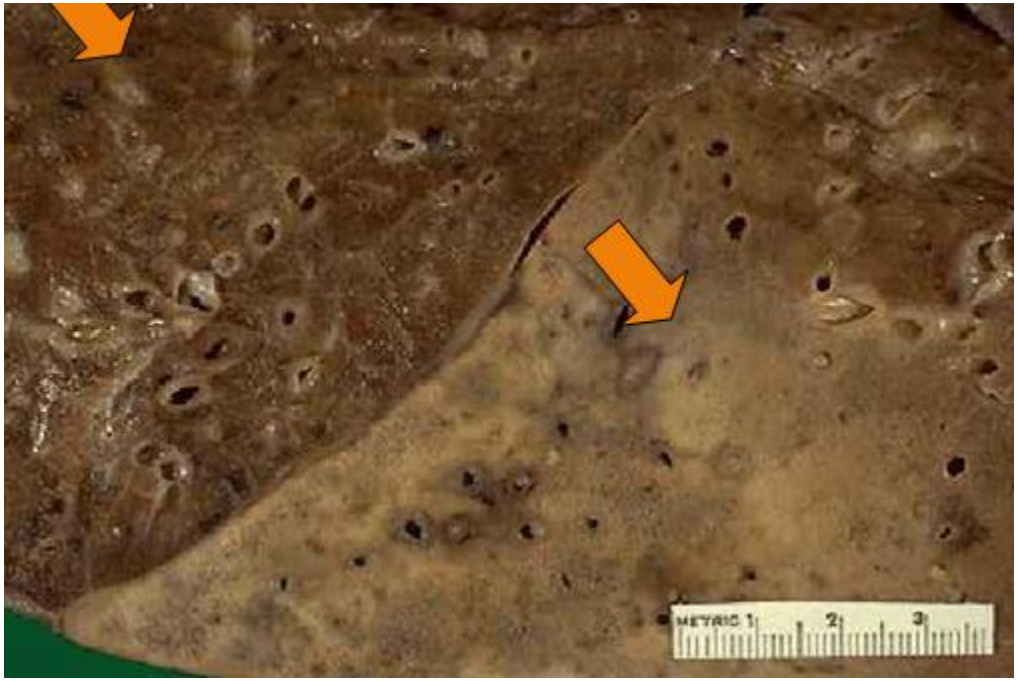
- viral and atypical pneumonias

  patchy areas of congestion with a variable distribution throughout one or all lobes

RED HEPATIZATION



GREY HEPATIZATION



Lung abscesses

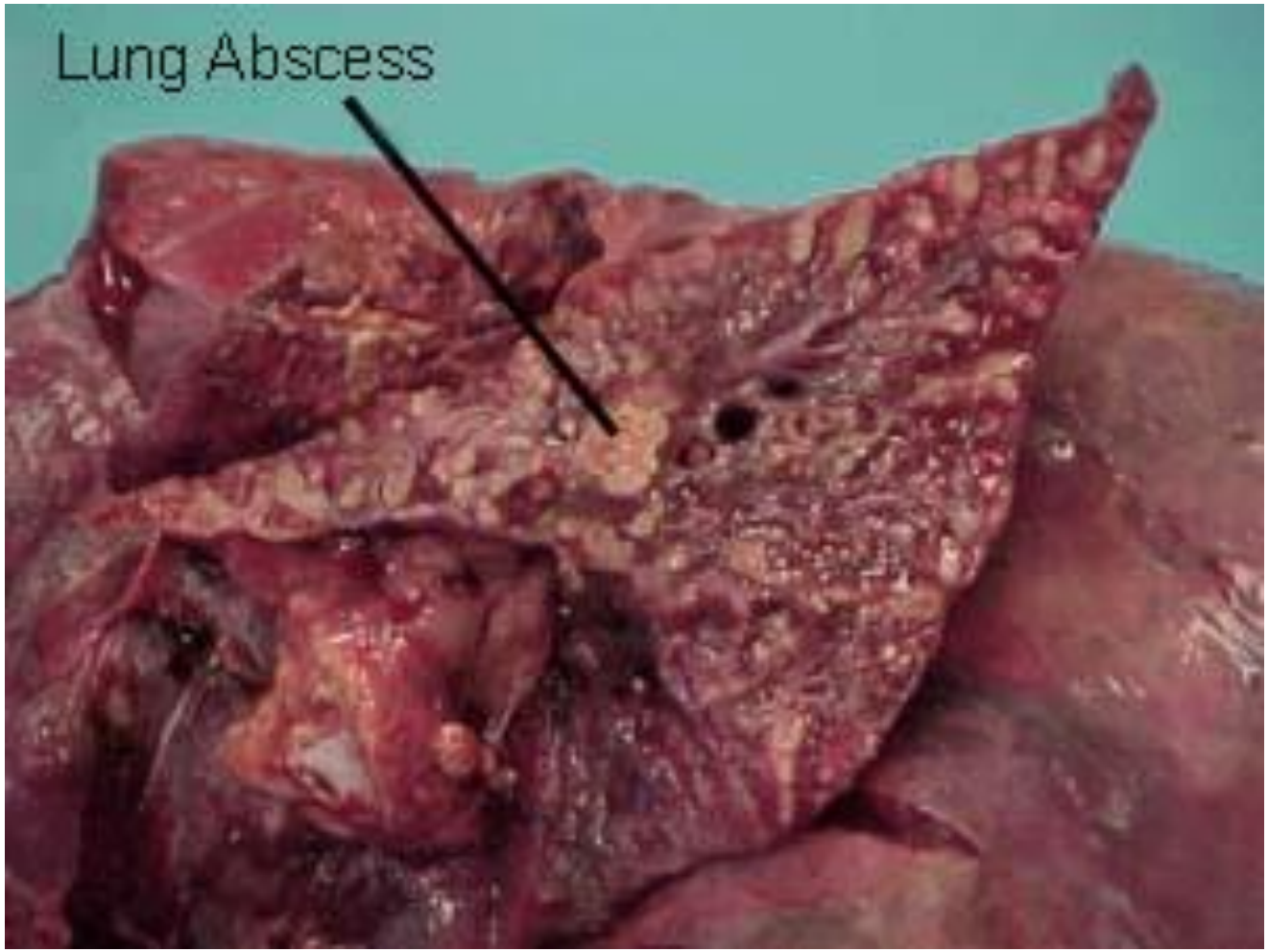
- Due to

  setting of aspiration (right lower lobe, single)

  secondary to pneumonia or bronchiectasis (basal, scattered, multiple)

  with septic emboli (any location, multiple)

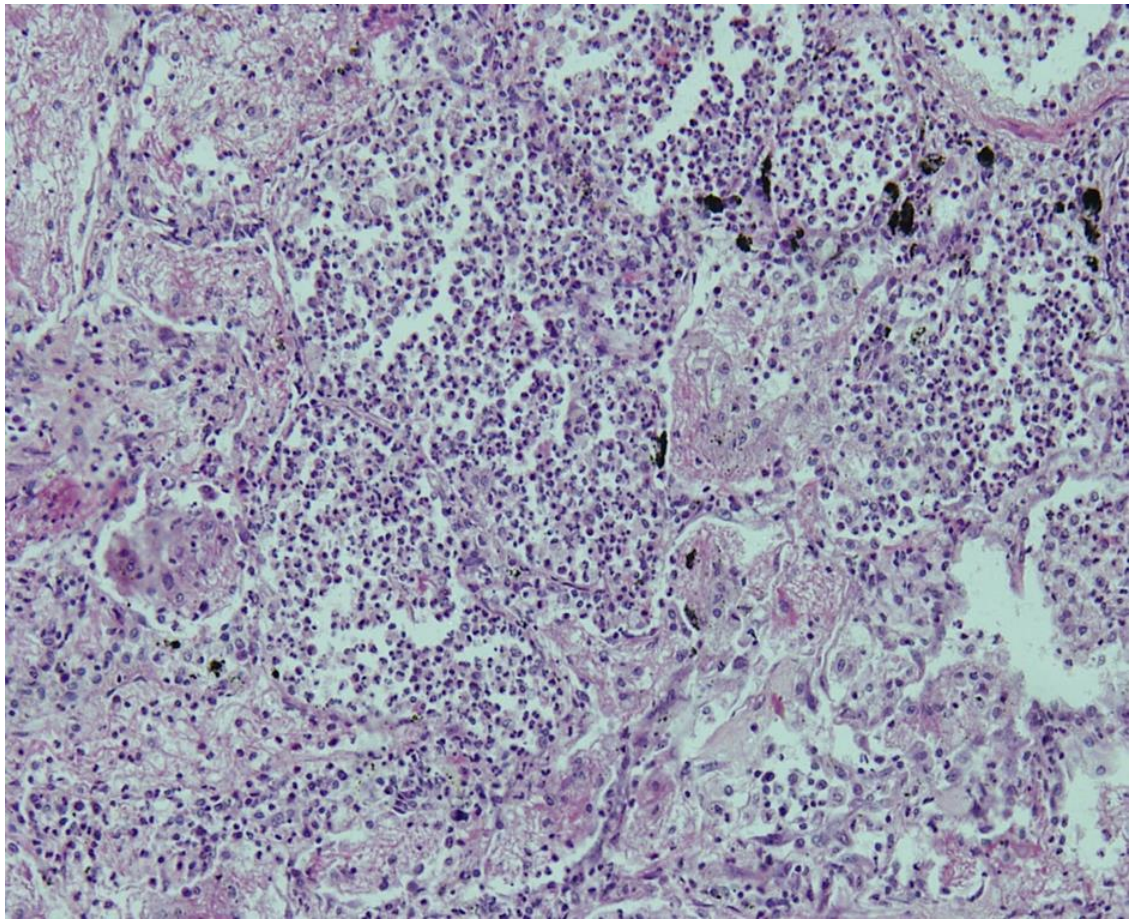
Suppurative destruction of the lung parenchyma with cavitation


Lung Abscess

Acute pneumonias microscopy

- Acute inflammation
- Edema
- Fibrinosuppurative exudate
- Organization, depending on the stage;

Alveolar neutrophil infiltration



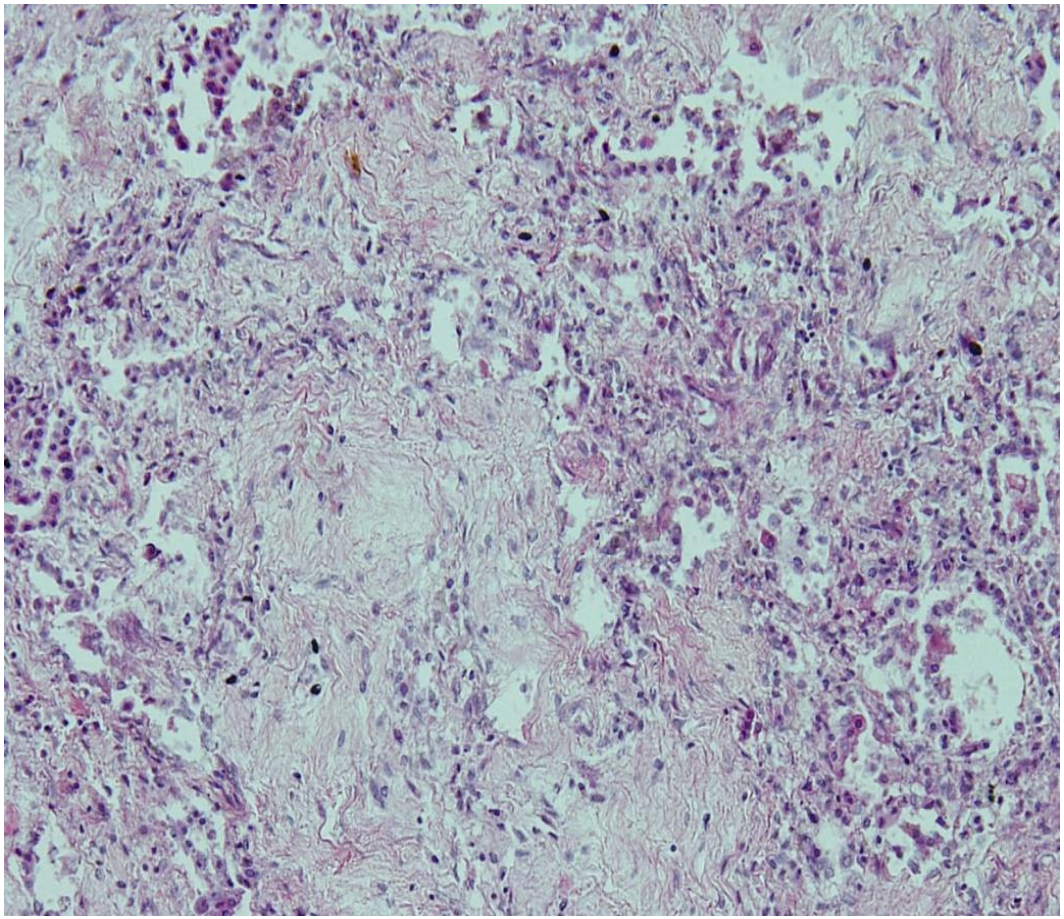
viral and atypical pneumonias microscopy

more interstitial edema

mononuclear inflammation confined to the alveolar walls

which appear widened and hypercellular; proteinaceous exudates

hyaline membranes can also be seen



- **Diffuse alveolar damage and fibrosis**

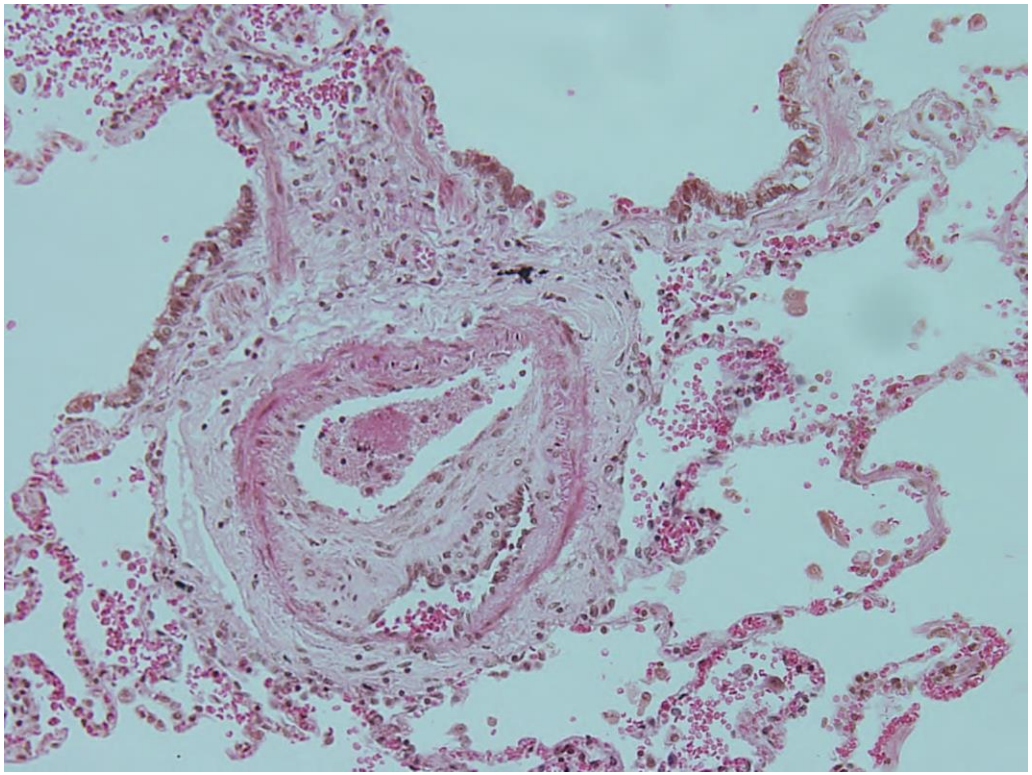
Pulmonary emboli microscopy

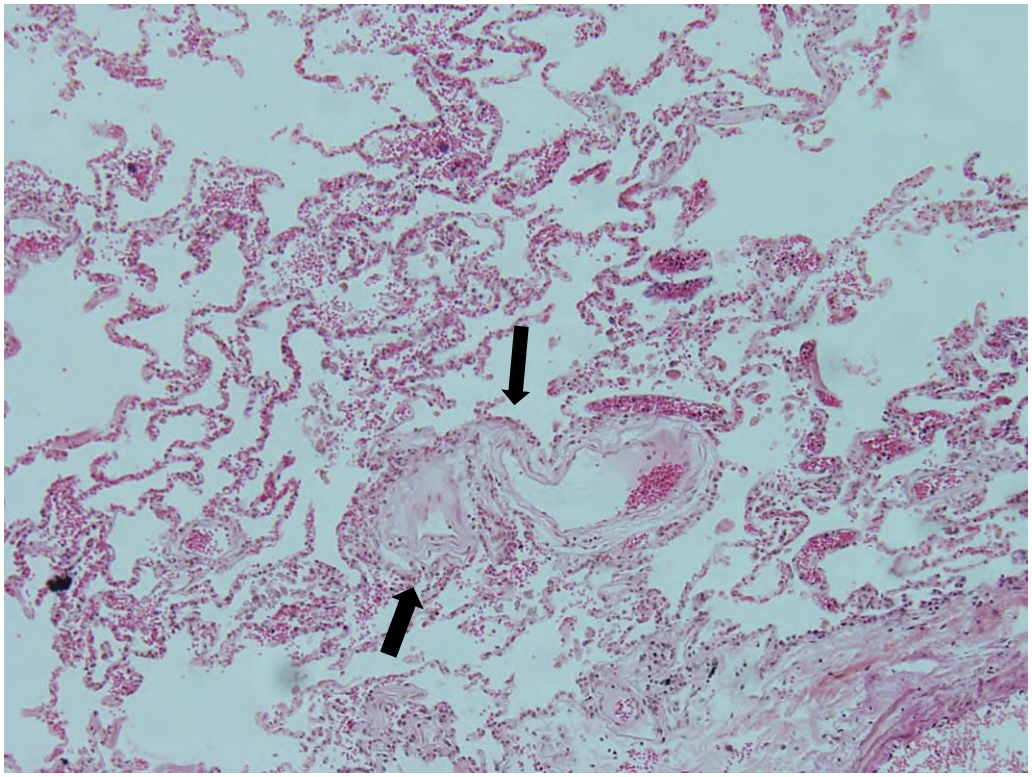
- Characteristic lines of Zahn
- Caused by separation and layering of
  - Platelets
  - Fibrin
  - and red cells in the clot

Pulmonary hypertension

- Arteries showing severe medial hypertrophy
- Complex lesions
  - Plexiform lesions
  - Angiomatoid lesion

Pulmonary hypertension





Central nervous system Conditions

Acute hemorrhage:

Atherosclerosis, hypertension, spontaneous rupture of aneurysm (eg, berry aneurysm), rupture of vascular malformation,

Thrombosis

Sagittal sinus thrombosis, cerebral amyloid angiopathy

Infection:

Leptomeningitis, encephalitis, abscess

Cerebral edema

RUPTURED BERRY ANEURYSM

- Common cause of sudden collapse and rapid death in young to middle-aged people
- Common in females
- Single or multiple
- Rupture due to sudden rise in blood pressure- exercise or emotion (death during sexual intercourse)
- Blood in base of brain
- Rapid death due to blood tracts down and compress brain stem

CEREBRAL HAEMORRHAGE, THROMOSIS AND INFARCTION

- Seen in old and with hypertension
- Haemorrhage often take place in external capsule of one hemisphere
- Rupture of lenticulo-striate artery
- Other places of bleeding-cerebellum, mid brain
- Cerebral thrombosis and infarction are causes of sudden deaths

Other CNS causes of sudden death

- epilepsy/seizure disorders
- multiple sclerosis,
- primary CNS tumors, or Chiari malformation
- neurodegenerative disorders.

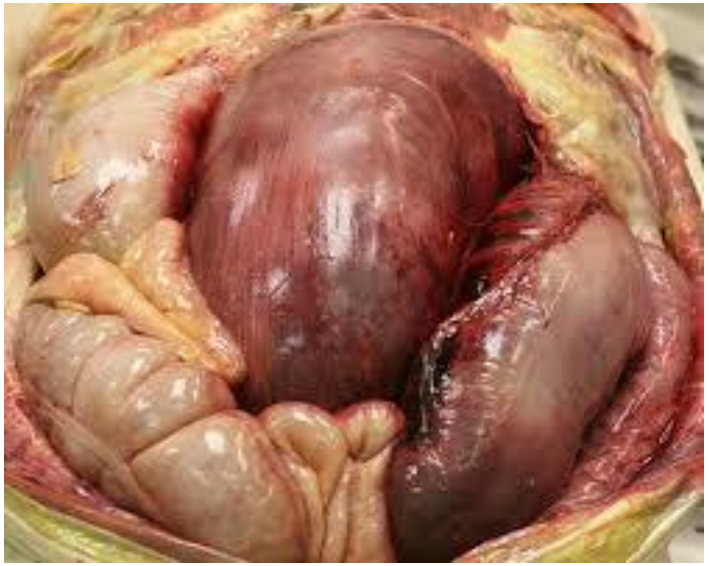
Gastrointestinal system

Massive gastrointestinal hemorrhage:

- Duodenal or gastric ulcers
- Esophageal varices
- Acute hemorrhagic gastritis
- Mallory-Weiss tears
- Boerhaave syndrome
- Vascular malformation
- Angiodysplasia
- Esophageal polyps

Obstruction (volvulus, intussusception, etc)

VOLVULUS



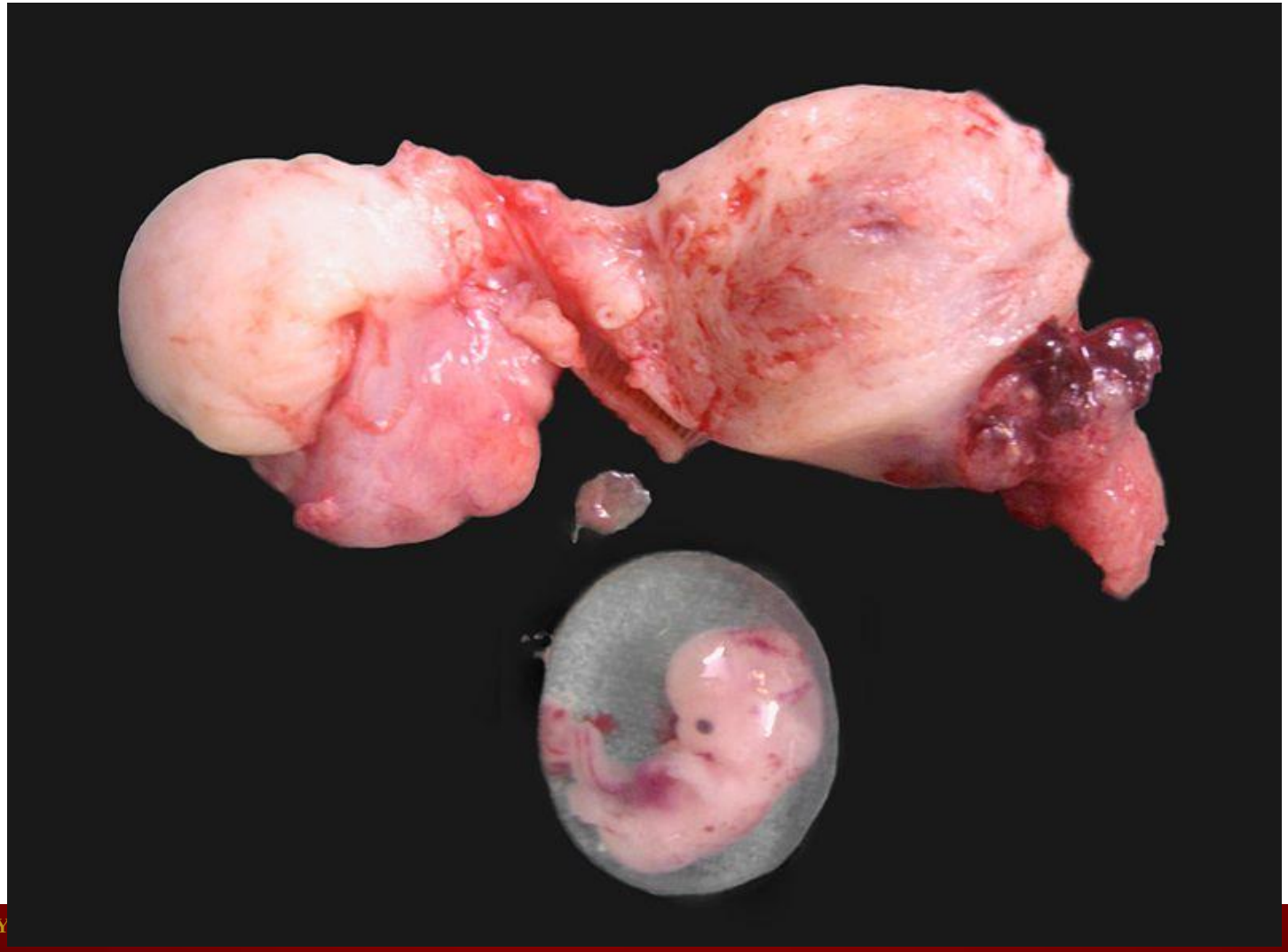
Other GI causes

- Acute pancreatitis
  - grossly identified by hemorrhage and necrosis
  - some may only be apparent microscopically
- Hepatic cirrhosis and steatosis
- In pregnancy,
  - both eclampsia and
  - acute fatty liver of pregnancy
  - subcapsular hepatic hematomas can be seen in eclampsia,

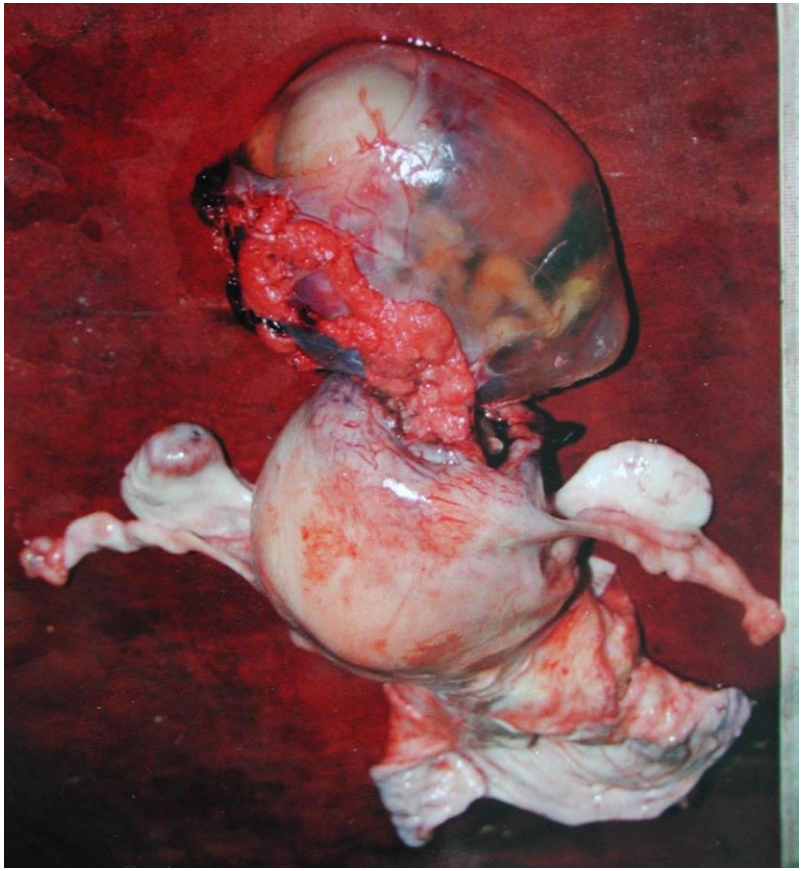
Miscellaneous

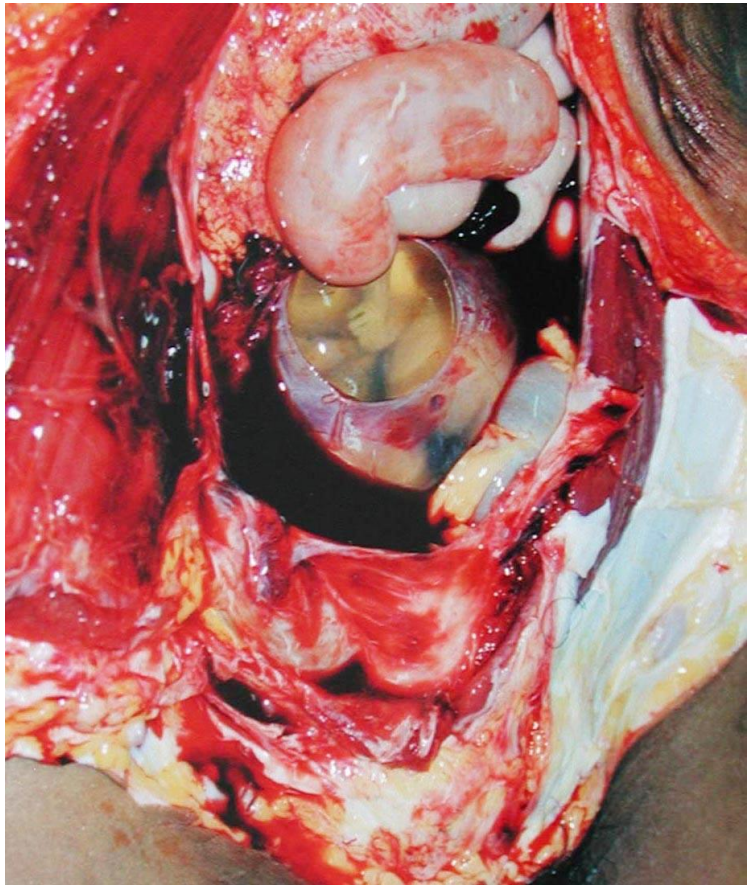
- Ruptured ectopic pregnancy
- Arterial fistula (aortoenteric, Aorto pulmonary fistula)

Ruptured ectopic pregnancy



Ruptured abdominal pregnancy





Summary

- Sudden natural death could result from cardiovascular, pulmonary, CNS conditions mainly
- Autopsy diagnosis needs meticulous autopsy, and ancillary testing
- Microscopy is the most useful ancillary testing
- There can be autopsy negative causes of sudden natural deaths


THANK YOU !