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05:38

**VENOUS Catheterization**

from femoral --> iliac --> IVC --> RA --> puncture through foramen ovale (which consists of thin membranous tissue)

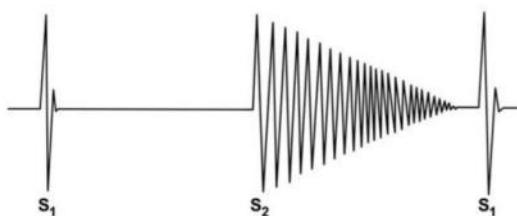
to :-

1. directly measure LA Pressure (instead of estimating with PCWP)

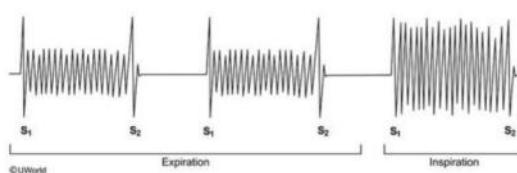
2. for radiofrequency ablation of the arrhythmogenic foci in the LA/ near the ostia of the Pul Veins.

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05:39

**Pulmonary regurgitation****UWORLD IMAGES**

05:39

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05:39

holosystolic murmur --> MR,TR,VSD

increased with respiration --> (think right sided) hence, TR

murmur which increases in intensity with inspiration (carvallo sign)

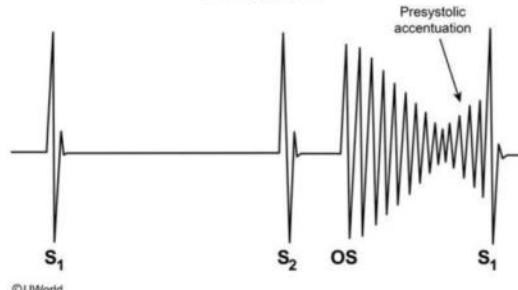
heard best at the Left 2nd and 3rd ICS/ left sternal border

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Mitral stenosis



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PSA is present ---> good (i.e. no AF), mild MS

no PSA --> bad, due to AF, severe MS.

S2-OS interval -

Long – mild MS

Short – sever MS

best in Left Lateral Decubitus @ the Apex.

h/o RHD (pt. from developing country)

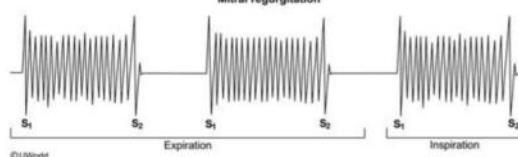
concomitant Aortic Valve insufficiency +-

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Mitral regurgitation



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holosystolic murmur not affected by respiration.

DD :-

MR

TR

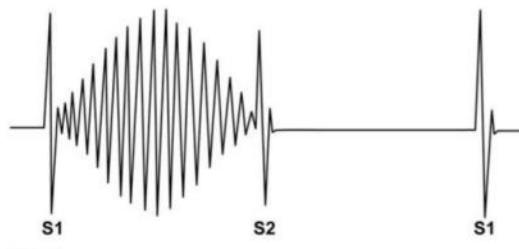
VSD

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05:42

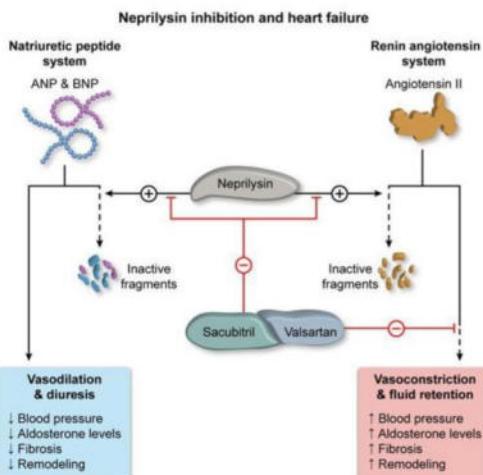
### Aortic stenosis



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note the irregular R-R Interval + absence of P-Waves

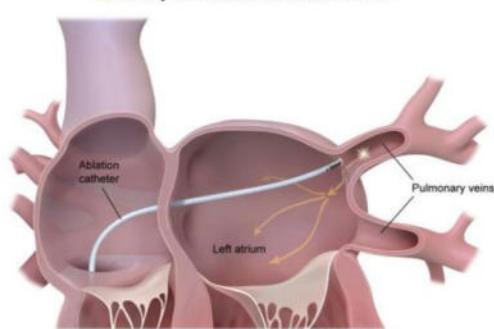
hence, A. Fib. (for flutter presence of saw-tooth pattern must)

most commonly from the Pul Vein Ostia in LA (aberrant electrical discharge)

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#### Pulmonary vein ablation for atrial fibrillation



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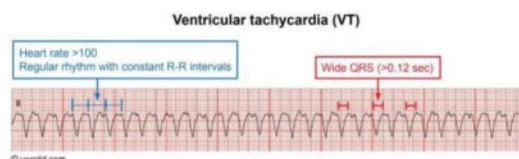
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05:45



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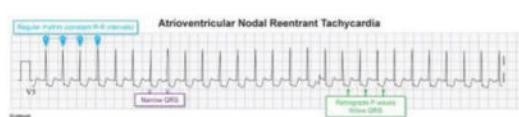
05:46

Monomorphic VT (most commonly originates) from areas of Ischemic Myocardium;

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05:55



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05:56

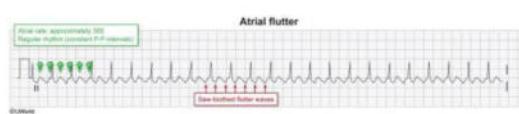
polymorphic nodal pathway i.e presence of dual (fast + slow) pathway within or near AV NODE

hence, AVNRT

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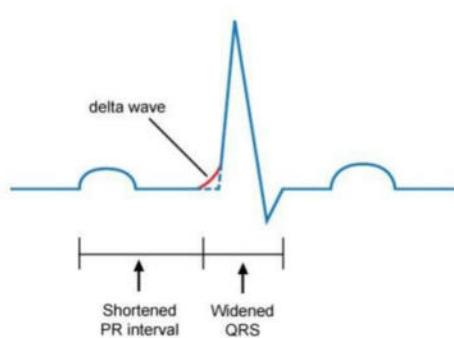


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**Wolff-Parkinson-White triad**



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WPW Syndrome can be

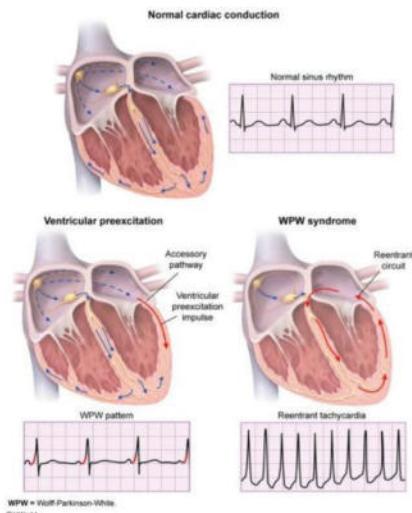
asymptomatic – normal sinus rhythm with these 3 changes

or

symptomatic – AVRT (palpitations and racing heart) ; absence of normal sinus rhythm

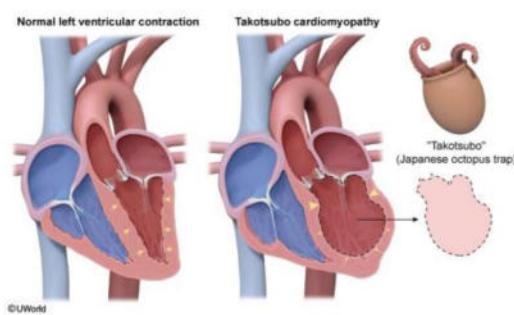
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### UWORLD IMAGES

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hypokinesia of apical and anterior walls of the LV

+

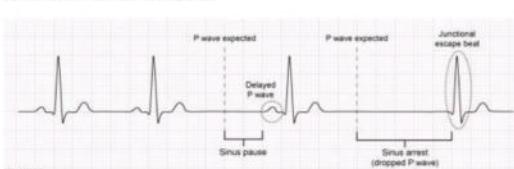
hyperkinesia of the BASAL wall of the LV

due to surge of catecholamines causing  
either direct myocardial dysfunction  
or  
microvascular spasm --> ischemia and myocardial stunning.

in the absence of Coronary obstruction (on angiography).

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seen in Sick Sinus Syndrome.

in elderly due to age related degeneration of SA Node.

hence, pacemaker function taken over by AV Node.

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1. delayed P- waves (sinus pause)

2. prolonged delayed/dropped P-wave (sinus arrest)

usually followed by

3. narrowed QRS complex (junctional escape beat)

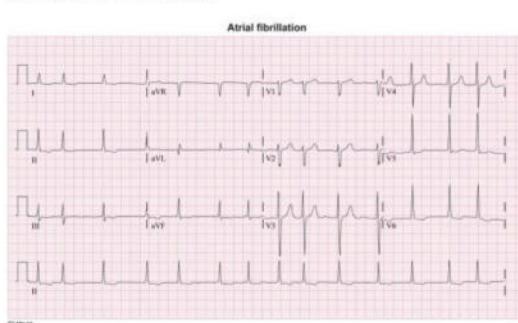
due to age related degeneration of SA Node --> Sick Sinus Syndrome  
hence, Pacemaker role is taken over by AV Node (hence, bradycardia).

pt. will be elderly presenting with  
dyspnea, fatigue, lightheadedness, pre-syncope, syncope.

occasionally, pt. may have brief periods of Atrial Tachycardia (A. Fib.)  
alternating with Bradycardia

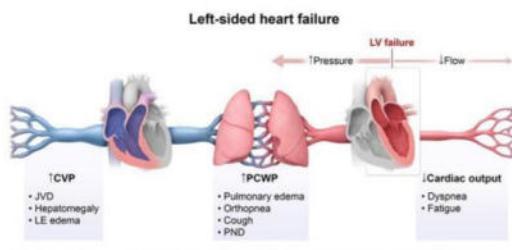
## UWORLD IMAGES

05:58



## UWORLD IMAGES

05:58



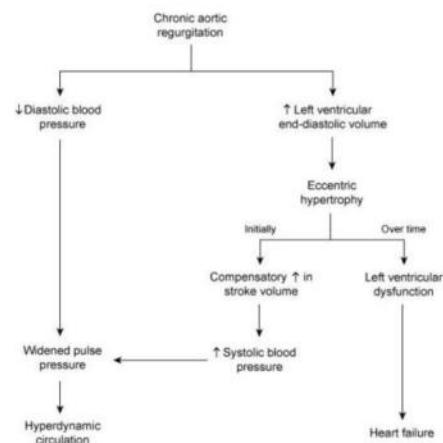
CVP = central venous pressure; JVD = jugular venous distension; LE = lower extremity; LV = left ventricular; PCWP = pulmonary capillary wedge pressure; PND = paroxysmal nocturnal dyspnea.

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### Pathophysiology of chronic aortic regurgitation

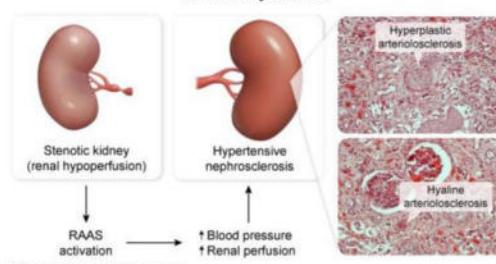


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### Renal artery stenosis



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STENOTIC KIDNEY --> renal parenchymal ischemia which manifests as :-

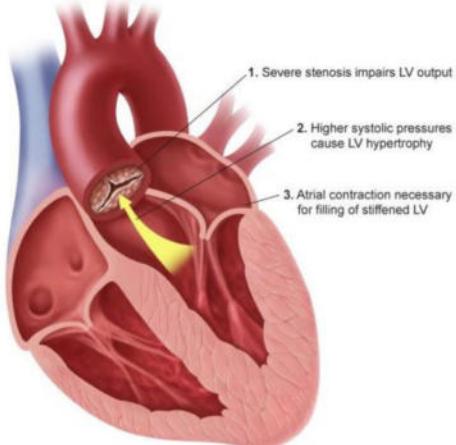
diffuse cortical thinning,  
tubular atrophy,  
interstitial fibrosis,  
small crowded glomeruli.

NON-STENOTIC KIDNEY --> effects of HTNsive nephrosclerosis

arteriolar wall thickening

usually caused in elderly due to atherosclerosis.

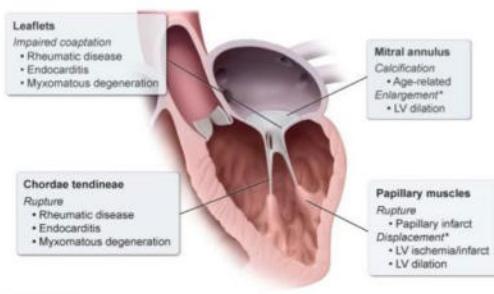
## Hemodynamics of severe aortic stenosis



**Atrial fibrillation:** Loss of contraction reduces LV filling, causing hypotension & pulmonary edema

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## Causes of mitral regurgitation



LV = left ventricular

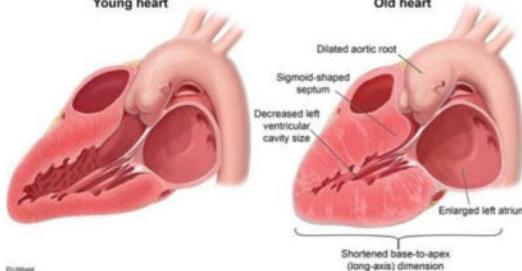
\*Secondary cause of mitral regurgitation.

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## Cardiac changes associated with normal aging

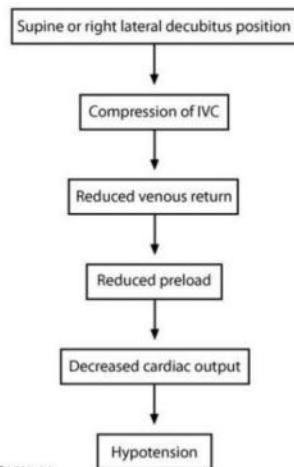
Young heart

Old heart



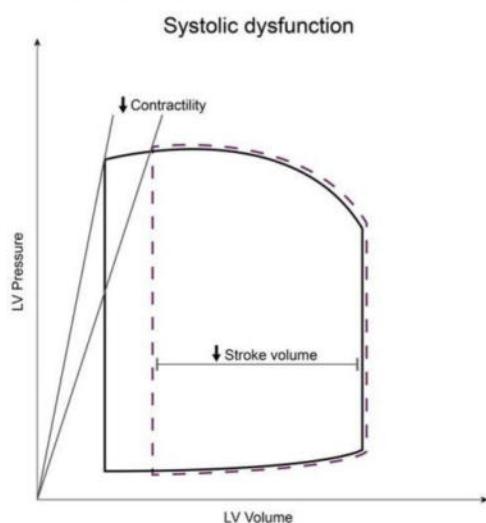
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### Supine hypotension syndrome



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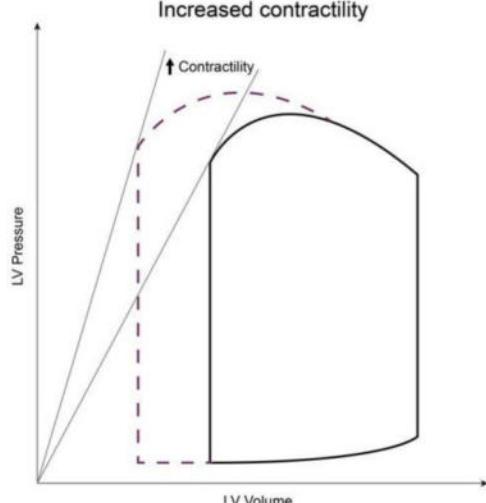
06:00



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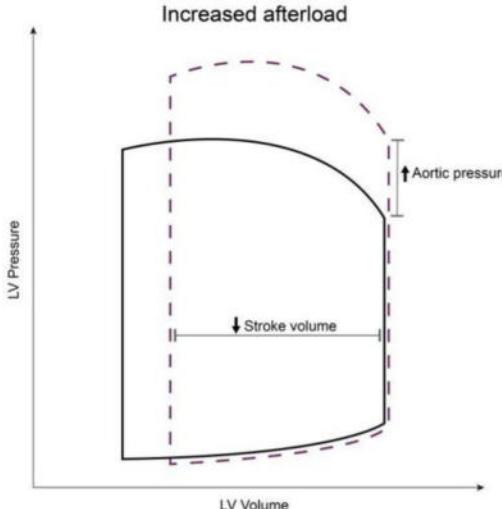
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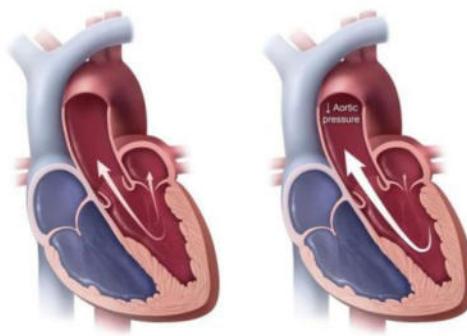
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06:00

Effect of blood pressure reduction on mitral regurgitation



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06:00

in MR :-

two directions of blood flow

FORWARD – main contributory factor is PVR/afterload

BACKWARD / REGURGITANT –

LA compliance + mitral valve orifice cross-section during systole.

to increase the forward flow --> reduce afterload

(eg :- nitroprusside, hydralazine, minoxidil)

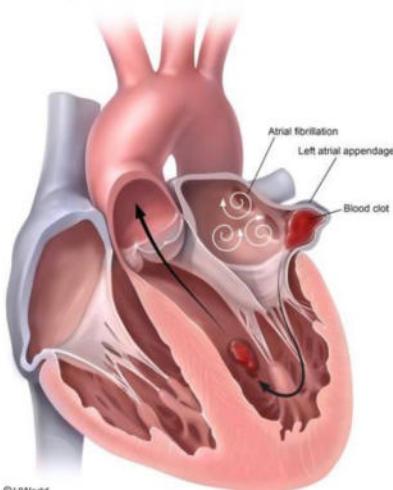
this is beneficial in acute decompensated heart failure to reduce pulmonary congestion.

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## Atrial fibrillation with thrombus formation



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Sources of potential Thrombus + Risk factors :-

in AF --> Left Atrial appendage

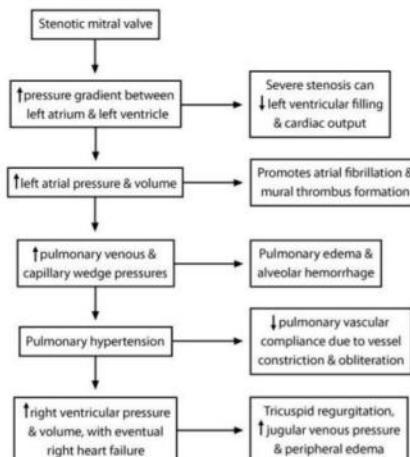
in cases of prior MI --> akinesia of the affected portion of Ventricular wall --> LV Apex (may be a possible source)

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06:01

Hemodynamic consequences of mitral stenosis

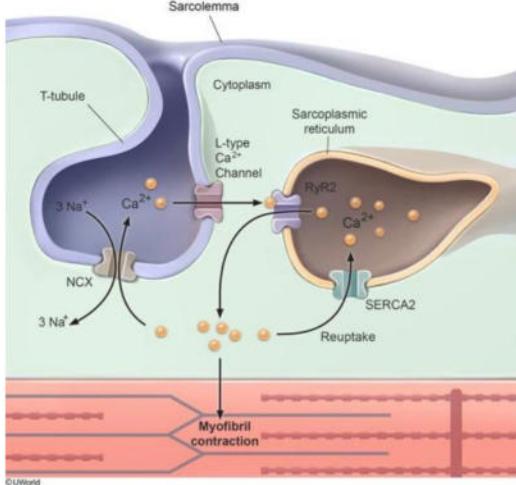


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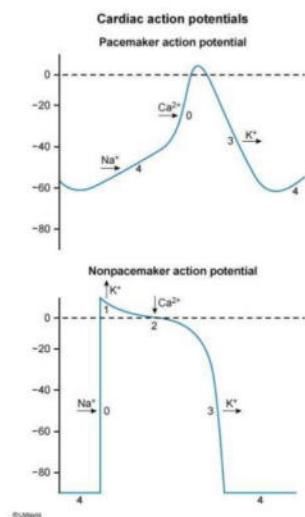
## Intracellular calcium regulation



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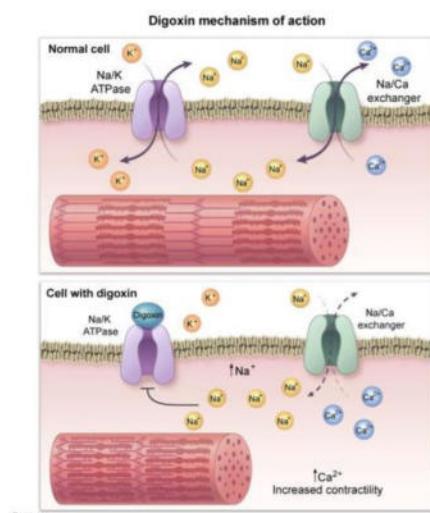
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06:01

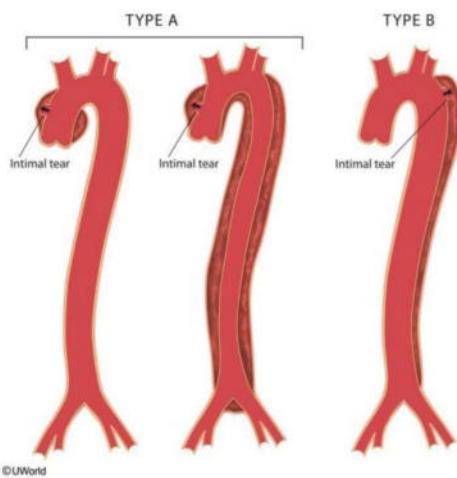


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06:02

## DISSECTING AORTIC HEMATOMAS



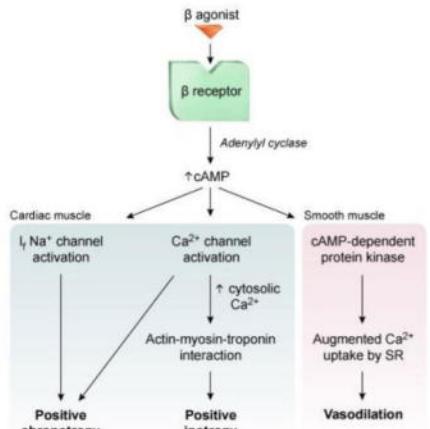
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### Dobutamine pharmacology



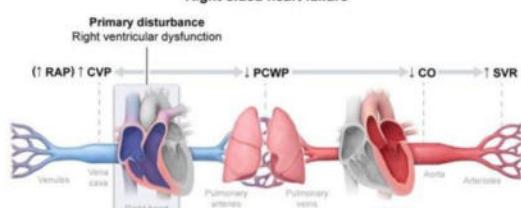
SR = sarcoplasmic reticulum.  
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### Right-sided heart failure

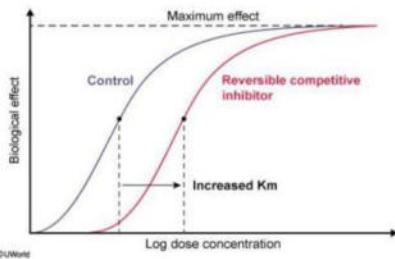
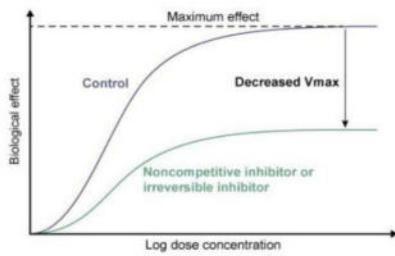


CO = cardiac output; CVP = central venous pressure;  
PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure; SVR = systemic vascular resistance.  
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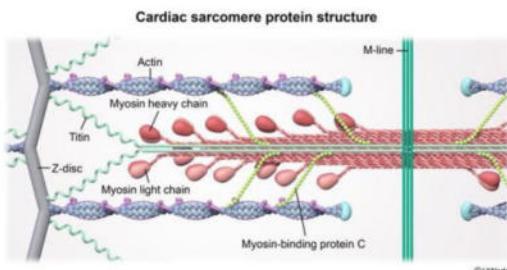
06:04



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## UWORLD IMAGES

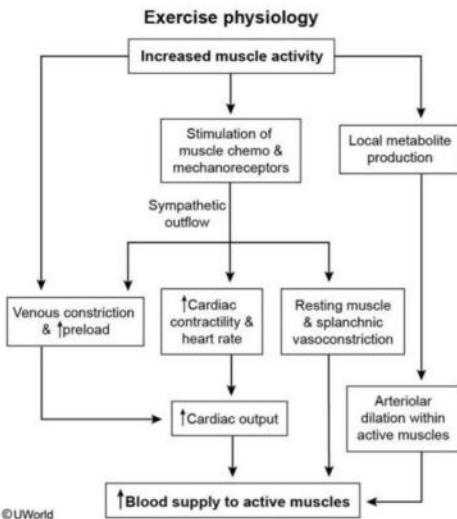
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## UWORLD IMAGES

06:04

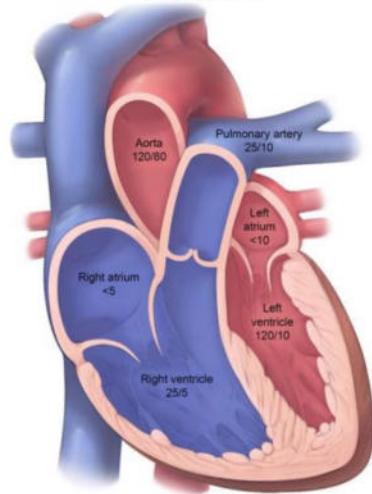


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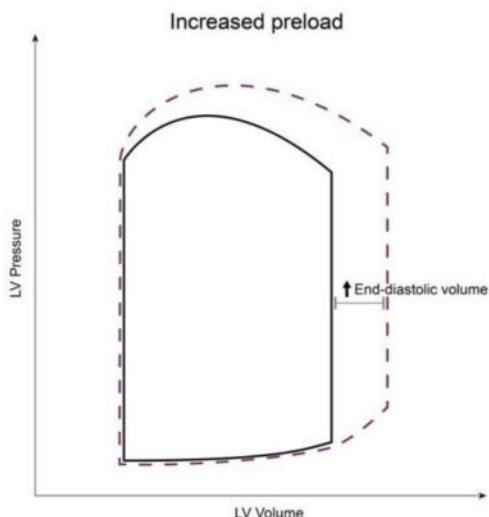
06:04

### Cardiac pressures



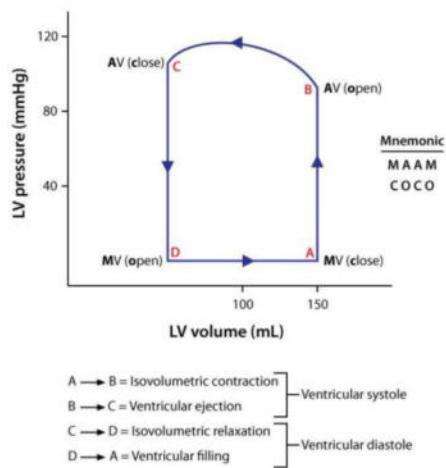
### UWORLD IMAGES

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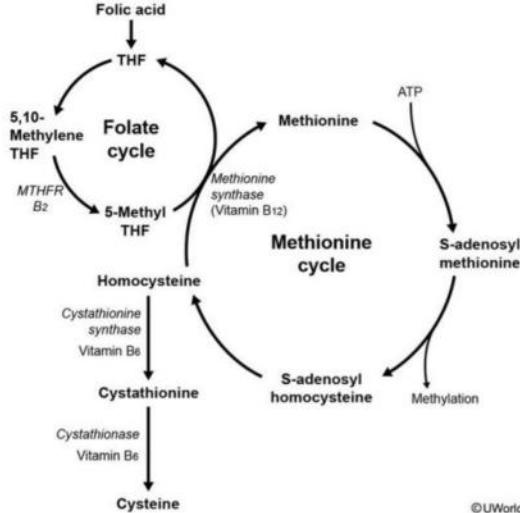
06:04



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## UWORLD IMAGES

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MTHFR gene mutation --> hyperhomocysteinemia -->

Ishemic Stroke

CAD

Venous thrombosis.

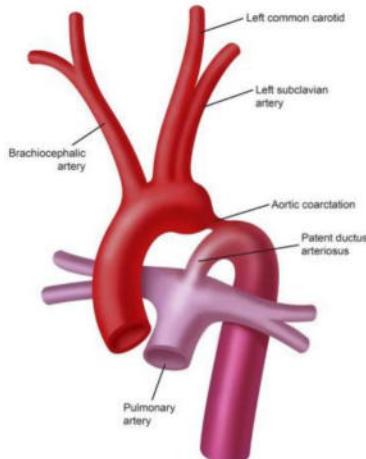
as elevated homocysteine levels leads to induction of endothelial damage.

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Aortic coarctation  
Patent ductus



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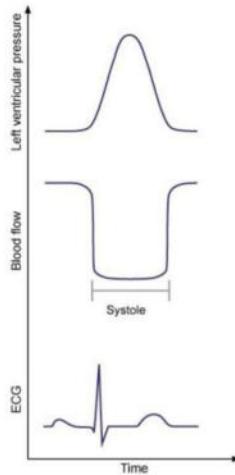
06:05

associated with

1. bicuspid aortic valve --> early (<50) onset calcification --> AS
2. turner's syndrome
3. berry's aneurysm --> spontaneous ICH in young

pulse discrepancies

BP discrepancies



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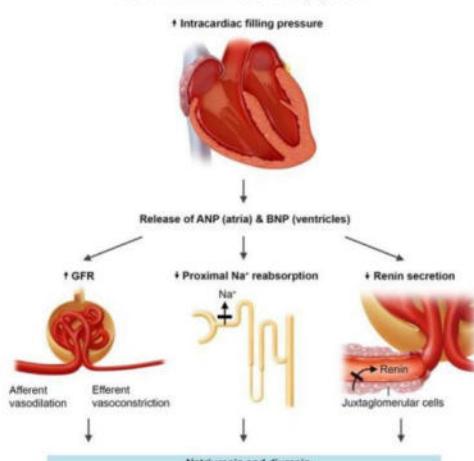
During Ventricular Systole, the coronary vessels supplying the LV are compressed by the surrounding muscle.

Hence, vast majority of blood flow occurs during diastole.

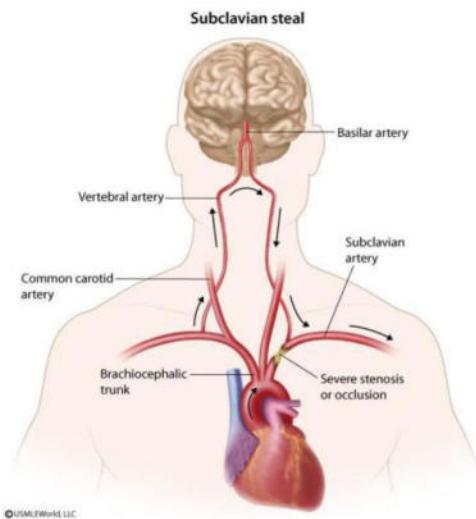
In RV, the systolic Pressure is much lower compared to LV (25 mmHg compared to 120 mmHg). Hence, coronary perfusion pressure is able to overcome the RV wall pressure throughout the cardiac cycle.

there is relatively constant blood flow to the RV myocardium.

#### Renal effects of natriuretic peptides



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06:06

Pt. will have the following s/s

1. due to Vertebro-basilar insufficiency – dizziness, vertigo, syncope.
2. arm ischemia – which may be symptomatic only on exertion, like (fatigue, pain, paresthesia)

Physical examination findings :-

difference between the SBP in two upper extremities by >15 mmHg.

obstruction proximal to the origin of the vertebral artery.

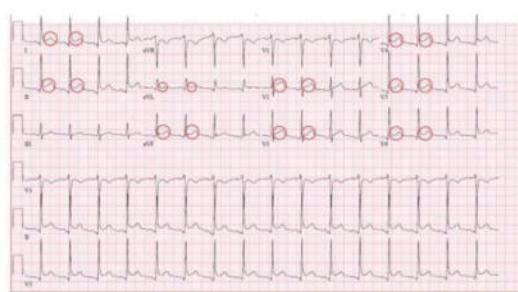
rare causes :-

Takayasu Arteritis

complication from heart surgery (repair of aortic coarctation)

## UWORLD IMAGES

06:06



## UWORLD IMAGES

06:06

Diffuse ST segment elevation --> due to inflammation of the ventricular myocardium.

seen in Pericardial Effusion.

most commonly due to

## Viral Infections

(assume if there is no significant medical history provided in the vignette)

other etiologies :-

RA, SLE (auto immune disorders)

Post-MI (peri-infarction or delayed i.e. dressler's syndrome)

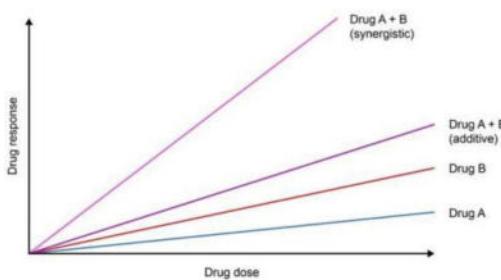
Para-Pneumonic (due to staph, strep)

pt. will be severely ill with fever and sepsis S/S

## UWORLD IMAGES

06:08

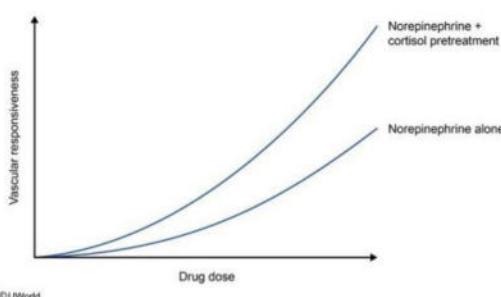
Differences between synergistic and additive response



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## UWORLD IMAGES

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## UWORLD IMAGES

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graph depicting

PERMISSIVE EFFECT of cortisol on Vessel responsiveness.

meaning

cortisol itself doesn't have any vasoconstrictive property.

But it upregulates (alpha 1 receptors on BV),

hence, increasing the action of Ang II and Norepinephrine.

## ADDITIVE EFFECT

combined effect of 1 + 2 = effect of 1 (alone) + effect of 2 (alone)

## SYNERGISTIC EFFECT

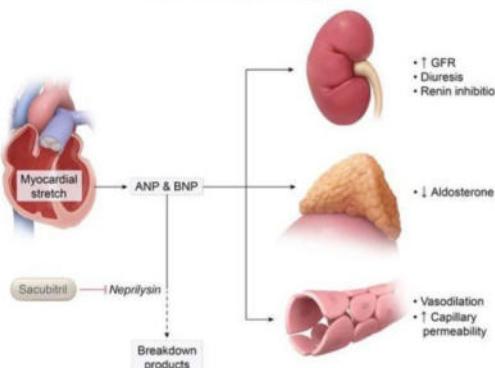
combined effect of 1 + 2 > individual effects of drugs.

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### UWORLD IMAGES

06:08

#### Effects of the natriuretic peptides

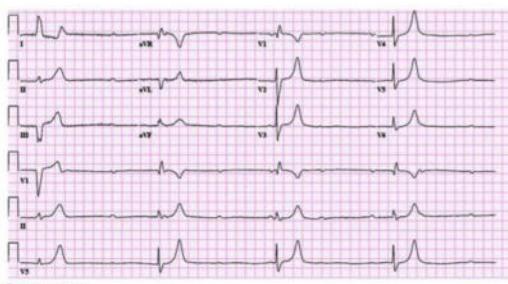


ANP = atrial natriuretic peptide; BNP = brain natriuretic peptide; GFR = glomerular filtration rate.  
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06:08



### UWORLD IMAGES

06:08

3rd degree AV Block

"atrio-ventricular dissociation"

(P waves and QRS complex beating independently of each other)

regular R-R interval (in lead 2)

regular P waves (in lead 2)

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06:09

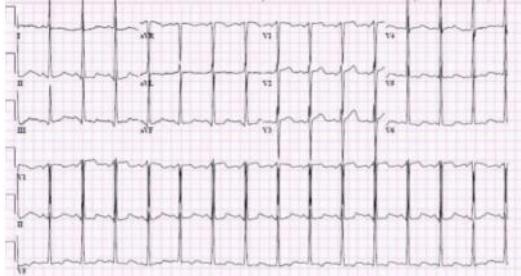
#### Complete AV block



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high voltage QRS complexes,

esp prominent in the :-

"S wave of V1 and V2"

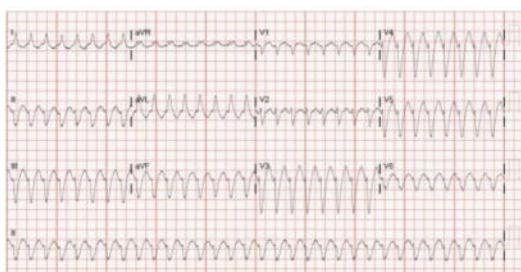
and the

"R wave of V5 and V6"

due to concentric LV hypertrophy

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06:10



### UWORLD IMAGES

06:10

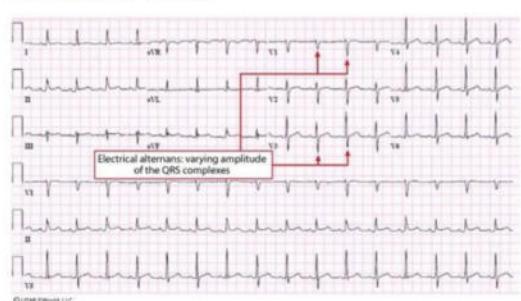
"Wide QRS complexes that are rapid and regular"

due to ventricular tachycardia.

may be triggered by ischemic and infarcted ventricular myocardium.

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06:10



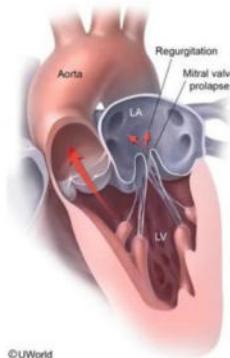
### UWORLD IMAGES

06:10

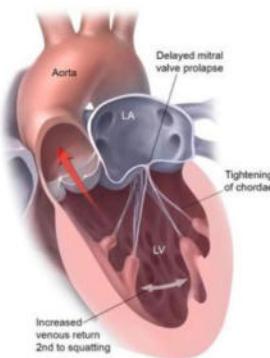
Cardiac tamponade

## Mitral valve prolapse

Standing



Squatting



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## Cardiac tissue conduction velocity

**\*Fastest\***

Purkinje system

↓ Atrial muscle

↓ Ventricular muscle

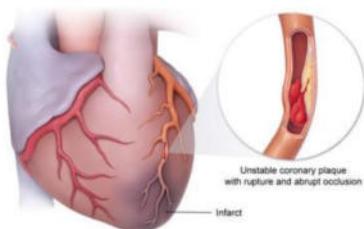
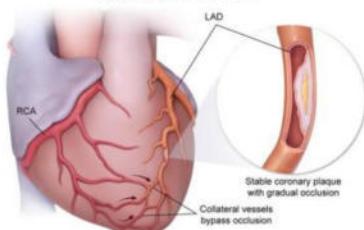
↓ AV node

**\*Slowest\***

Mnemonic: "Park At Venture Avenue"

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## Coronary collateral circulation



LAD = left anterior descending artery; RCA = right coronary artery.

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characteristics of an unstable plaque :-

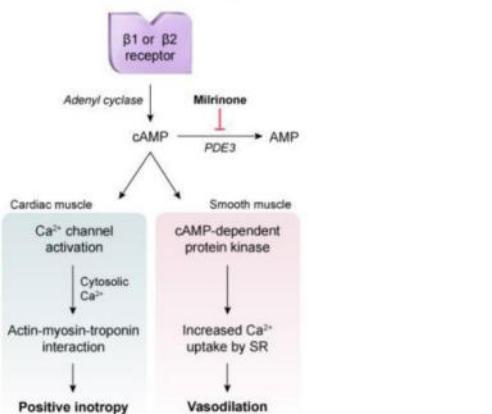
1. active inflammation – active macrophages that secrete MMP and

- degrade collage,  
2. high core concentration of lipid  
3. thin fibrous cap.

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### Milrinone pharmacology

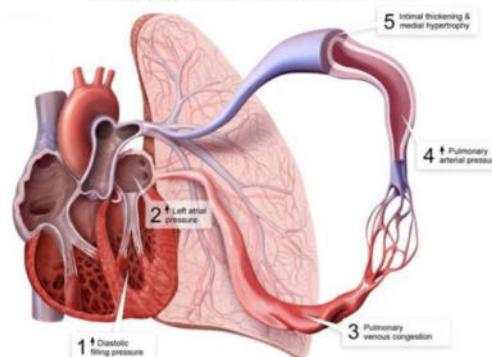


cAMP = cyclic adenosine monophosphate.  
PDE-3 = phosphodiesterase-3; SR = sarcoplasmic reticulum.  
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### Pulmonary hypertension due to left-sided heart failure



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non-enhancing septum dividing the lumen of the ascending and descending aorta.

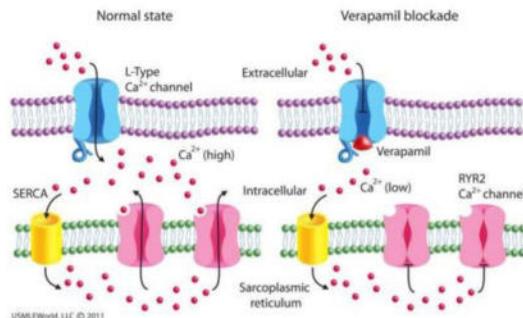
"septum" is actually the tunica intima of the aorta which has been torn from the remainder of the aorta wall.

HTN is the single most important risk factor for aortic dissection.

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Effect of Verapamil on smooth muscle cell  
(through luminal  $\text{Ca}^{2+}$ )



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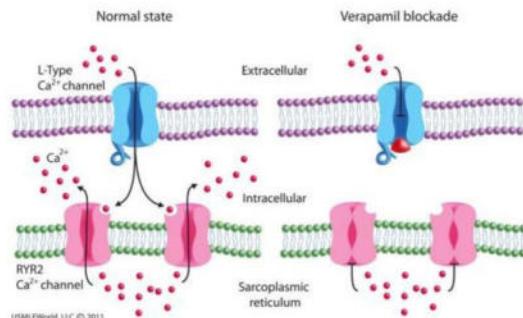
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Ca induced ca release

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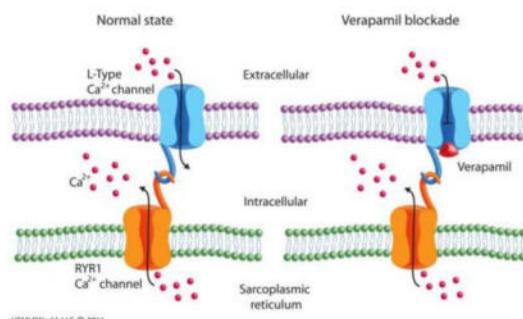
Effect of Verapamil on cardiac muscle cell  
( $\text{Ca}^{2+}$  induced release) (CICR)



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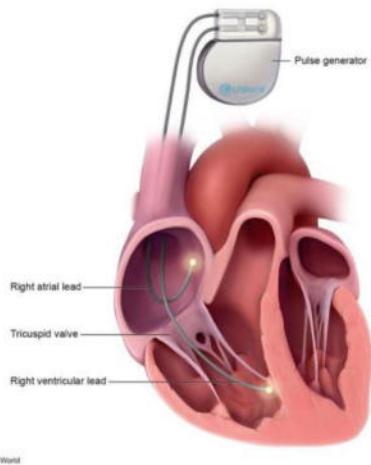
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Effect of Verapamil on skeletal muscle cell  
(mechanical coupling)



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permanent Pacemaker implantation --&gt;

can damage valve leaflets / impaired coaptation of Tricuspid Valve--&gt;

severe TR --&gt;

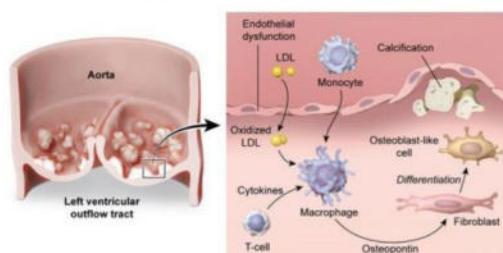
Right Sided HF.

(absence of Pulmonary edema --&gt; rules out Left HF)

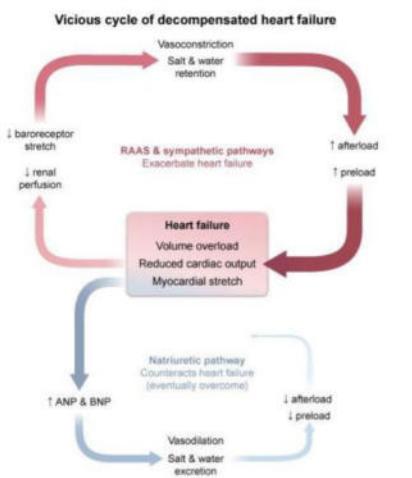
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## Pathogenesis of calcific aortic valve disease

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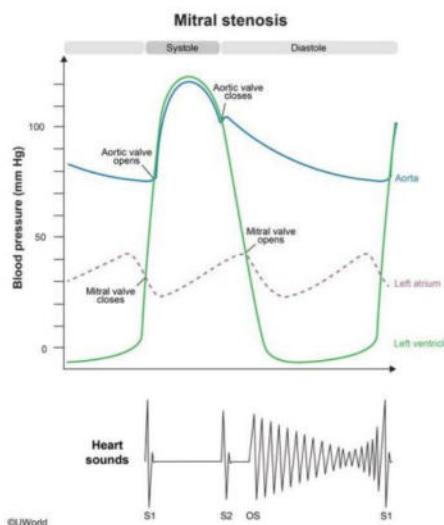
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ANP = atrial natriuretic peptide; BNP = brain natriuretic peptide; RAAS = renin-angiotensin-aldosterone system.  
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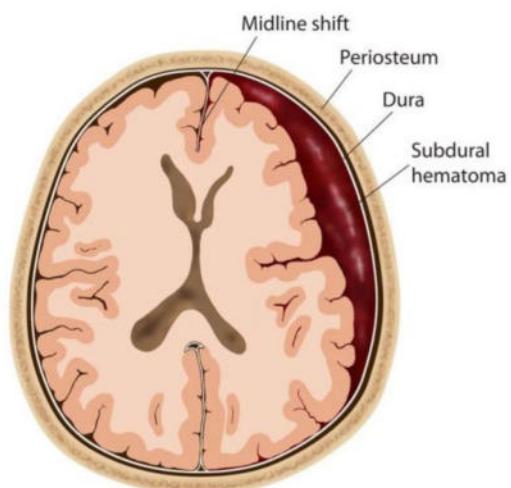
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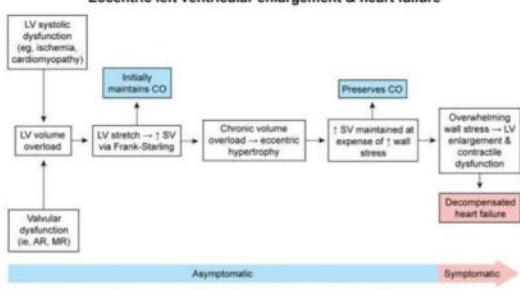


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### Eccentric left ventricular enlargement & heart failure

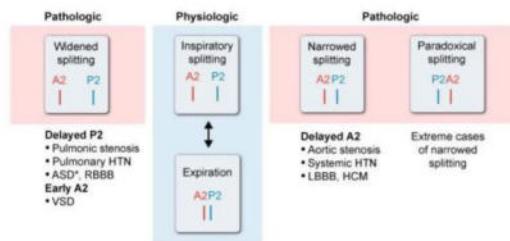


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### Splitting of S2



\*Widened splitting is fixed throughout respiratory cycle.

ASD = atrial septal defect; HCM = hypertrophic cardiomyopathy; HTN = hypertension; LBBB = left bundle-branch block;

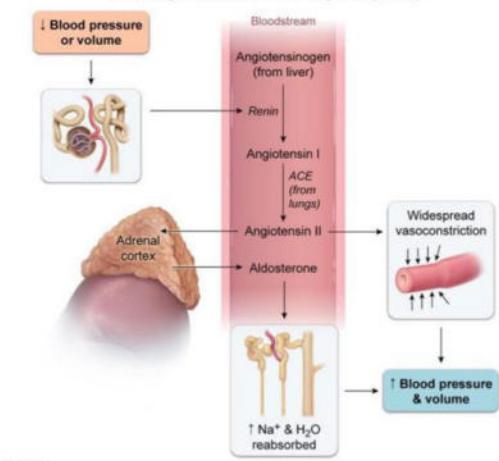
RBBB = right bundle-branch block; VSD = ventricular septal defect.

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### Renin-angiotensin-aldosterone system (RAAS)



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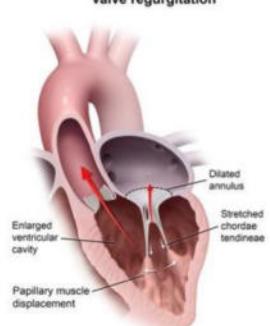
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### Normal



### Functional mitral valve regurgitation



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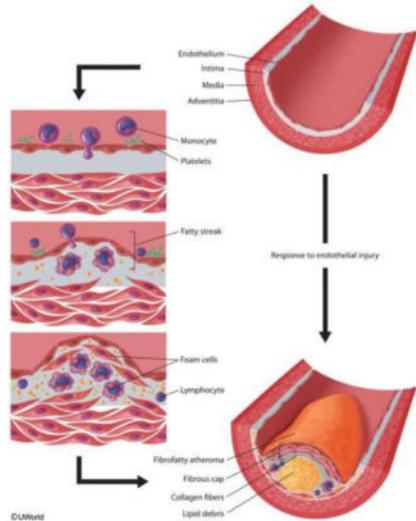
meaning temporary MR murmur (holosystolic).

due to decompensated HF --> increased LV EDV --> shown changes.

treatment with diuretics, vasodilators --> reduce the LV EDV and resolution of the MR murmur.

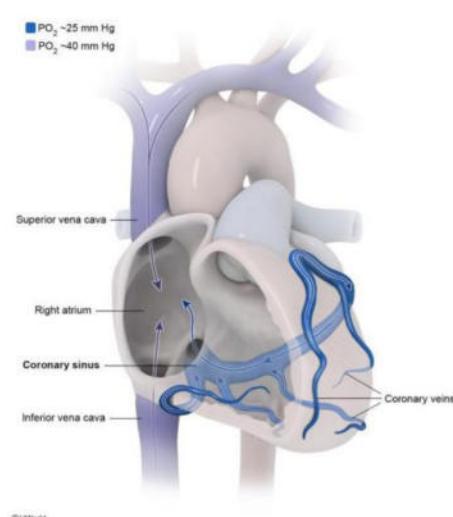
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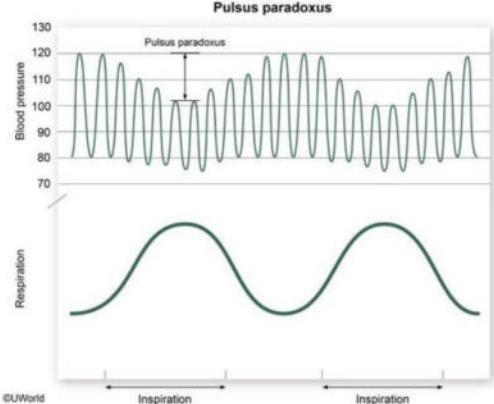
## UWORLD IMAGES

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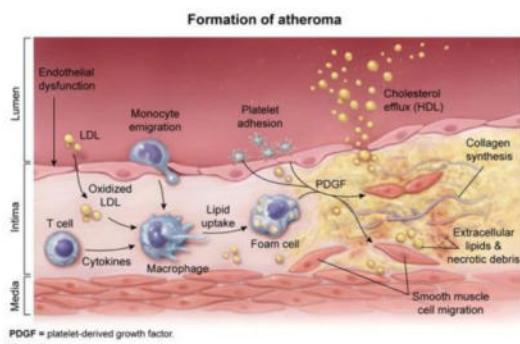
## UWORLD IMAGES

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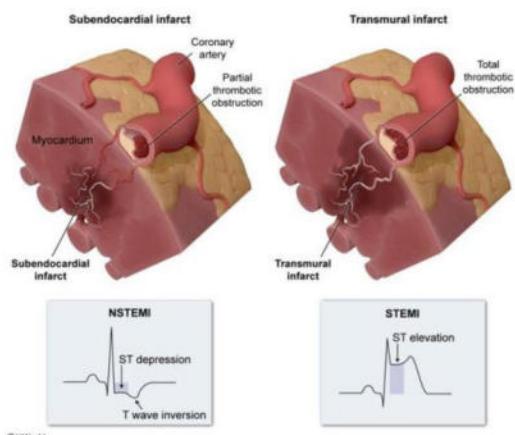
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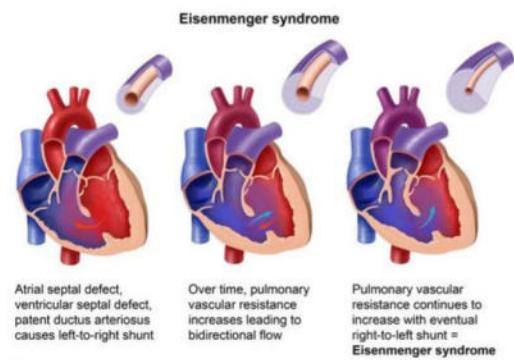
## UWORLD IMAGES

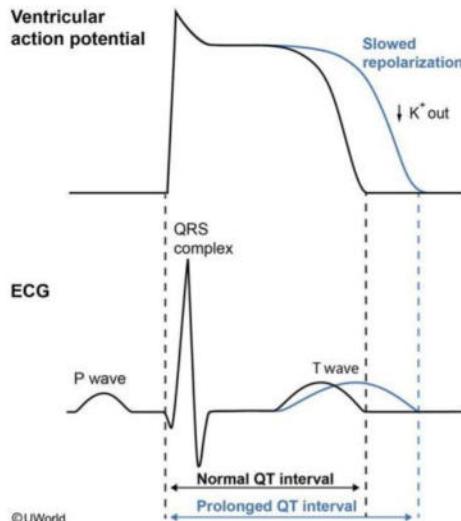
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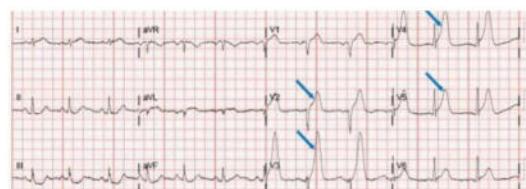
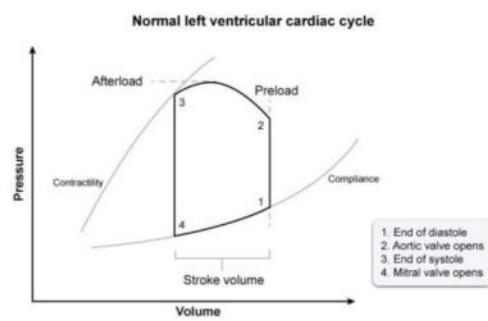




genetic defect in a K<sup>+</sup> channel protein --> slow outward K<sup>+</sup> current

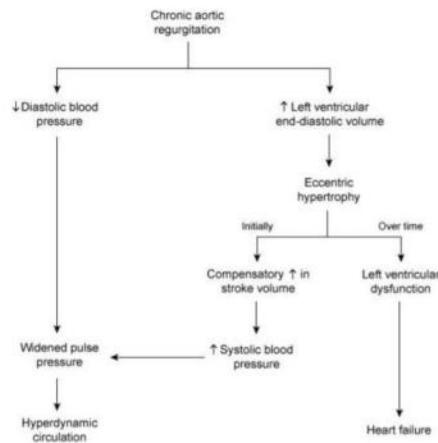
--> congenital long QT syndrome

pt. is predisposed to life threatening Ventricular arrhythmias.  
(torsades de pointes)



ST- elevation in V2, V3, V4, V5 --> Anterior Wall MI (thrombus in LAD).

## Pathophysiology of chronic aortic regurgitation



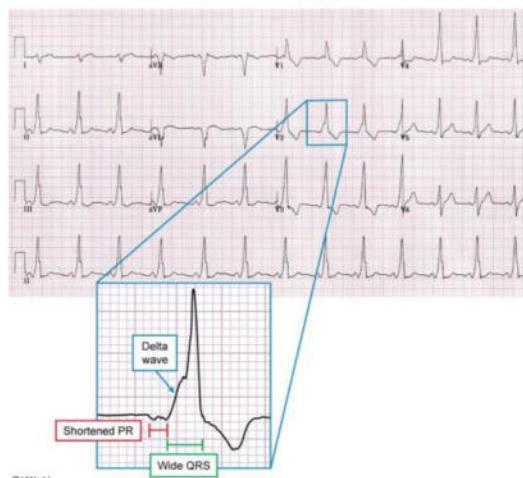
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Wolff-Parkinson-White pattern



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due to the presence of an accessory pathway (which bypasses the normal SLOW AV node).

hence, the ECG changes.

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note :-

excess of P-waves + regular Rate + flutter waves (saw-tooth pattern).

these changes are seen in "Atrial Flutter"

usually caused by a large re-entrant circuit that traverses the CavoTricuspid isthmus.

(area b/w the tricuspid annulus and the IVC in the RA)

atrial rate = 300/min

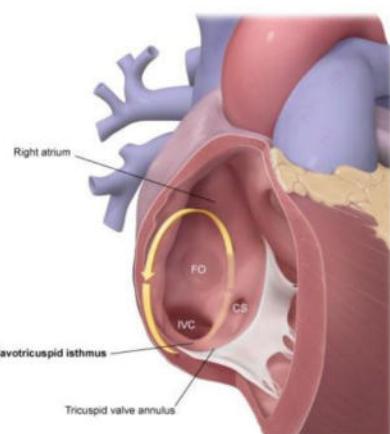
ventricular rate = 150/min, 100/min, 75/min due to 2:1, 3:1, 4:1 conduction

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Reentrant circuit in atrial flutter



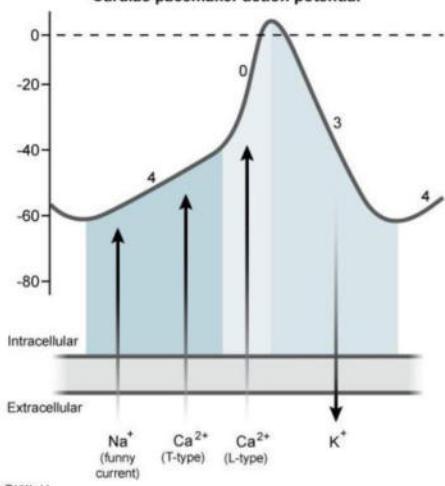
CS = coronary sinus, FO = fossa ovalis, IVC = inferior vena cava.  
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Cardiac pacemaker action potential



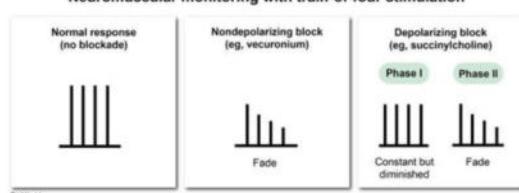
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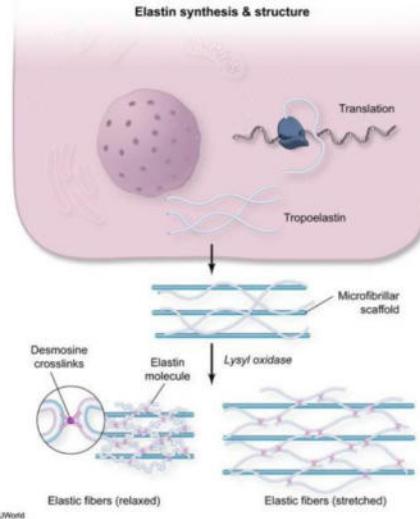
Neuromuscular monitoring with train-of-four stimulation



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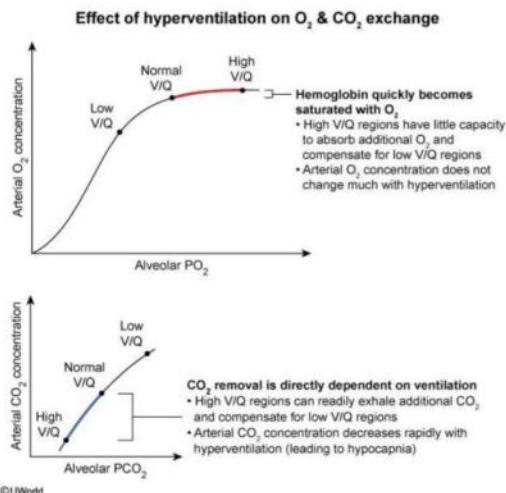
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CO<sub>2</sub> removal is closely tied to ventilatory rate.

alveolar hyperventilation leads to washout of CO<sub>2</sub> --> hypocapnia

--> respiratory alkalosis.

vs

rate of O<sub>2</sub> absorption is capped once Hb in blood is saturated (which occurs at low partial pressure i.e. 85% saturation at PaO<sub>2</sub> 50 mmHg)

hence, blood in highly ventilated lung regions can't absorb extra O<sub>2</sub>, to compensate for the poorly ventilated regions, as in Pul. Embolism or Pneumonia.

usually in such patients, prolonged hyperventilation -->  
resp. muscle fatigue -->  
respiratory failure (hypoventilation with hypercapnia and resp. acidosis)

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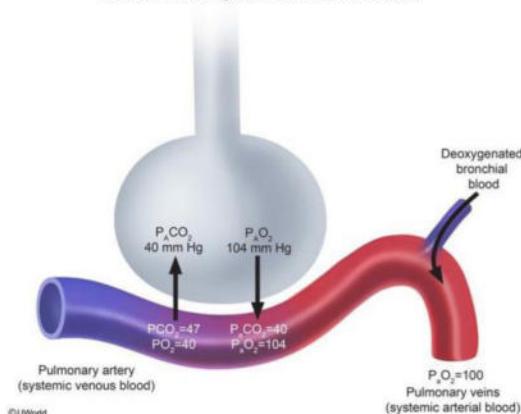
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**Pulmonary arterial hypertension**

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**Anatomic shunting due to bronchial circulation**

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Bronchial arteries supply oxygenated blood to bronchi and bronchioles.

Bronchial Venous (deoxygenated blood) has 2 fates :-

minor fate to the Right Heart via azygos and hemi-azygos veins.

major fate to the Left heart via mixing into Pulmonary vein

+ mixing of small cardiac veins/thebesian veins directly into Left atrium and ventricles

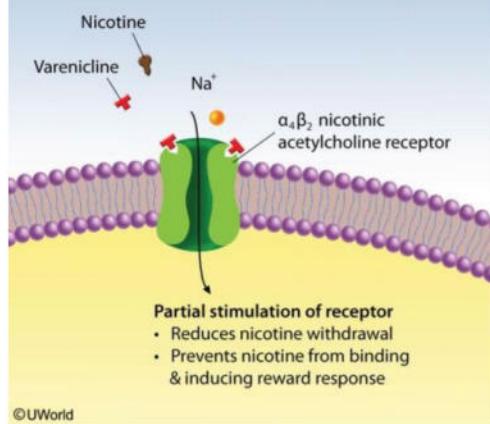
this causes drop from 104 mmHg --> 100 mmHg

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## Mechanism of action of varenicline



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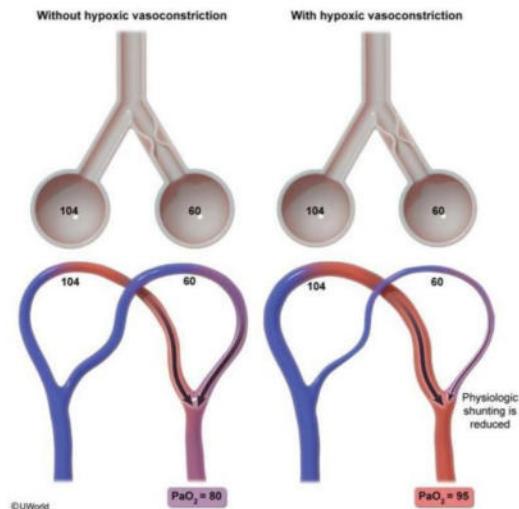
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Varenicline --> is a partial Nicotinic receptor agonist.

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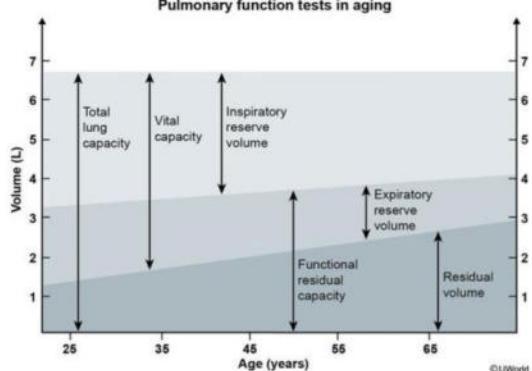
LUNGS are a unique site where

small muscular pul. arteries vasoconstrict in response to hypoxia, to minimize V/Q mismatch.

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Lung Compliance --> Increases  
(due to loss of elastic recoil esp. of alveolar ducts)

this is counterbalanced by

Decreased <-- Chest Wall Compliance  
(due to stiffening)

hence, the TOTAL LUNG CAPACITY --> unchanged

BUT increase in RV  
(due to loss of elastic recoil --> air trapping)

hence, FVC --> decreases.

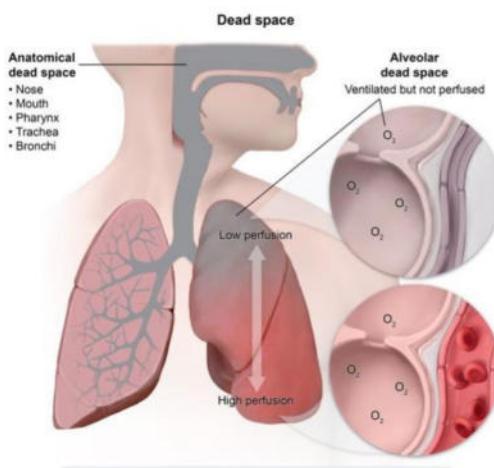
also,

FEV1 --> decreases

DLCO --> decreases

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dead space ventilation is responsible for the difference b/w the

Minute Ventilation (RR \* TV)

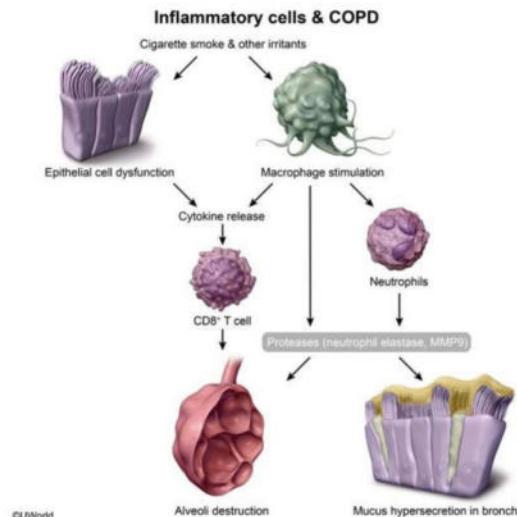
&

Alveolar Ventilation [RR \* (TV – dead space volume)]

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Primary mediators of disease in COPD are :-

Neutrophils

CD8+ T cells

Macrophages

these cells release destructive enzymes and protease responsible for alveolar destruction in "Emphysema"

and

increased mucus production in "Chronic Bronchitis".

ASTHMA (type 1 HST reaction)

Mast Cells

(initial phase ma release of (pre formed) histamine and LT)

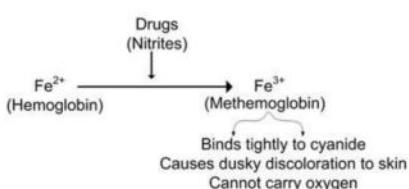
Eosinophils

(late-phase component ; recruited to help sustain the localized inflammatory response following exposure to allergens.)

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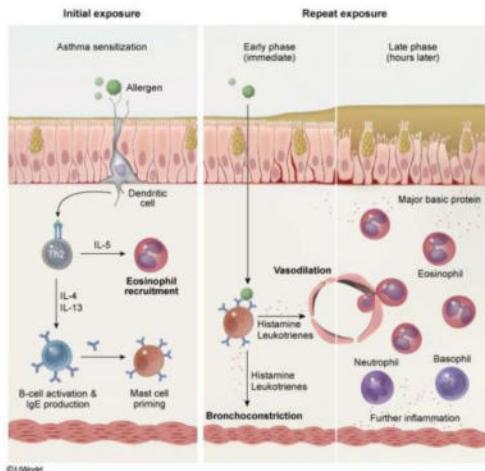


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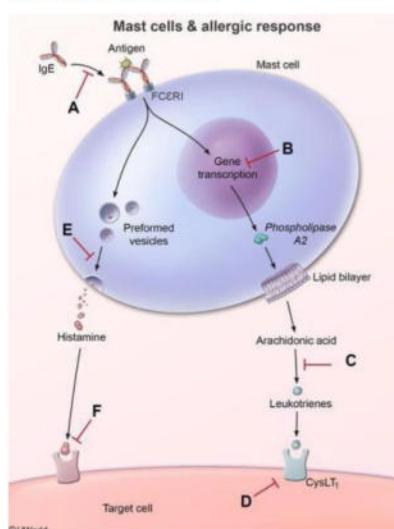
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### Pathogenesis of asthma



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A – Omalizumab  
(in severe, persistent asthma)

B – Corticosteroids

C – Ziluton

D – Monte- / Zafir- lukast  
(for chronic prophylaxis)

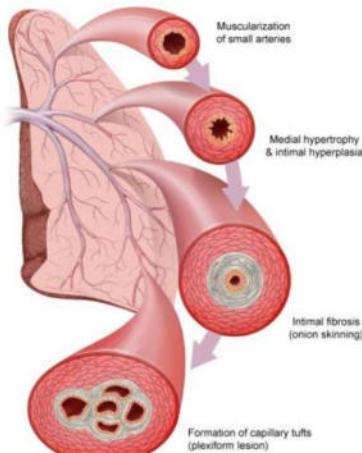
E – Sodium cromoglycate, nedocromil  
(2nd Line, low grade asthma)

F – anti -H1  
(to blunt acute allergic response / in allergic rhinitis)

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Pathogenesis of pulmonary arterial hypertension



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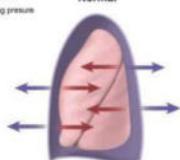
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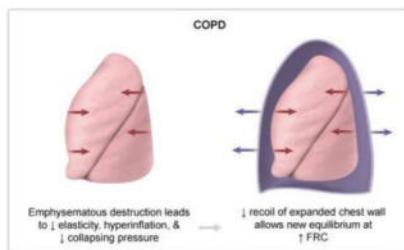
Increased functional residual capacity (FRC) in COPD

■ Lung collapsing pressure  
■ Chest wall expanding pressure

Normal



Pressures are in equilibrium at FRC

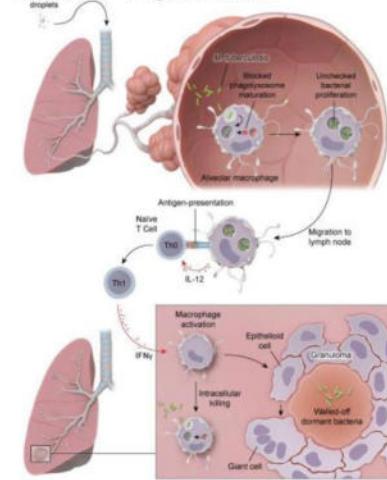


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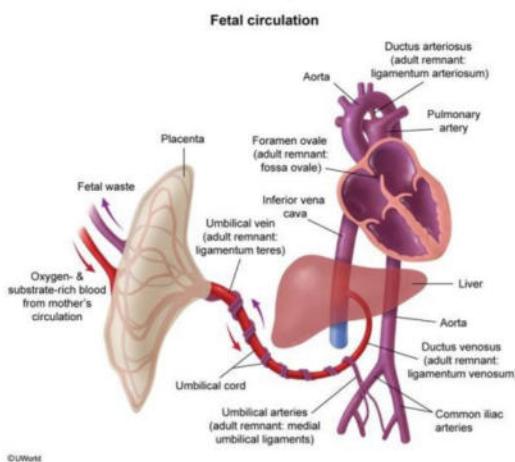
Pathogenesis of tuberculosis



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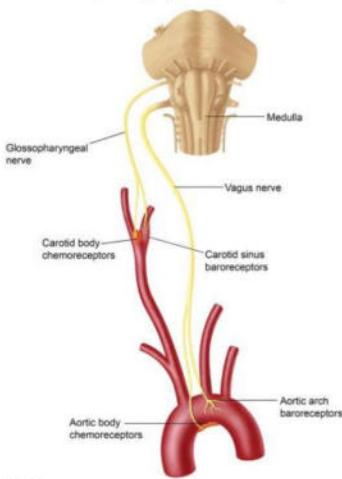
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Baroreceptors & peripheral chemoreceptors



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### MEDULLA ma – RESPIRATORY CENTRE

inputs :-

Chemo + Mechano sensors.

Chemo receptors driving the respiratory rate :-

Central – inc. in PaCO<sub>2</sub> (in healthy)

Peripheral – dec. in PaO<sub>2</sub> [in long term COPD pt;  
CO<sub>2</sub> tolerance develop thai gayo che, hence, hypoxemia --> stimulate these peripheral chemoreceptors. and they are the one maintaining the respiratory drive]

Mechano receptors in Airway + Lung – respond to stretch.

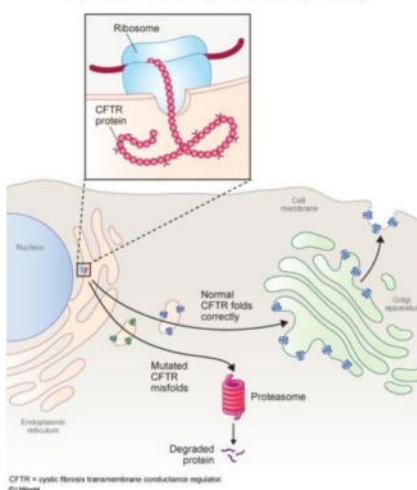
control duration of Inspiration depending on the degree of Lung distension (Hering–Breuer Reflex)

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## ΔF508 mutations &amp; CFTR post-translational processing



CFTR = cystic fibrosis transmembrane conductance regulator  
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Lumacaftor and Ivacaftor combination therapy can be used in CF patients having homozygous delta-F508 mutation.

L – corrects the processing and trafficking of the protein --> enabling it to reach the surface.

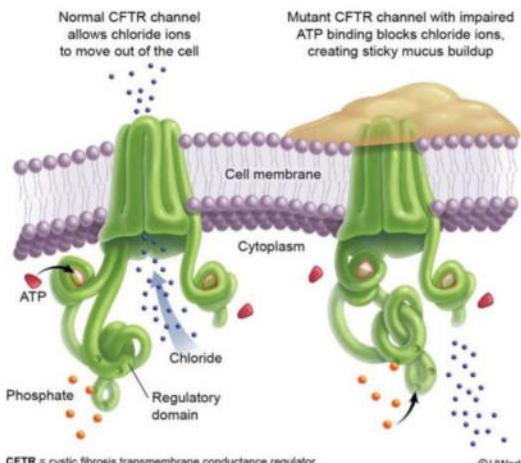
I – improves its functioning (chloride ion transport) at the surface.

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## Cystic fibrosis pathogenesis



CFTR = cystic fibrosis transmembrane conductance regulator.  
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CFTR Protein is responsible for

1. keeping the mucous membranes HYDRATED

2. Sweat secretion hypotonic.

It's an ATP-gated channel.

Other types of channels :-

Voltage-Gated -->

Temperature Gated --> external temperature sensation

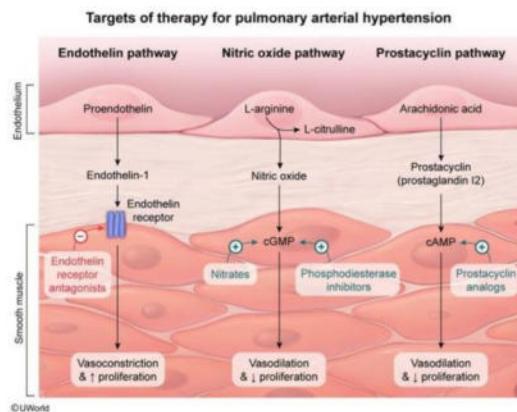
cyclic nucleotide Gated (cAMP) --> photoreceptor & olfactory

Mechanically Gated --> touch & hearing

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Pulmonary HTN :-

GROUP 1 :-

idiopathic/ isolated Pul. HTN

- hereditary
- connective tissue disease (RA, SLE)
- HIV infection

GROUP 2 :-

due to an underlying disorder somewhere in the body.

- HEART – LV failure/ valvular defect
- LUNGS – chronic hypoxemia (OSA, Interstitial fibrosis), chronic recurrent Pul. Embolism
- OTHERS :-

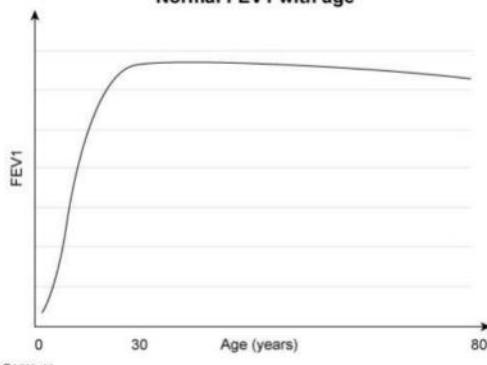
1. Systemic Sclerosis

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### Normal FEV1 with age



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smoking accelerates this age related decline in FEV1

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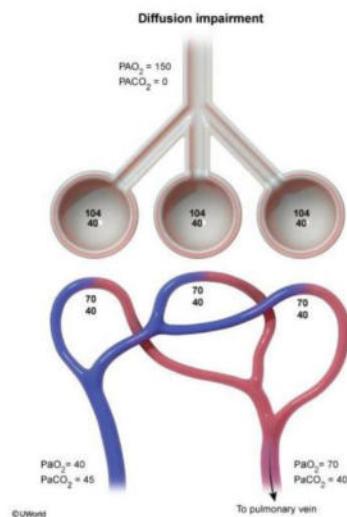
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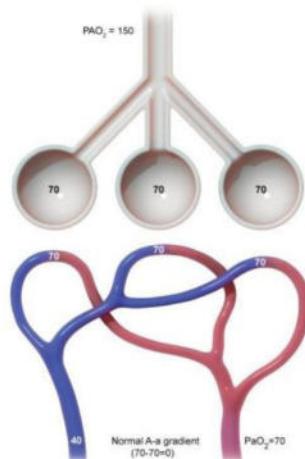


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Decreased alveolar ventilation

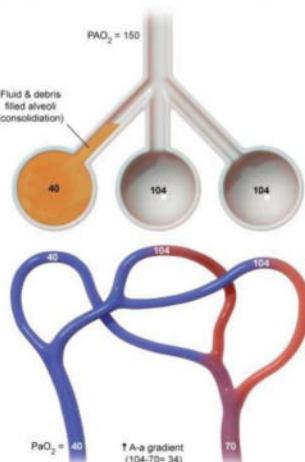


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Physiologic intrapulmonary shunting with V/Q mismatch

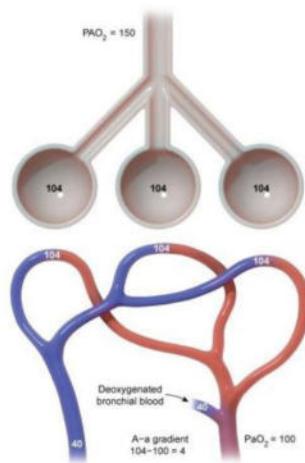


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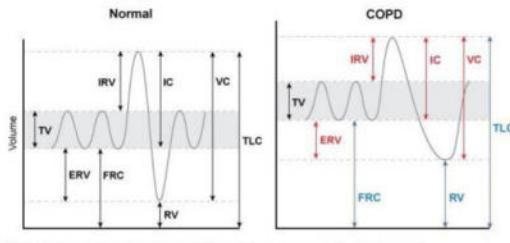
Normal bronchopulmonary shunting



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COPD = chronic obstructive pulmonary disease; ERV = expiratory reserve volume; IC = inspiratory capacity; IRV = inspiratory reserve volume; FRC = functional residual capacity; RV = residual volume; TLC = total lung capacity; TV = tidal volume; VC = vital capacity.

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### Lung Volumes in COPD

decrease in ERV

increase in RV

(absolute volume of air in the lung that is not respired)

increase in RV > then TLC.

HENCE,

RV/TLC ratio – increased

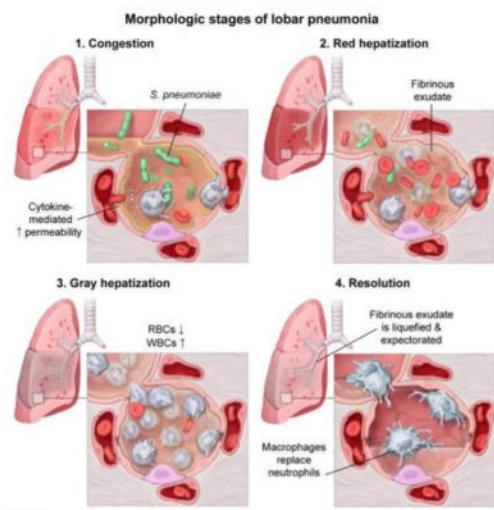
"fraction of air in the lung that is not involved in respiration"

FRC – increased

pt. breathes at higher baseline lung volumes.

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06:55

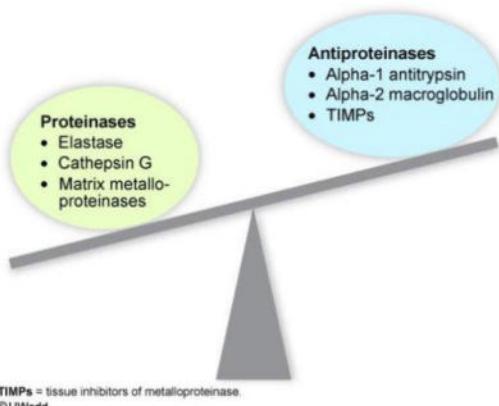


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06:56

## Proteinase/antiproteinase balance



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Alveolar Macrophages --> secrete elastase (contained in lysosomes) (which can degrade extra-cellular matrix proteins)

Neutrophils --> elastase (stored in azurophilic granules)

alveolar elastase inhibited by TIMPs

neutrophil elastase inhibited by serum A1AT

but each can destroy the other's inhibitor.

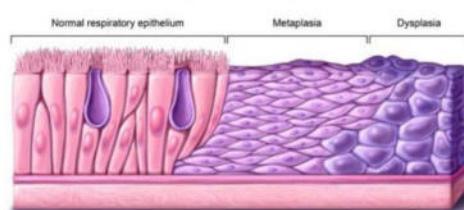
hence, in conditions (emphysema) when these two elastase are together increased risk of damage to alveoli.

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Squamous metaplasia



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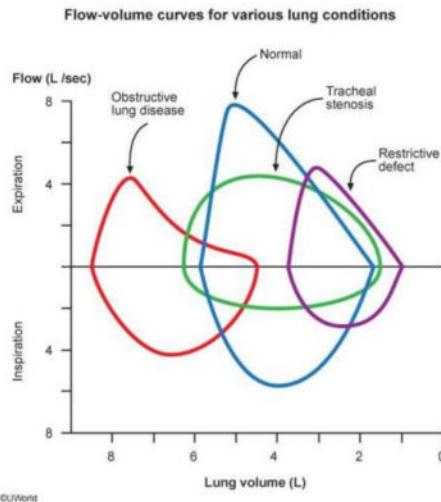
similar to Barrett's Esophagus.

due to smoking --> ADAPTIVE Response of the epithelium (as squamous epithelium is more resistant to irritation)

reversible

if smoking not stopped --> may progress to dysplasia --> cancer.

due to this metaplasia --> impaired muco-ciliary clearance of dust particles --> recurrent respiratory infections



Restrictive lung disease :-

note that any process which causes destruction of lung/ prevents it from doing its work :-

atelectasis (ARDS, fluid in alveoli)

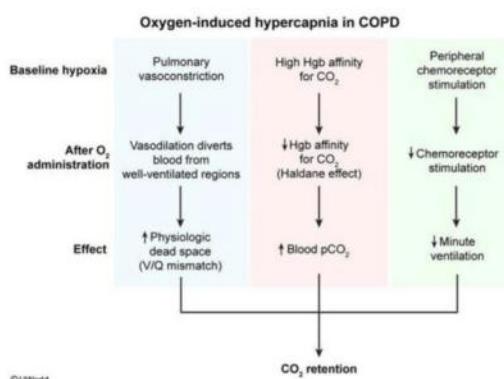
fluid in pleura (extrinsic compression of lung)

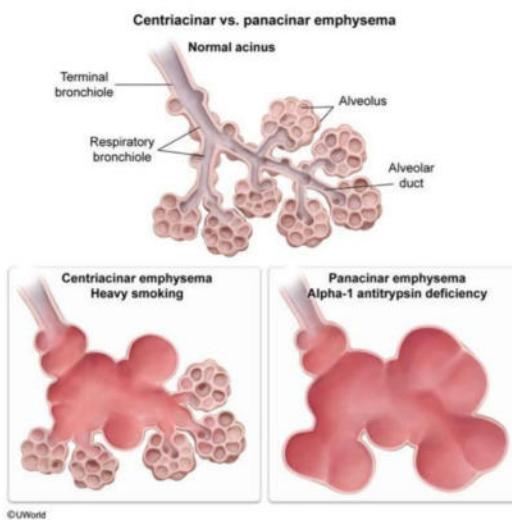
Pulmonary fibrosis

will result in Restrictive Flow–Volume Loop.

in obstructive lung disease..

vice versa



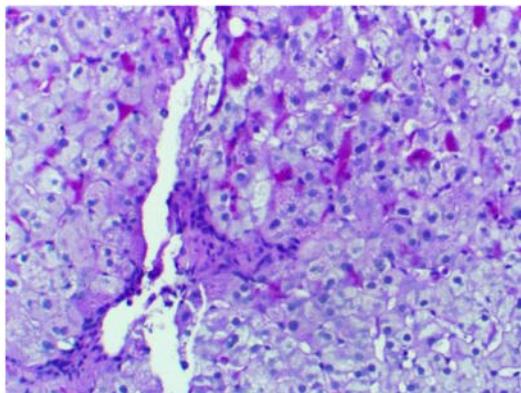


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diastase (which normally digests intracellular glycogen) resistant misfolded AAT granules accumulated in hepatocytes.

seen in AAT deficiency (PiZZ)

Autosomal Co-Dominance.

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07:01

"intra hepatic reddish-pink granules" accumulation.

stained with PAS

due to abnormal folding of alpha 1 anti-trypsin (AAT) --> polymers

accumulate in the peri-portal hepatocytes --> cirrhosis +- HCC

deficiency results in "Inter Alveolar Septa destruction" --> Emphysema (non smoker presents @ 51

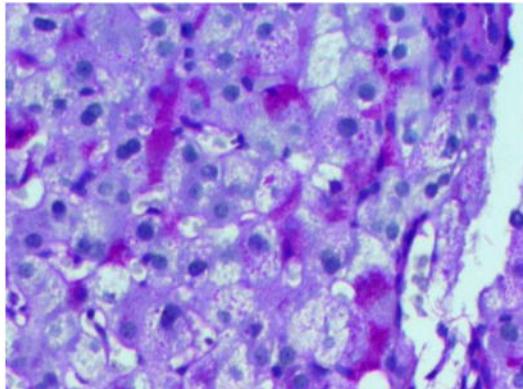
(progressive dyspnea)

smoking is synergistic (causes accelerated destruction, pt presents @ 36 year)

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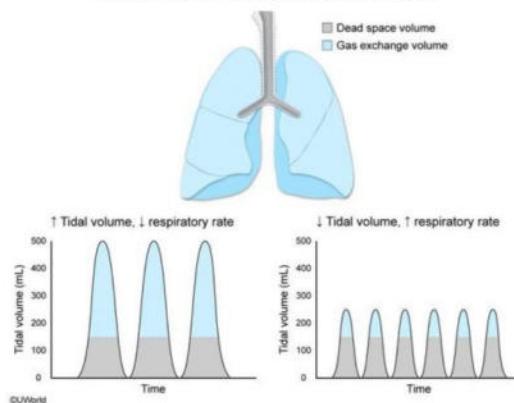


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Low tidal volume increases dead-space ventilation



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In pts. weaned off ventilator --> respiratory muscles are weak -->

hence, shallow breathing (low TV) --> hypoventilation -->  
consequently, the respiratory rate increases --> to maintain the minute ventilation.

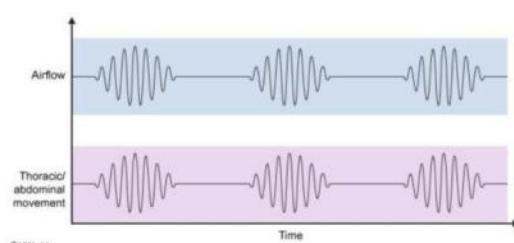
RSBI (rapid shallow breathing index) = RR/TV ;

low index --> high TV, efficient breathing.

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07:02



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seen in Central Sleep Apnea / advanced heart failure,

note the absence of lack of muscular activity --> hence lack of airflow.

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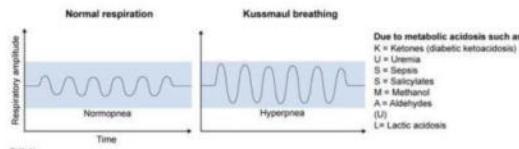
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Cheyne stokes breathing

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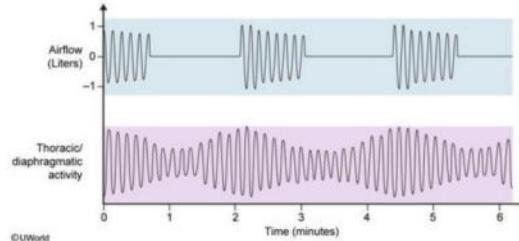
07:02



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07:03



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07:03

note the absence of airflow despite continuous Diaphragmatic activity. this shows that there is Obstruction to the normal airflow.

OSA

pt. will have :-

Loud Snoring with episodes of apnea (>10 sec upto 1 min)  
Poor Sleep and daytime somnolence.

other types of breathing patterns :-

1. In Central Sleep Apnea / advanced heart failure,

Cheyne- Stoke Breathing (apnea -- crescendo-decrescendo -- apnea)

2. Kussmaul's Breathing

in DKA pt. (deep, rapid breathing with increased diaphragmatic activity)

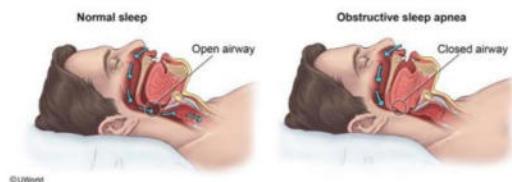
3. in Restrictive Lung Disease,

due to reduced lung compliance --> inc. rate of breathing (RR) to compensate for reduced (TV) --> normalize Minute Ventilation

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07:03



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### Sleep Apnea

primary defect is in narrowing of Oropharynx

Anatomical or Neuromuscular Weakness

(relaxation of dilator muscles of oropharynx during sleep, causing obstruction)

pt. has the following S/S :-

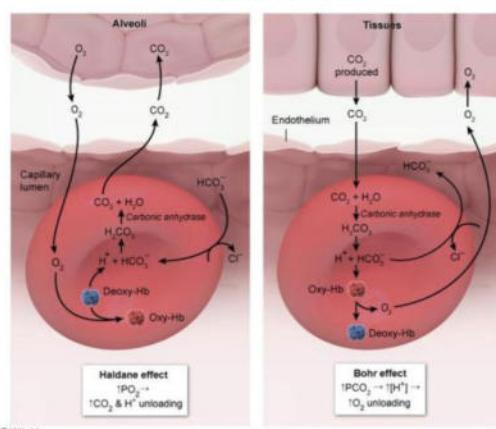
1. Loud Snoring
2. frequent gasping episodes --> poor sleep --> daytime somnolence

Hypoglossal Nerve stimulating implant (which supplies the intrinsic muscles of the tongue) will contract the muscles and increase the A-P diameter of the oropharynx.

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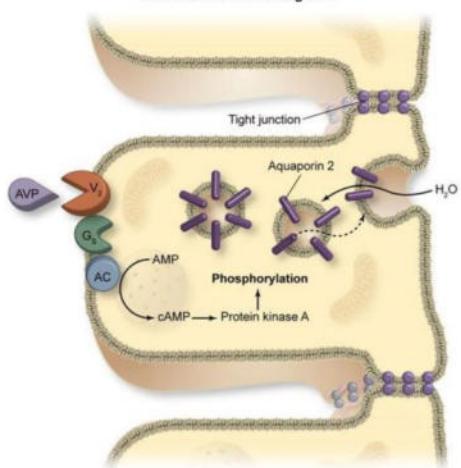
The Haldane & Bohr effects



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### ADH action on collecting duct

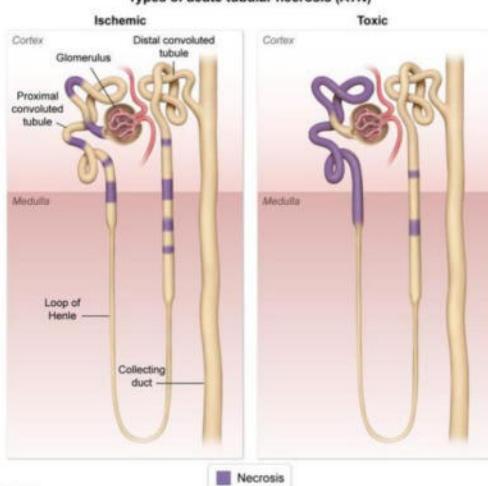


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### Types of acute tubular necrosis (ATN)

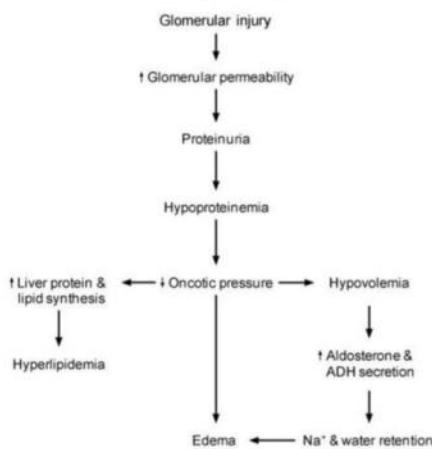


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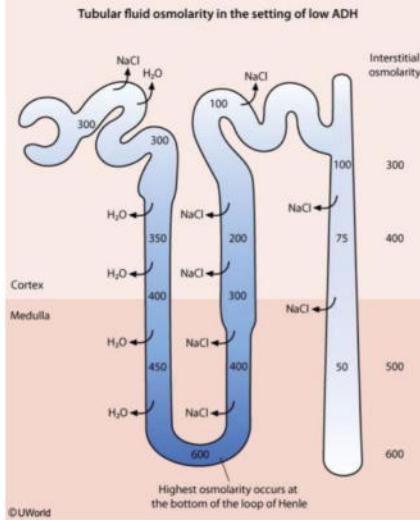
### Overview of nephrotic syndrome



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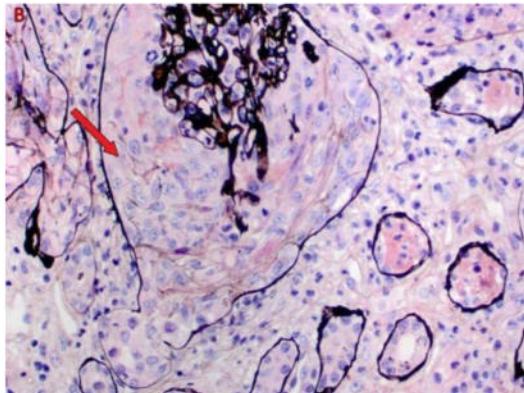
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"crescent formed by fibrin deposition and parietal cell proliferation"

in RPGN.

immune or inflammatory- mediated damage to capillaries -->

infiltration of

1. coagulation factors (eg - fibrinogen) &
2. inflammatory cells (lymphocytes/macrophages)

through the damaged GBM -->

release of inf. cytokines and growth factors -->

activation of coagulation cascade (fibrin deposition) +  
parietal cell proliferation +  
fibroblast recruitment -->

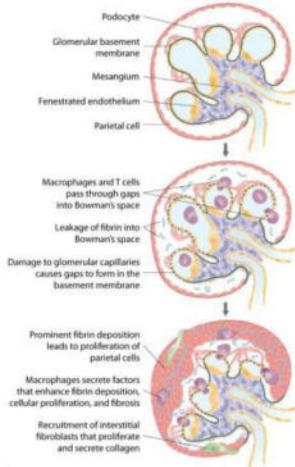
glomerular hypercellularity, fibrosis -->

irreversible damage and reduction in GFR.

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Pathogenesis of crescent formation  
in rapidly progressive glomerulonephritis



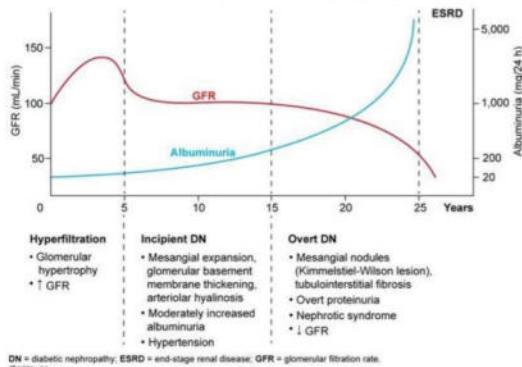
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## Natural history of diabetic nephropathy



DN = diabetic nephropathy; ESRD = end-stage renal disease; GFR = glomerular filtration rate.

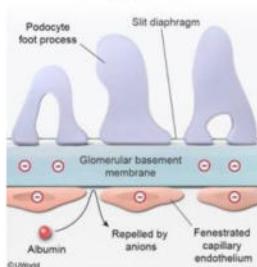
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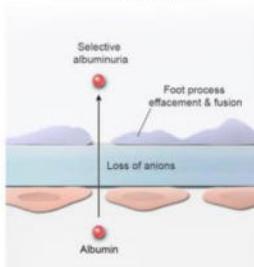
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## Normal



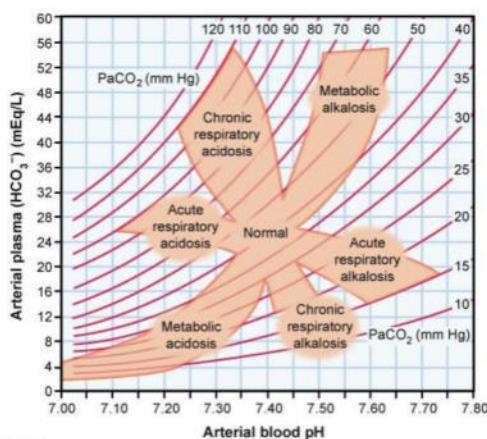
## Minimal change disease



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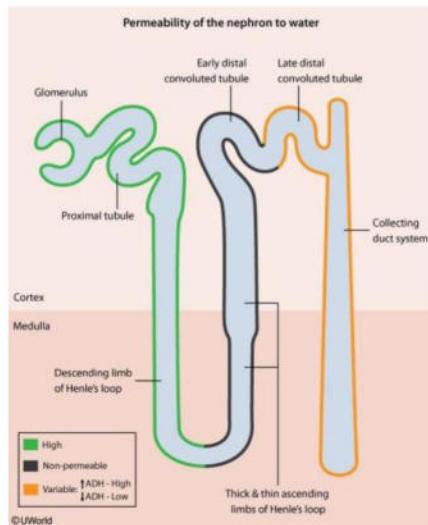
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### GREEN PORTION

>60% of water reabsorbed in PCT

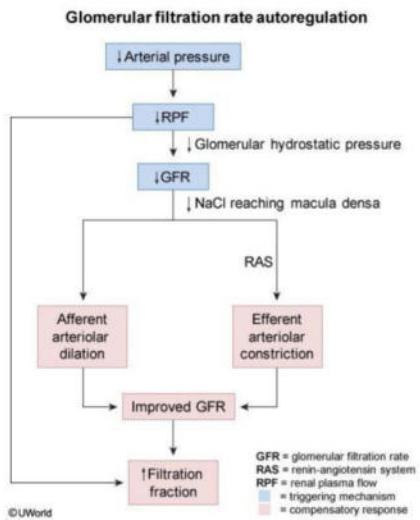
(isotonically i.e. passively along with other filtered substances)

-20% in descending loop of henle  
 (due to increasing cortico-papillary gradient)

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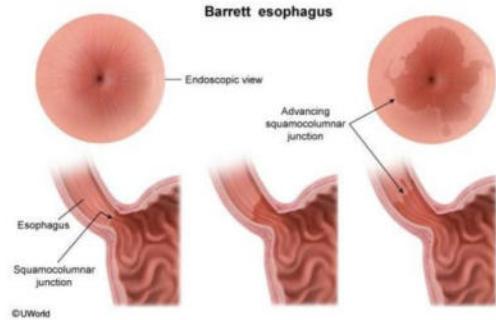
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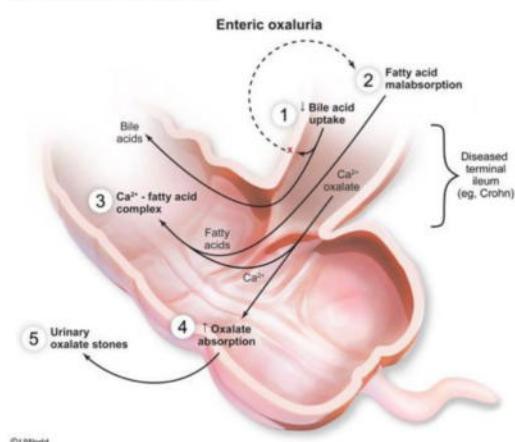
GERD > Obesity

NO RISK by H Pylori.

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## UWORLD IMAGES

07:07

DD OF DIFFERENT RENAL STONES :-

1. Crohn's ma "Ca Oxalate Stones". Also, chronic intake of high dosages on Vit C, increases its formation.

2. Upper Urinary Tract Infection ma "Struvite" (Magnesium Ammonium Phosphate) + "CaCO<sub>3</sub> apatite" due to the presence of Urease producing organisms. (*Klebsiella*, *Proteus*)

3. "Hexagonal" Cystine Stones (due to hereditary defect in dibasic aa transporter in PCT)

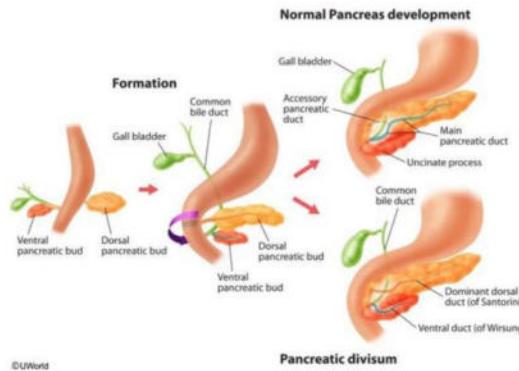
4. Uric Acid Stones in Chronic Hemolysis/ Tumor Lysis.

5. Primary HyperPTH can cause Renal Stone formation.

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Pancreas forms from Endoderm

Ventral (small bud) gives rise to :-

MAIN Pancreatic Duct + Uncinate Process + Inferior Head

Dorsal (large bud) gives rise to all other structures.

ACCESSORY Pancreatic Duct.

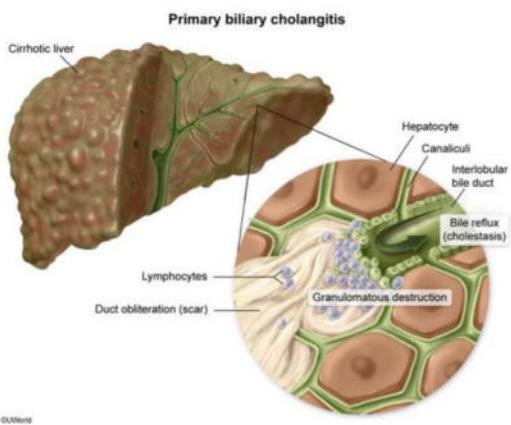
this accessory duct's proximal end/opening into the duodenum may be resorbed.

CBD and bulk of the pancreatic secretions drain through this MAIN Pancreatic Bud which arises from the VENTRAL BUD.

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Histo-Pathologic findings :-

"Patchy lymphocytic Infiltrate in the intra hepatic bile ducts with necrosis and micro nodular regeneration of the Peri Portal tissues/ Bile Duct Proliferation".

this disease affects

"Small and Mid- sized" Intra hepatic Bile Ducts

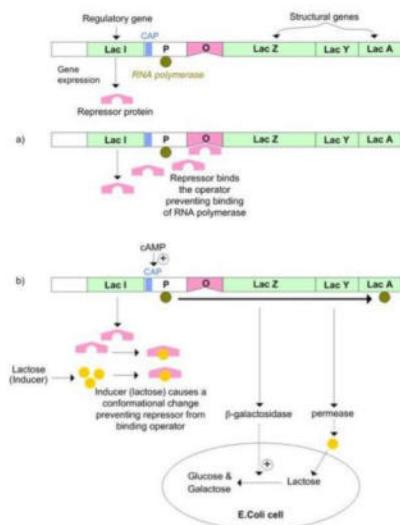
VS

"Large Intra- and Extra- Hepatic Bile Ducts" in PSC.

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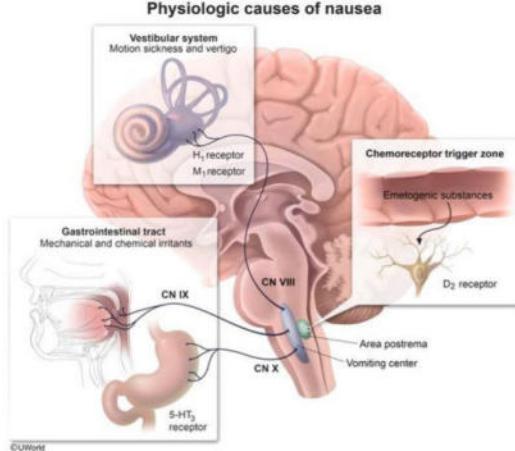
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07:10

5-HT<sub>3</sub> receptors are located :-

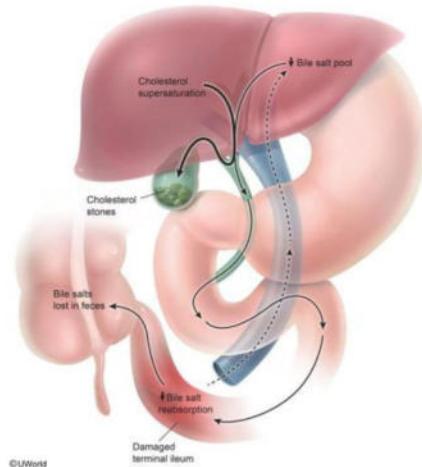
1. In the pre-synaptic Vagus nerve terminals of the GI Tract.
2. in the CTZ (central)
3. in the NTS (central)

Ondansetron/Grani/

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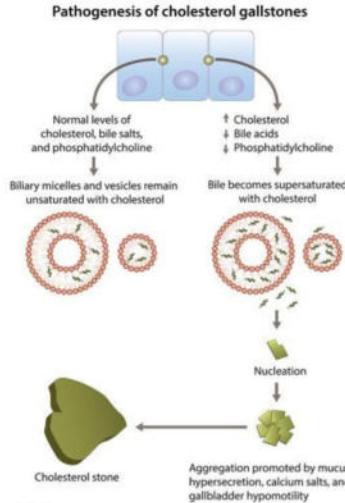
07:11

Pathogenesis of gallstones in Crohn disease



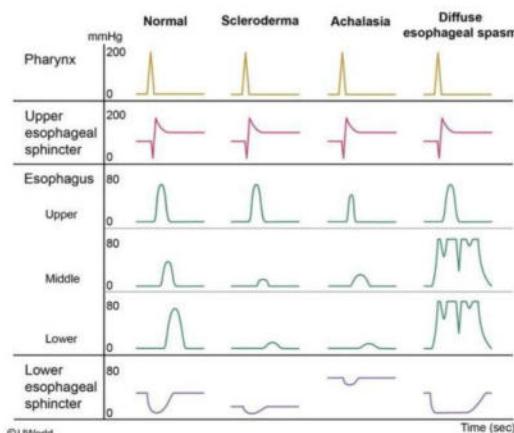
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07:11

## UWORLD IMAGES



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## UWORLD IMAGES

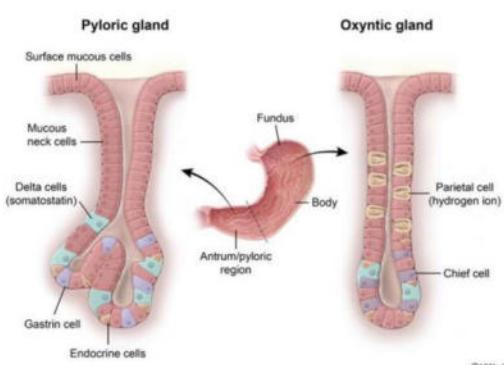
Scleroderma ma LES "tone decreased and incomplete relaxation"

Achalasia ma Esophageal Body reduced amplitude of peristalsis + Increased Resting Tone of LES with incomplete relaxtion.

Diffuse Esophageal Spasm ma Multiple/Simultaneous Esophgeal body spam with relaxed LES

07:11

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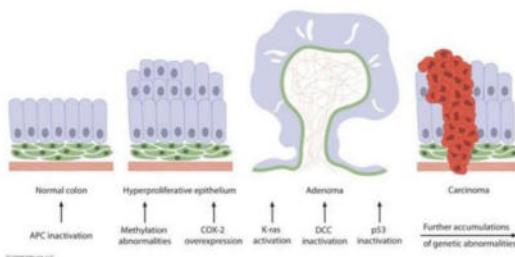


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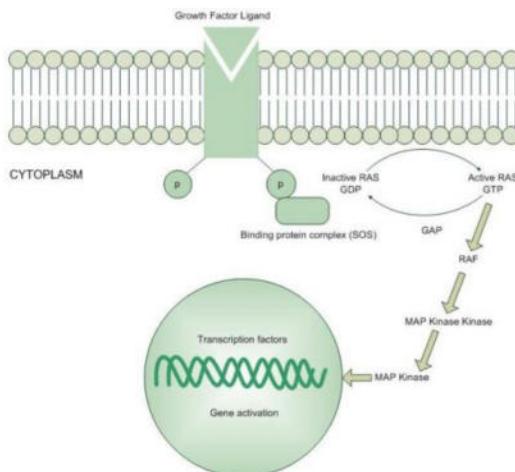
## Adenoma to carcinoma sequence



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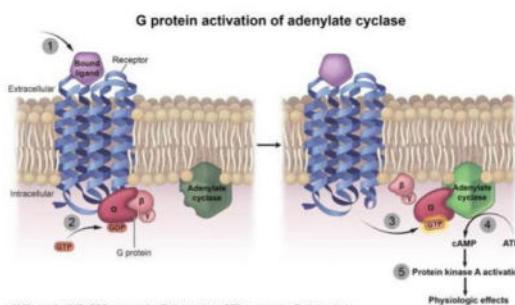
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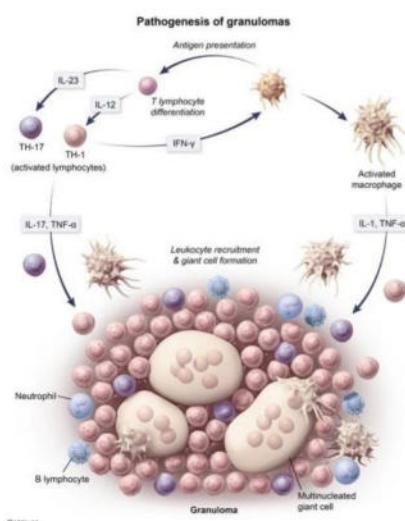
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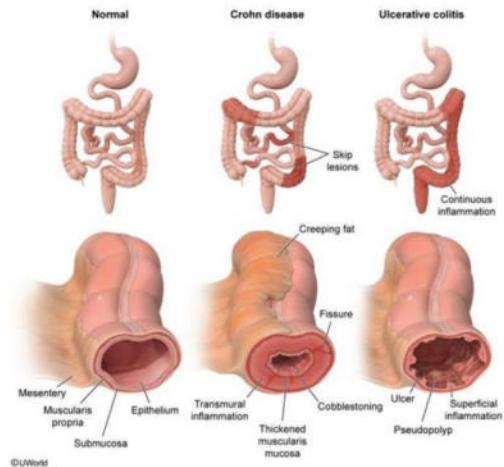
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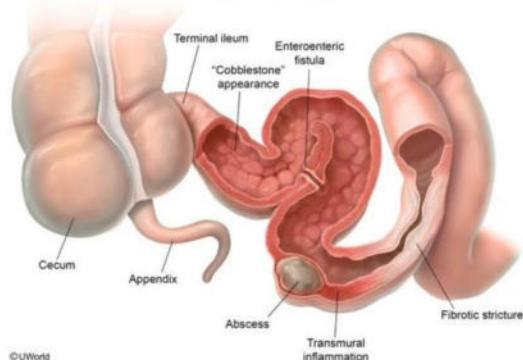


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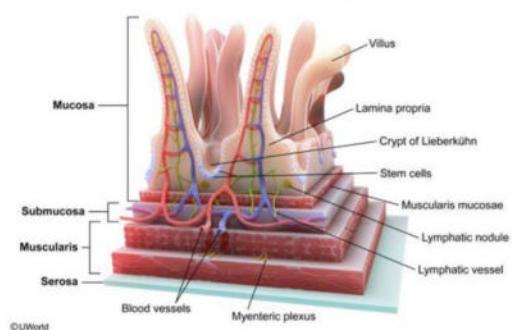
## Crohn disease vs ulcerative colitis



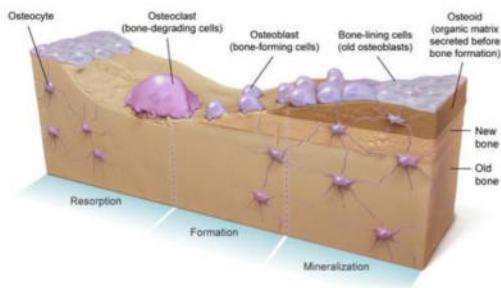
## Crohn disease



## Small intestinal histology



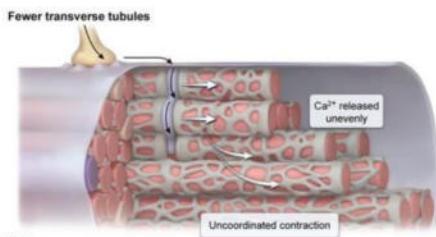
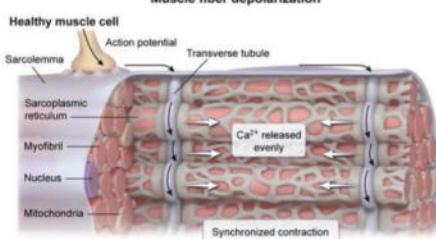
### Bone remodeling



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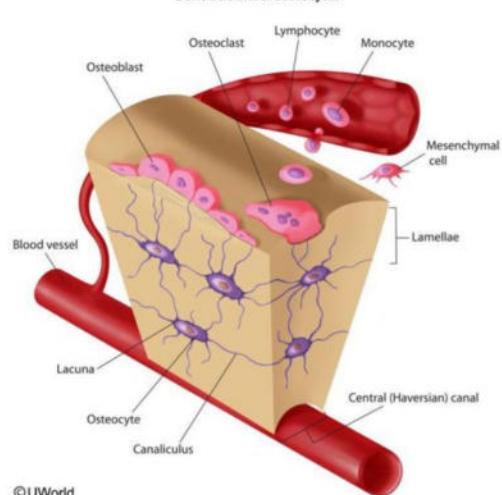
### Muscle fiber depolarization



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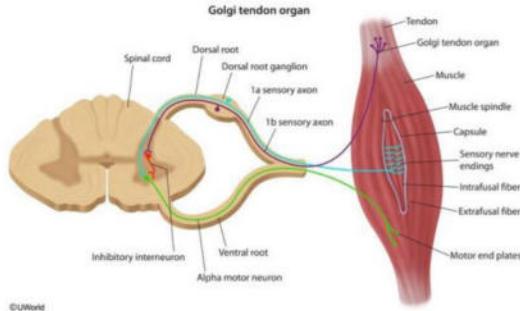
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### Bone Structure: Osteocytes



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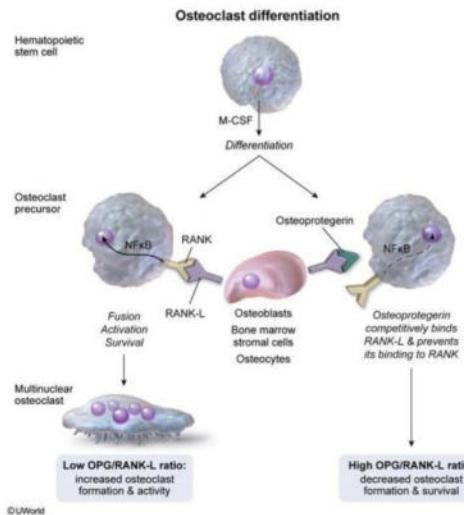
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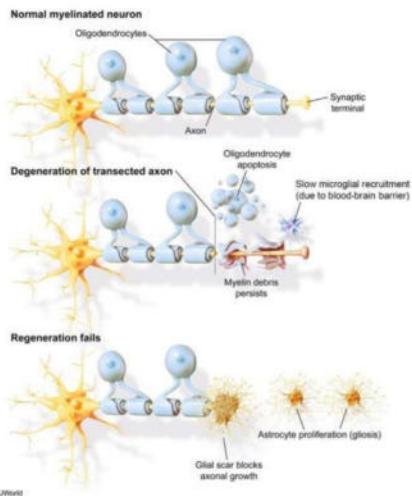
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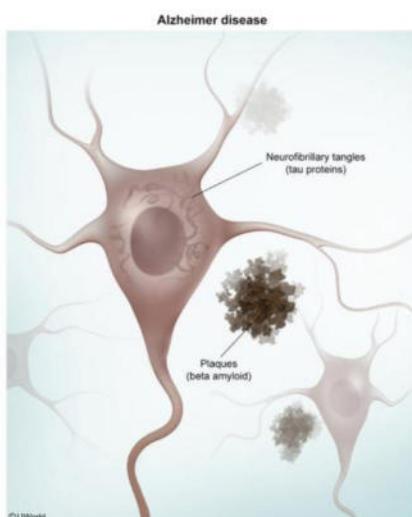
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### Wallerian degeneration in CNS



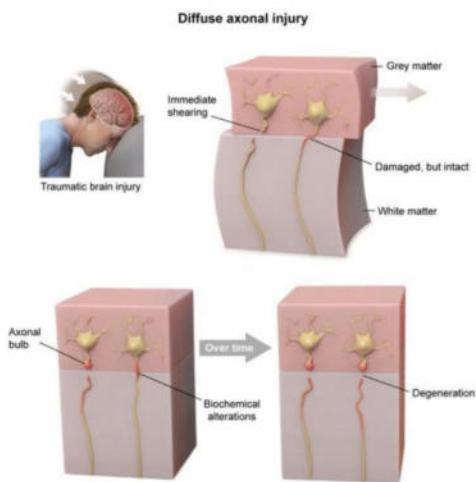
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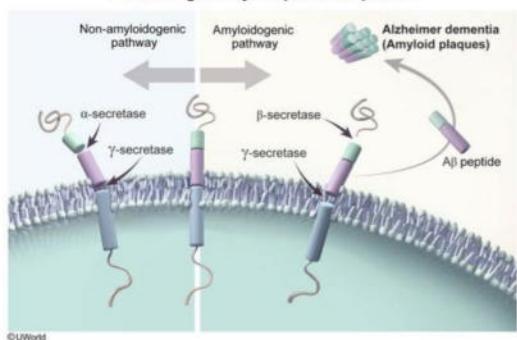
07:14



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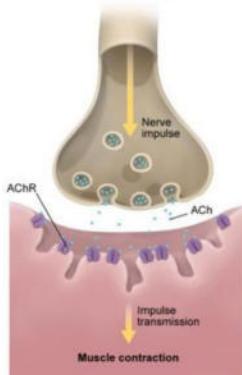
## Processing of amyloid precursor protein



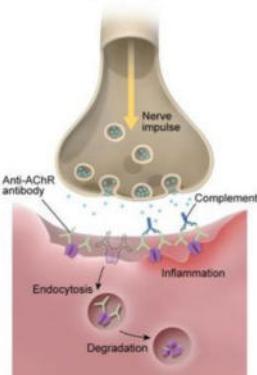
## UWORLD IMAGES

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Normal neuromuscular junction



Myasthenia gravis

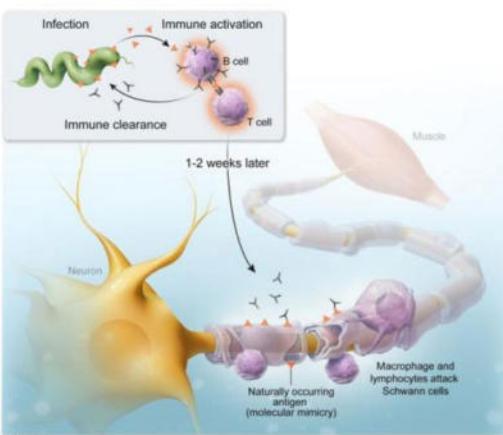


ACh = acetylcholine; AChR = acetylcholine receptor.  
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Pathogenesis of Guillain-Barré Syndrome



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## Pronator drift



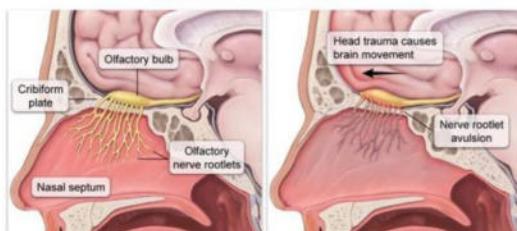
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Olfactory nerve rootlet avulsion with TBI



TBI = traumatic brain injury

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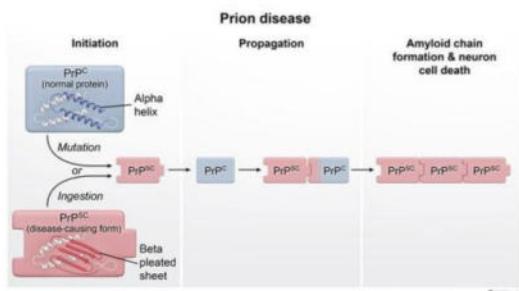
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Kayser fleischer ring

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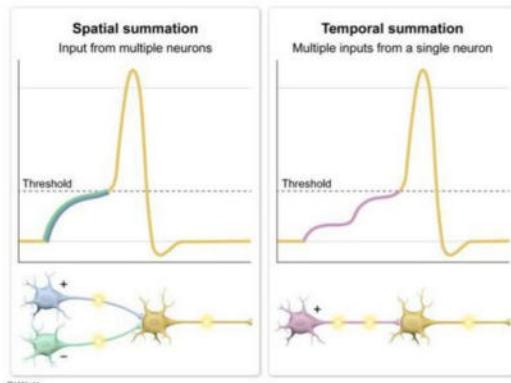


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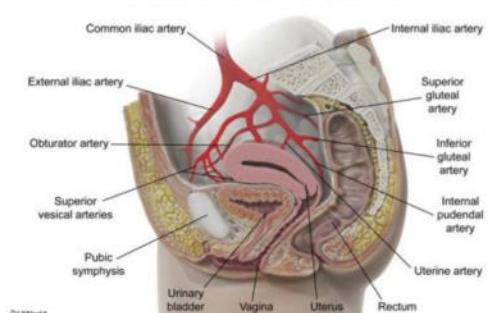


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Branches of the internal iliac artery in the female

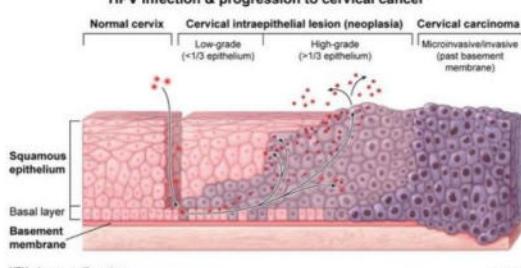


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HPV infection &amp; progression to cervical cancer



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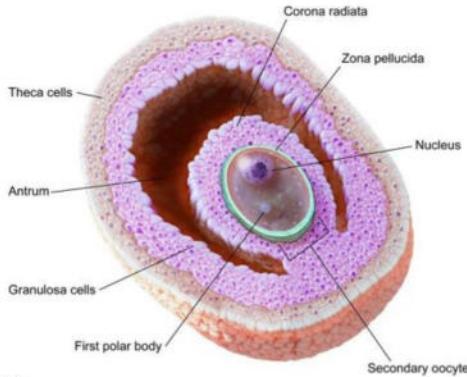
Pemphigus vulgaris

07:17

## UWORLD IMAGES

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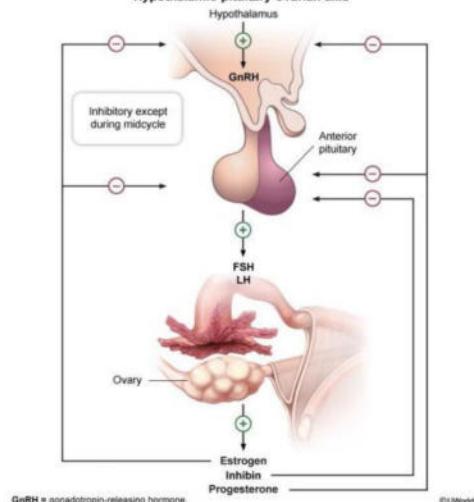
**Ovarian tertiary follicle**

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**Hypothalamic-pituitary-ovarian axis**

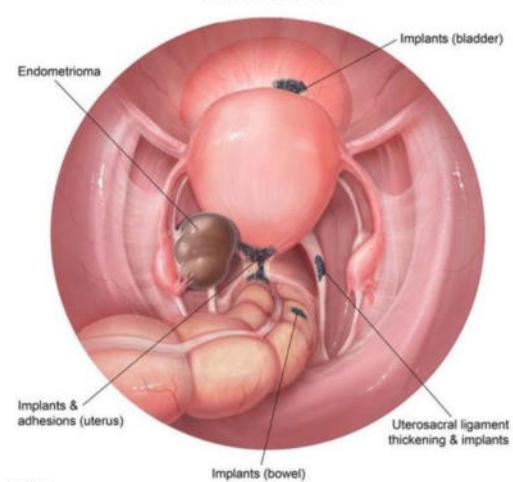
GnRH = gonadotropin-releasing hormone.

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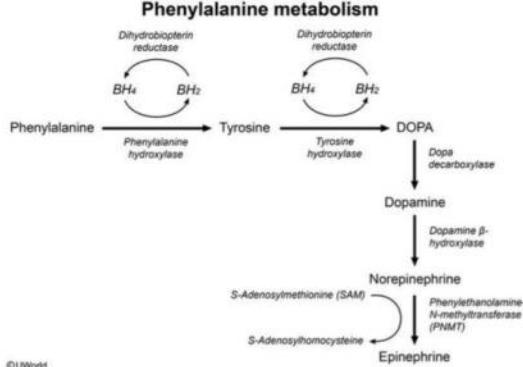
**Pelvic endometriosis**

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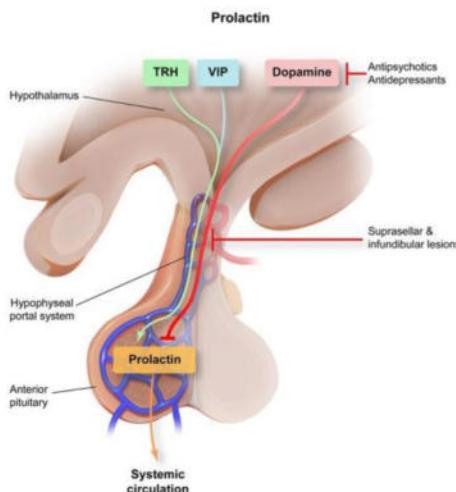
**UWORLD IMAGES**

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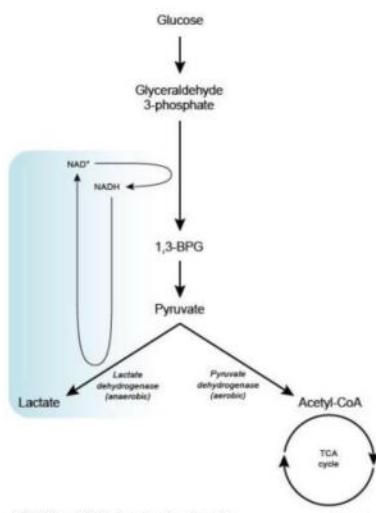
## UWORLD IMAGES

07:18



## UWORLD IMAGES

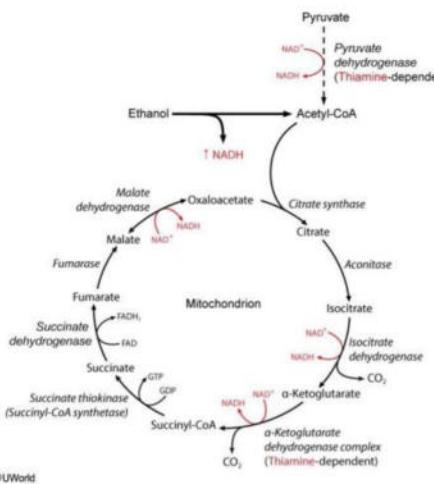
07:18



## UWORLD IMAGES

07:18

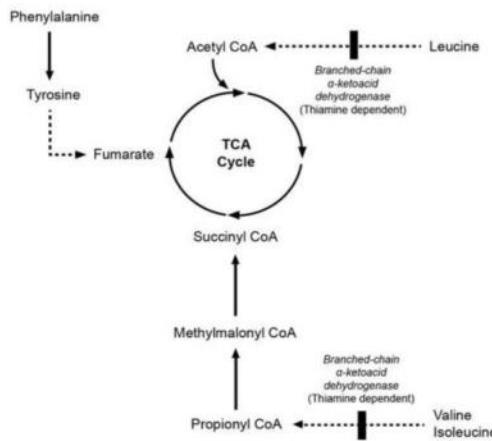
## Inhibition of the citric acid cycle by ethanol



## UWORLD IMAGES

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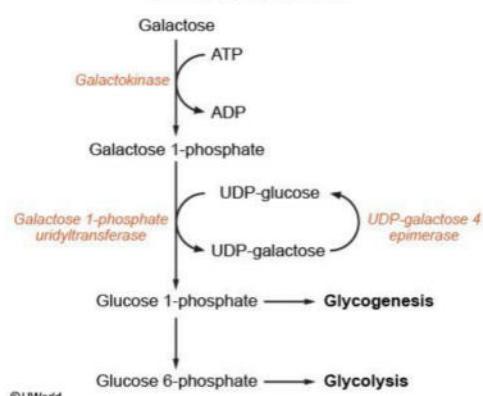


## UWORLD IMAGES

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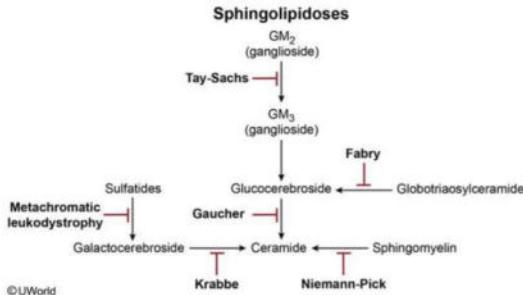
### Galactose metabolism



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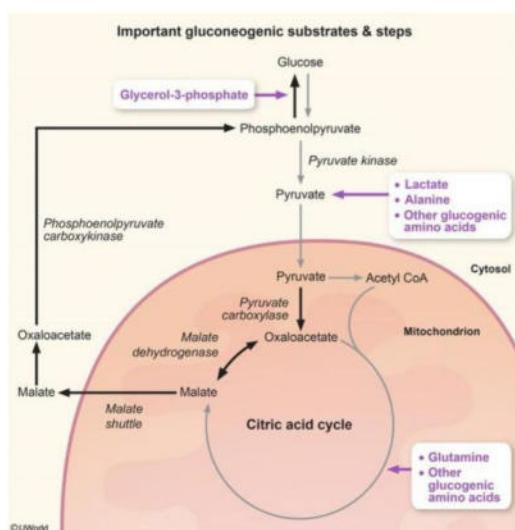
07:18

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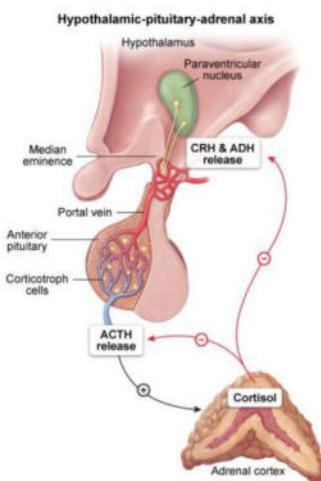
## UWORLD IMAGES

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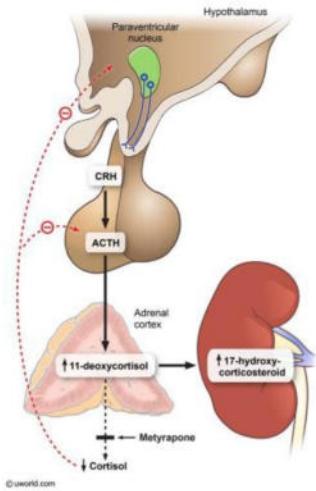
## UWORLD IMAGES

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## UWORLD IMAGES

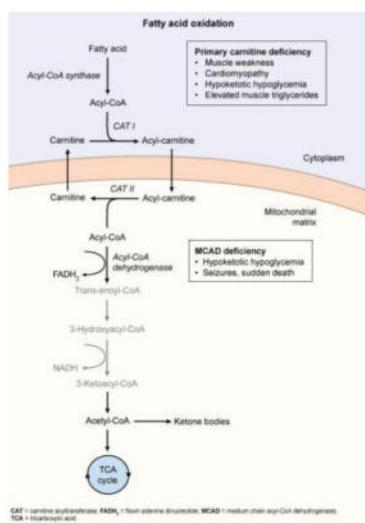
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**Metyrapone stimulation test**

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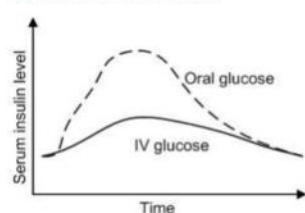
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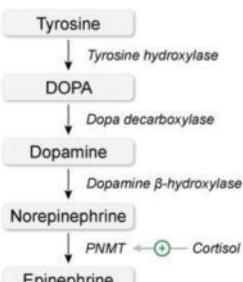
07:18



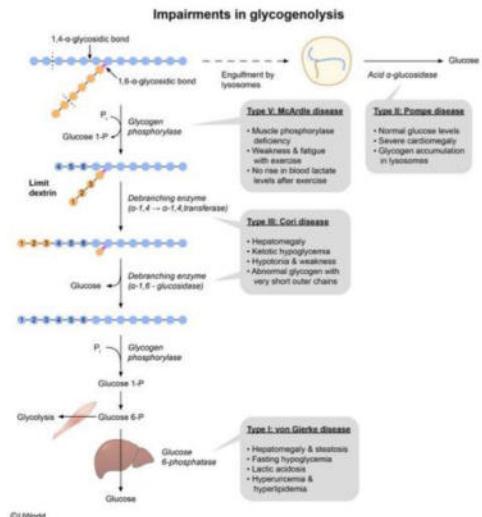
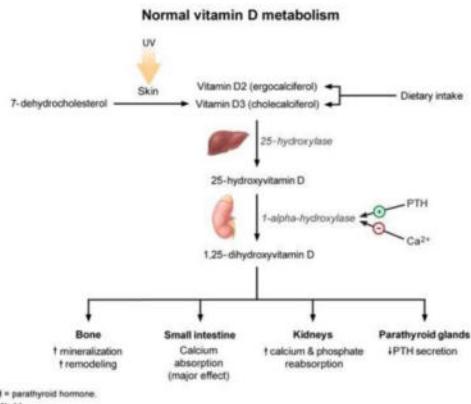
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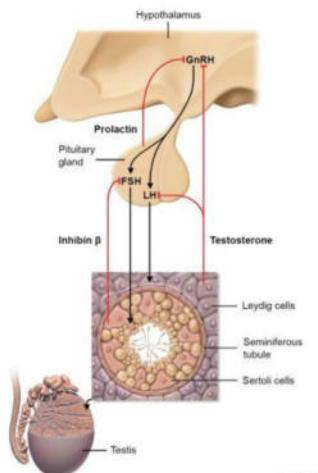
07:19

**Catecholamine synthesis**

PNMT = phenylethanolamine-N-methyltransferase.  
©UWorld

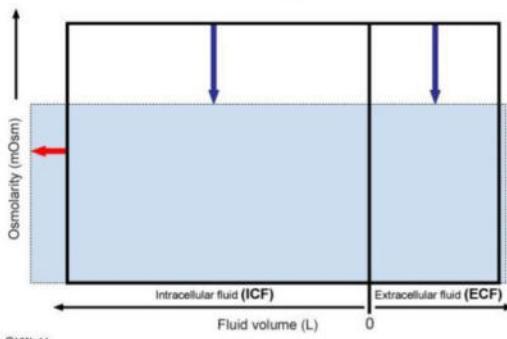


### Prolactin and gonadotropin production



Primary polydipsia & SIADH

↓Osm      ↑ICF



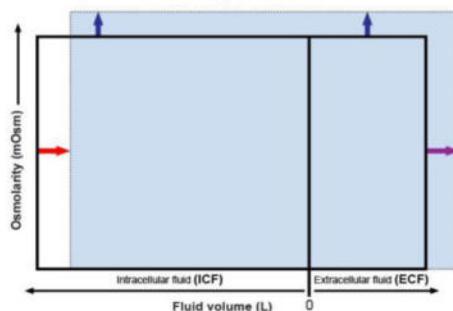
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Hypertonic saline infusion

↑Osm      ↓ICF      ↑ECF



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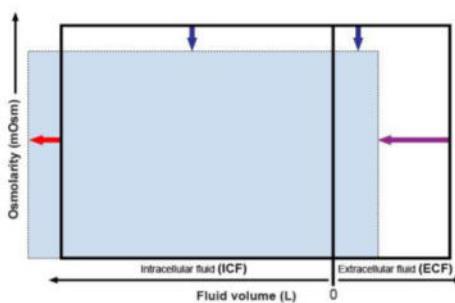
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07:19

Adrenal insufficiency

Hypotonic loss of Na<sup>+</sup>

↓Osm      ↑ICF      ↓ECF



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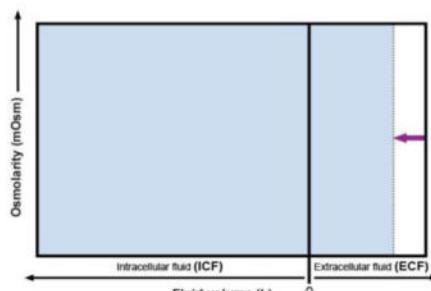
UWORLD IMAGES

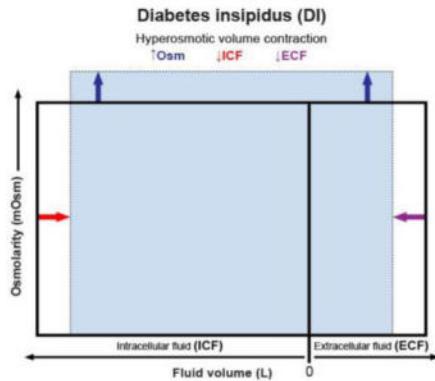
07:19

GI hemorrhage (or diarrhea)

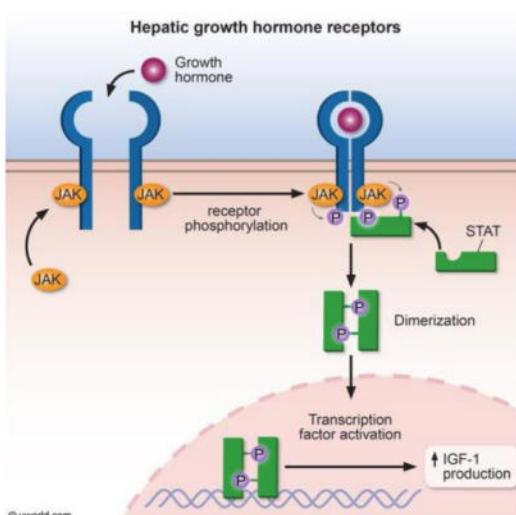
Loss of isotonic fluid

No change Osm      Normal ICF      ↓ECF

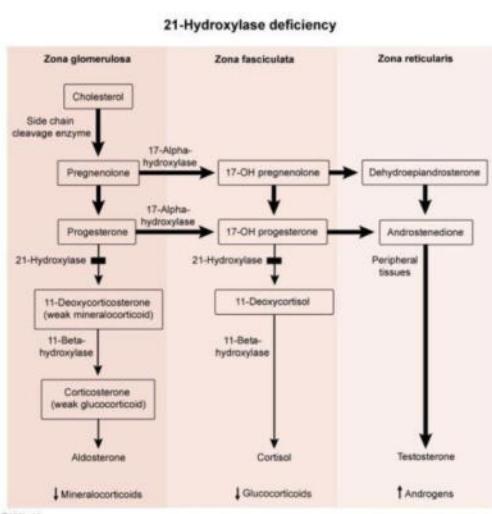




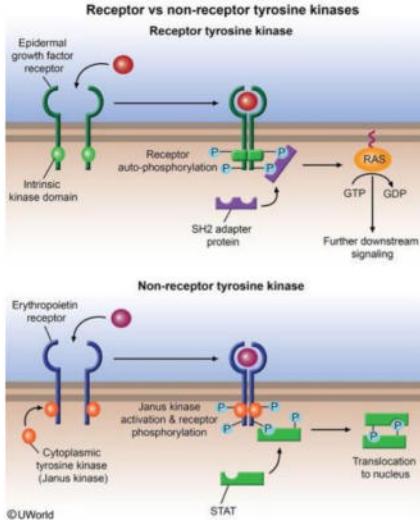
©UWorld



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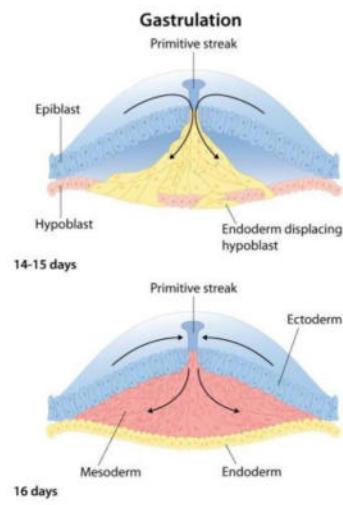


©UWorld



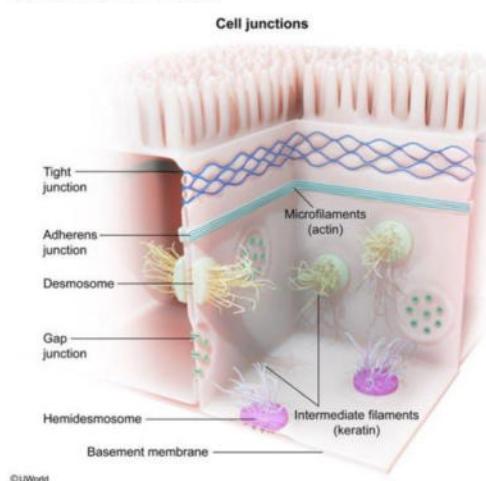
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07:20



## UWORLD IMAGES

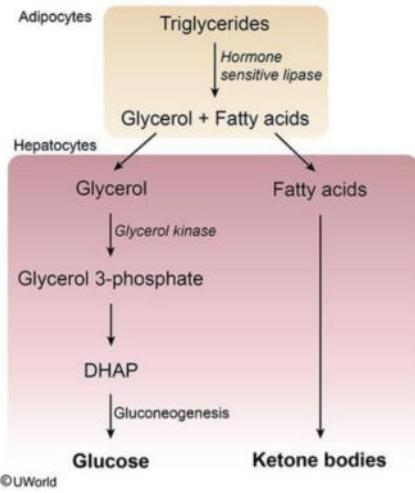
07:21



## UWORLD IMAGES

07:21

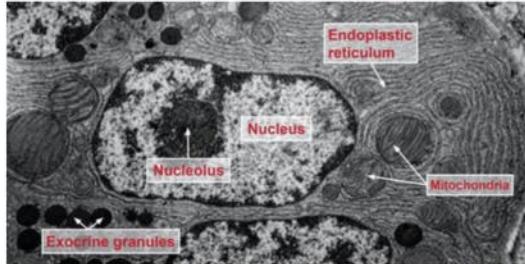
## Triglyceride metabolism during fasting



07:22

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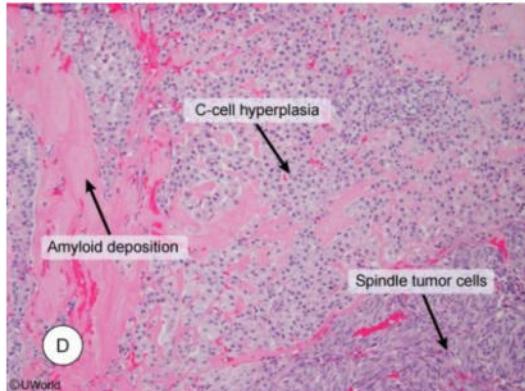
## UWORLD IMAGES



07:22

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## UWORLD IMAGES



07:23

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Medullary Thyroid Carcinoma -

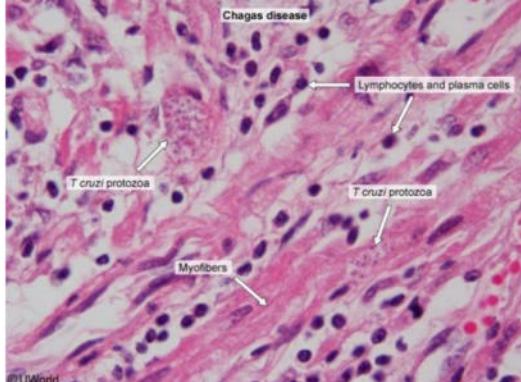
C-cell hyperplasia

07:23

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07:23



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07:23

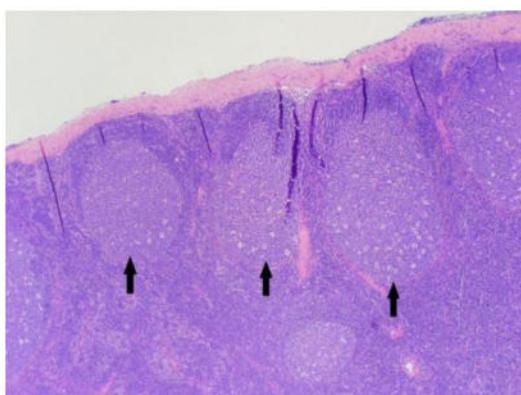
Germinal Centers of a normal lymph nodes.

site of B-cell proliferation, i.e Isotype Switching (requires CD-40L activated T-cell to bind with CD-40 on B-cell)

& Affinity Maturation (via Somatic Hypermutation)

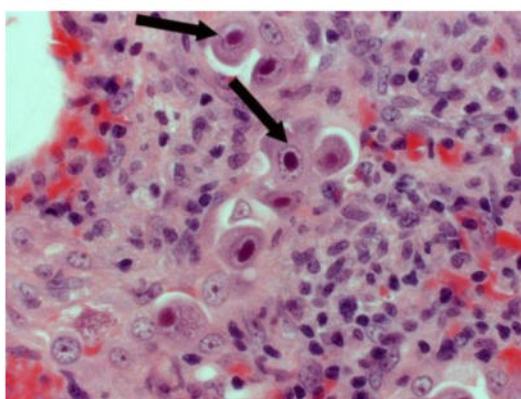
**UWORLD IMAGES**

07:23



**UWORLD IMAGES**

07:23



**UWORLD IMAGES**

07:24

enlarged cells with intranuclear inclusions surrounded by halo ("Owl's Eye") + Intracytoplasmic inclusions (viral particles)

seen in infection with CMV Pneumonitis (usually in Post lung transplant patient)

in AIDS pt. CMV causes

retinitis, esophagitis and colitis.

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### UWORLD IMAGES

07:24

"foamy trophozoites" (resembles macrophages)

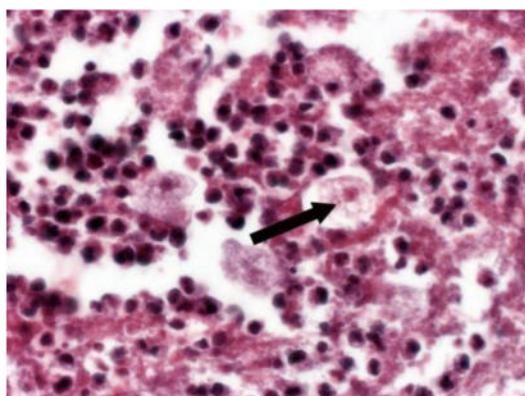
seen in amoebic dysentery (bloody diarrhea)

causes flask shaped ulcers.

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07:24



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### UWORLD IMAGES

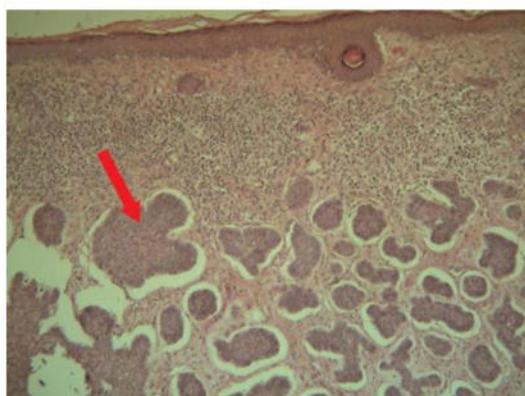
07:24



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07:25



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07:25

**Basal Cell carcinoma**

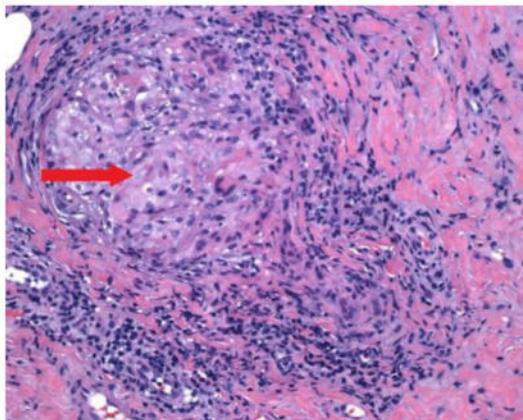
arrow --&gt;

Basal epidermal cells (basophilic) forming islands surrounded by clear spaces and extending across the basement membrane.

U

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07:25



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07:26

(in the context of the question)

Arrow --&gt; a (retained) foreign body and a granuloma

Histologic,  
foreign bodies eliciting a granulomatous response.

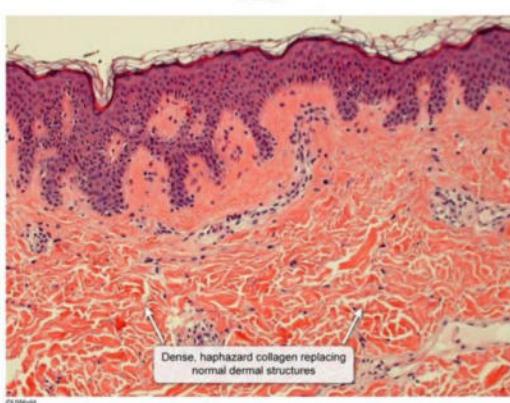
gross,  
tender erythematous/brown/purple papule, nodule, plaque.

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07:26

Keloid



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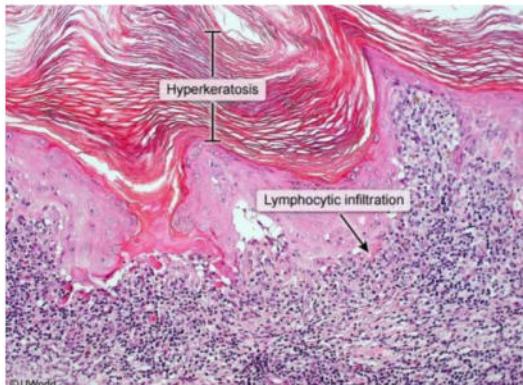
07:26

note the haphazard arrangement of thick, pink collagen bundles resulting from excessive granulation tissue [KELOIDS]

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07:26



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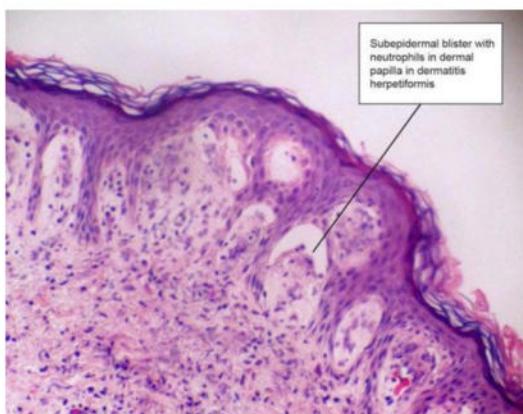
07:27

**Lichen Planus****lymphocytic (CD8+ve T lymphocytes) at the dermal epidermal junction.**

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07:27



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07:27

**neutrophils on the tips of dermal papillae  
(forming micro abscesses)****seen in  
Dermatitis Herpetiformis ass. with celiac disease.**

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**UWORLD IMAGES**

07:27



histologic findings seen in Lichen Planus (ass with Hep C)

1. hyperkeratosis (thickening of the S. Corneum)
2. hypergranulosis
3. saw toothing of the rete ridges
4. CD8+ T- lymphocytic infiltration at the dermal epidermal junction
5. degeneration of the basal layer of the epidermis. scattered eosinophilic, colloid (Civatte) bodies in the papillary dermis (apoptotic keratinocytes)



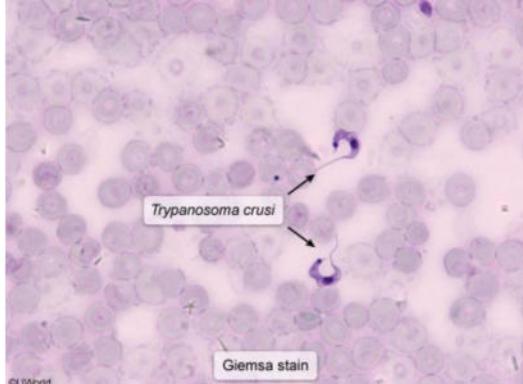
PSORIASIS – chronic inflammatory disorder.

silvery scales  
(thickening of stratum corneum – HYPERKERATOSIS)  
+  
(retention of nuclei in the stratum corneum – PARAK.)

and

on top of erythematous plaques  
(diffuse epidermal hyperplasia – ACANTHOSIS)  
+  
elongated and clubbed rete ridges

Skin lesion on the extensor aspect of knees and elbows.



### UWORLD IMAGES

07:28

Acute Phase :- U/L Peri-orbital swelling, myo- / peri- carditis

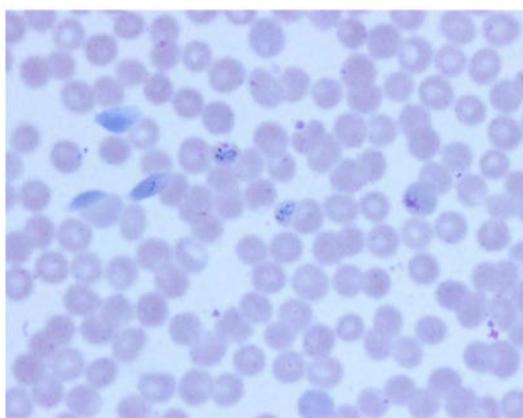
years late due to Immune related cross-reactivity leads to destruction of (submucosal) Meissner's Plexus and (muscularis) Auerbach's Plexus, causing

Megacolon, Achalasia (Megaesophagus).

Dilated cardiomyopathy

### UWORLD IMAGES

07:28



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Intraerythrocytic Ring Forms

Malaria (female anopheles mosquitoes)

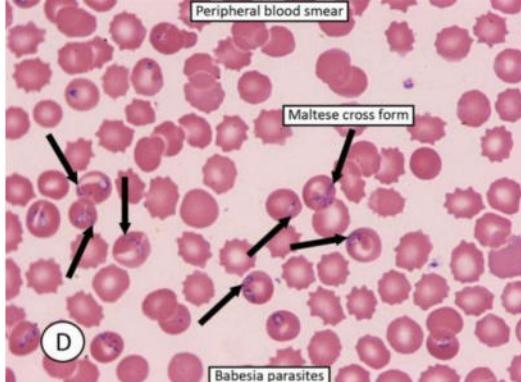
Babesiosis (Ixodes Scapularis Tick)

Channel photo changed



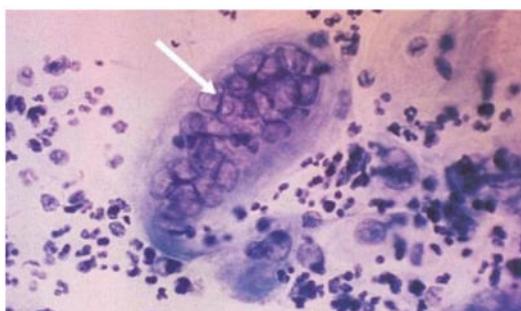
### UWORLD IMAGES

07:43



**UWORLD IMAGES**

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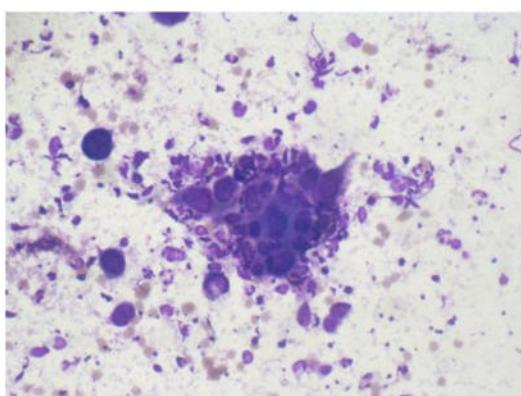
07:43

Multinucleate Giant cell seen in HSV infection.

seen when infected epithelial cells scraped from ulcer base are stained by Tzanck Stain.

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07:43



**UWORLD IMAGES**

07:43

Multinucleated Giant Cell with "ground-glass opacities" when stained with TZANCK Stain.

seen in HSV Infection.

spread by direct contact with herpetic sores / infected genital secretions during sexual activity.

**MULTIPLE Small (sub-cm) Painful Genital Ulcers**

with  
shallow, erythematous BASE  
+  
systemic s/s  
fever, LAD, dysuria

vs

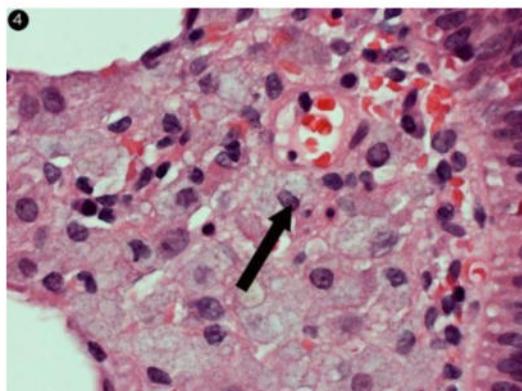
CHANCROID (by H. Ducreyi)

MULTIPLE deep Painful Genital Ulcers  
with ragged borders &  
gray to yellow EXUDATIVE BASE  
+  
Painful LAD

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07:43



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07:44

foamy macrophages in the lamina propria of the small intestine.

caused by gram +ve Tropheryma whipplei.

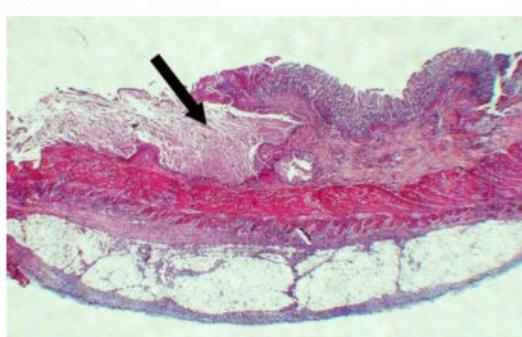
s/s :-

arthralgia  
chronic diarrhea with malabsorption  
weight loss

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07:44



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07:44

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flask-shaped ulcers (with overhanging edges)

in the caecum and ascending colon seen in Bacillary Dysentery.

caused by *E. Histolytica*.

invasive diarrhea – RBC + Leukocytes seen in stools

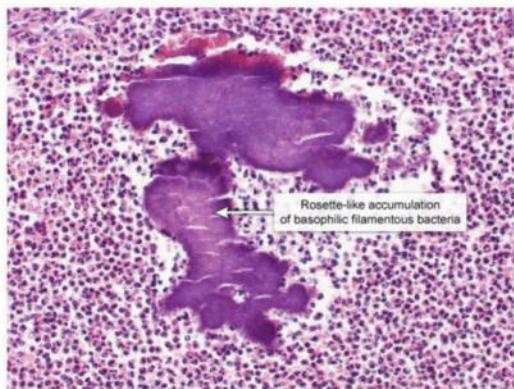
can also cause Liver Abscess .

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Pulmonary actinomycosis



## UWORLD IMAGES

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Actinomycosis

gram positive

anaerobes

filamentous/branching bacteria

usually causes :-

1. CervicoFacial Abscesses (slowly progressive) across various planes with draining sinuses and Sulfur granules

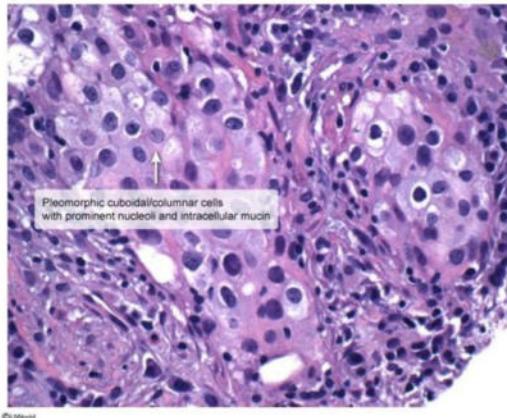
2. may cause Pulmonary actinomycosis, if pt. is alcoholic and aspirates.

SNAP

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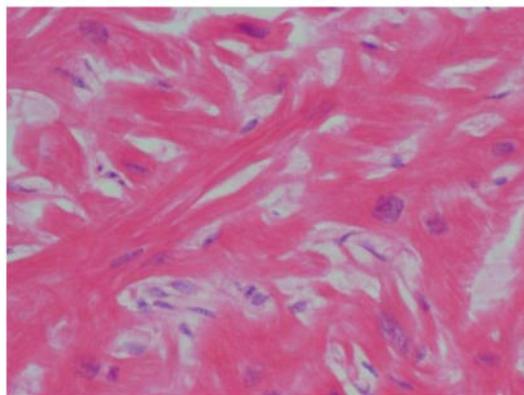
07:44

nonsmokers with cough,  
hemoptysis and  
pulmonary consolidation with air bronchograms  
(air filled bronchi with surrounding alveolar opacification)

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07:44



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**UWORLD IMAGES**

07:45

irregular arrangement of "abnormally shaped cardiomyocytes"  
with bizarrely shaped nuclei  
+  
increased interstitial fibrosis (not shown)

HOCM

sudden death in young pt. (due to V. Fib/ V. Tachy.)

pt will be ASYMPTOMATIC or

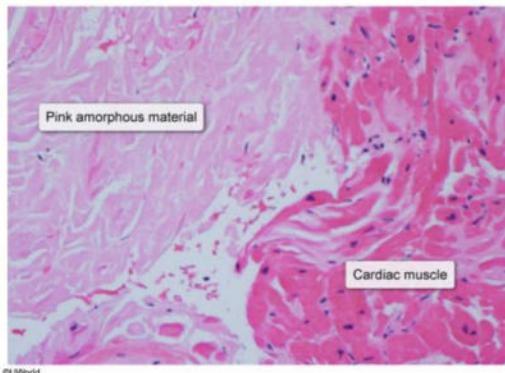
may have h/o exertional dyspnea, fatigue, chest pain, syncope.

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07:45

## Amyloid



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07:45

seen in Amyloidosis

(diastolic failure due to filling defect, consequent to stiffening)

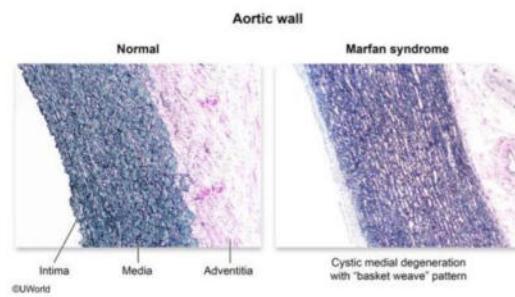
vs

(thickening in concentric cardiomyopathy)

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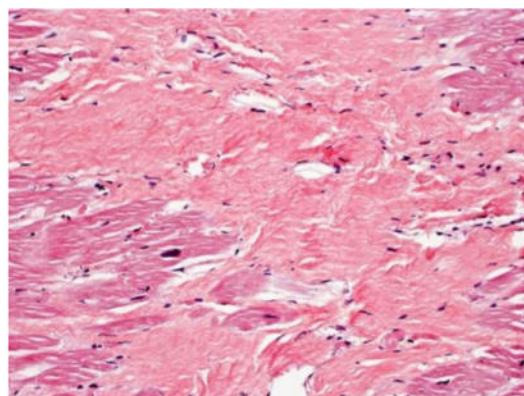
07:45



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07:45



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07:45

(light pink region)

are "type 1 collagen fibres"

(provides strength and support to cellular tissues and organs)

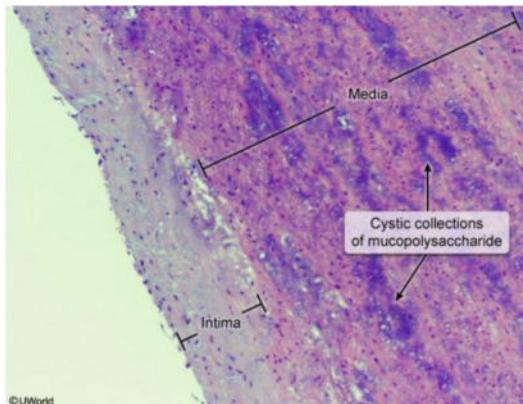
(are found in scar tissues)

due to scarring post-MI

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07:45



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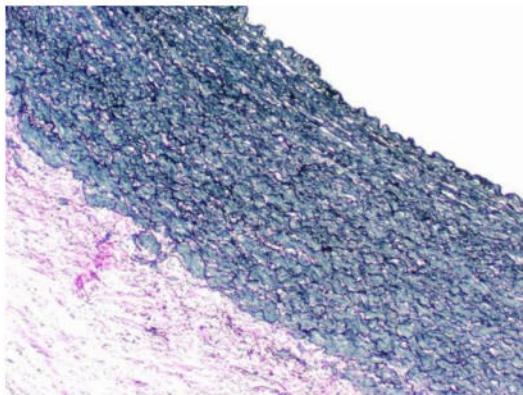
07:46

seen in cystic medial degeneration due to marfan's

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07:46



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07:46

normal media of the arterial wall.

compared to Cystic Medial degeneration

in

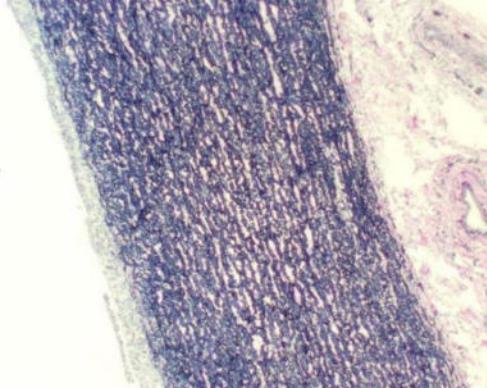
AA

AD

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07:46



### UWORLD IMAGES

07:46

"fragmentation of elastic tissue" in "BASKET WEAVE pattern"

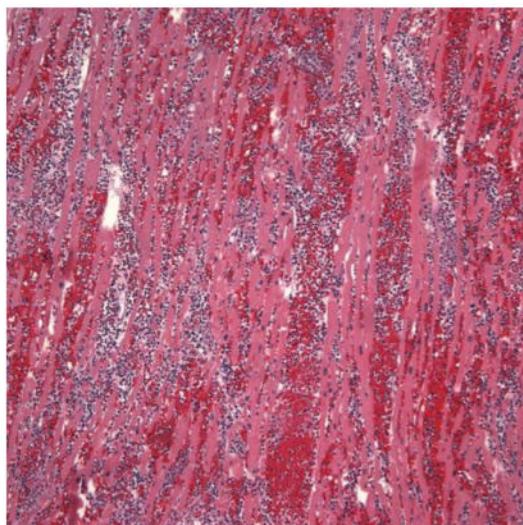
(note the white colored spaces)

"small, cleft-like spaces that become filled with amorphous ECM"

(these white spaces are not present in normal media of the arterial wall)

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07:47



### UWORLD IMAGES

07:47

"mononuclear inflammatory infiltrates" (blue colored)

+

"myofibrillary necrosis" (Dark Red colored)

seen in "VIRAL MYOCARDITIS"

S/S :-

usually self-limiting

but

(in minority) may progressive with long term complications

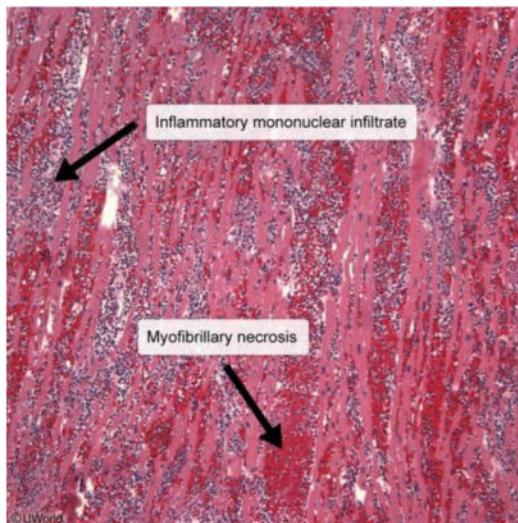
complications aren't symptomatic until advanced stages of decompensated HF.

1. Dilated Cardiomyopathy (systolic HF)

2. may lead to SCD (due to ventricular arrhythmia)

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07:47



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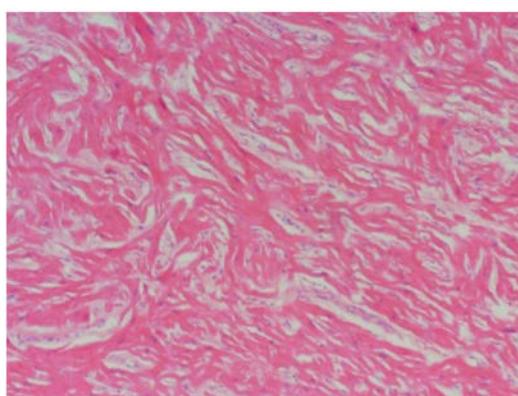
07:47

seen in Viral Myocarditis

(adeno, influenza, coxsackie B, parvo, HIV, HHV-6)

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07:47



**UWORLD IMAGES**

07:47

cardiomyocyte hypertrophy with haphazard cellular arrangement (pink colored cells)

with

prominent interstitial spacing and fibrosis (white/violet colored spaces)

seen in HOCM

usually cause SCD in young pt. with exertion

but

can also cause death at rest (rare)

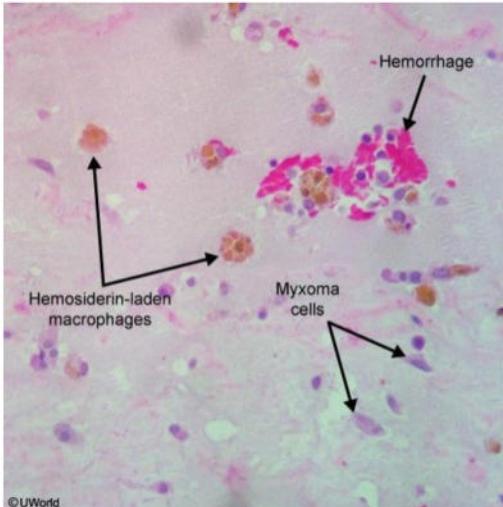
(bcz cardiomyocyte disarray and fibrosis alters spatial relationship of the intercalated discs, which are the primary mediators of organise conduction),

hence, increasing susceptibility to ventricular arrhythmias.

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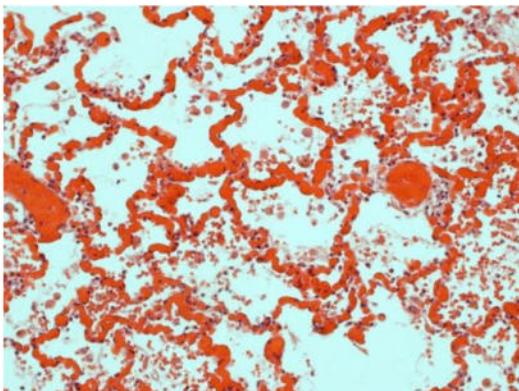
07:47

seen in cardiac Myxoma

U

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07:48



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07:48

"pink, acellular material within the alveolar space"

due to transudation of fluid plasma across the capillary–alveolar membrane.

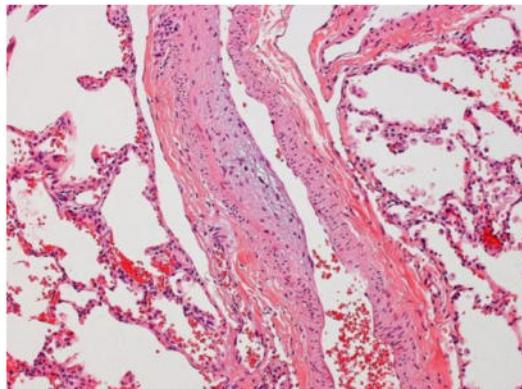
seen in Pulmonary Edema

U

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**UWORLD IMAGES**

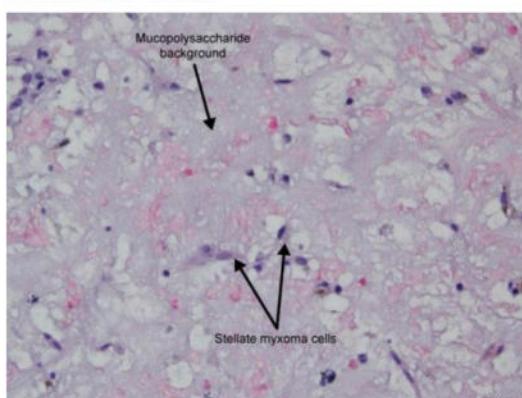
07:48

hypertrophy of pulmonary vascular smooth muscle, seen in Pulmonary arterial HTN.

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07:48



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07:48

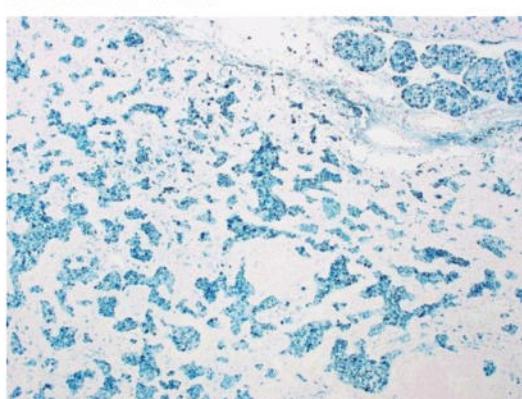
seen in Cardiac Myxoma (M/C primary cardiac tumor)

originating 90% from LA > RA

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"blue colored cytoplasmic granules" on prussian blue staining.

Siderophages (alveolar macrophages laden with Iron)

Siderophages "when stained with H&E stain) --> golden-brown cytoplasmic granules.

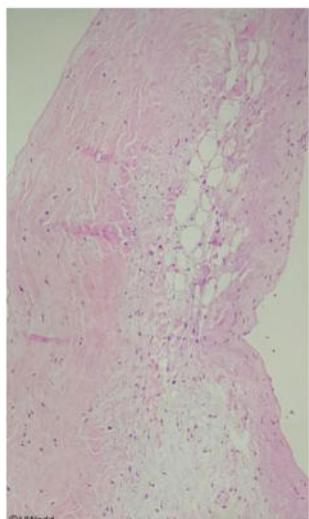
Prussian Blue stain --> detects Intracellular Iron.

(contains "colorless" potassium ferrocyanide which in the presence of iron gets converts to "blue" colored Ferric Ferrocyanide"

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cystic medial degeneration.

increases the risk for aortic dissection.

medial degeneration --> collagen, elastin, smooth muscle are lost

with aging.

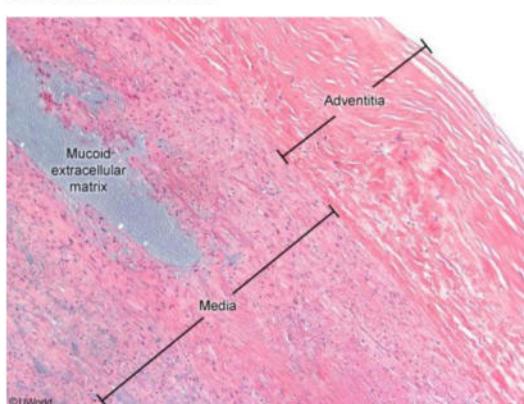
cystic --> and are replaced by basophilic mucoid extracellular matrix

+ collection of mucopolysaccharide.

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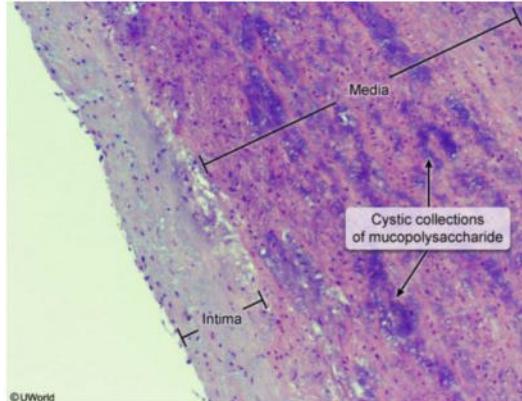
normal finding with aging.

but increases the risk for Aortic Dissection.

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with aging there is destruction of collagen and elastic tissue

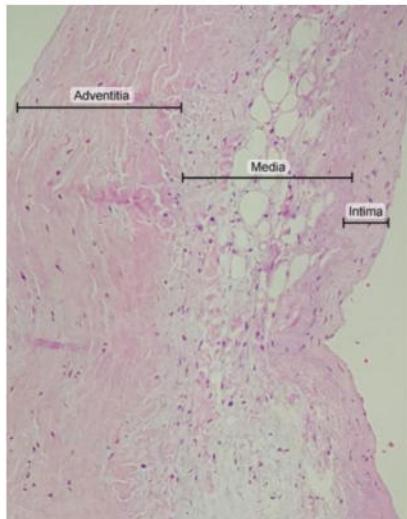
typically in "basket-weave pattern" --> cysts (which are filled with mucopolysaccharide".

predisposing for Aortic Dissection.

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Cystic Medial Degeneration of Aorta.

causes Aortic Dissection.

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07:50

Atherosclerosis :-

Intima affected primarily --> Acute Coronary Syndrome.

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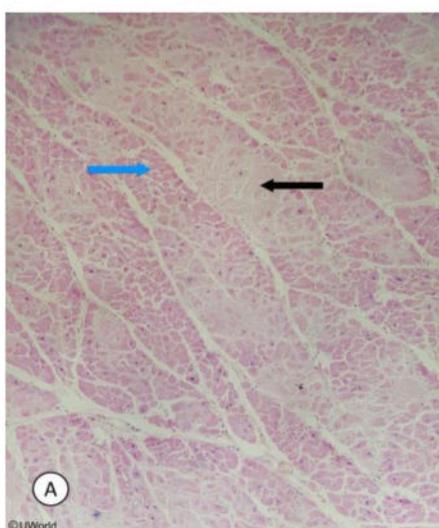
### UWORLD IMAGES

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Calcified Atherosclerotic Plaque"

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### UWORLD IMAGES

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blue arrow --> "normal cardiac myocytes"

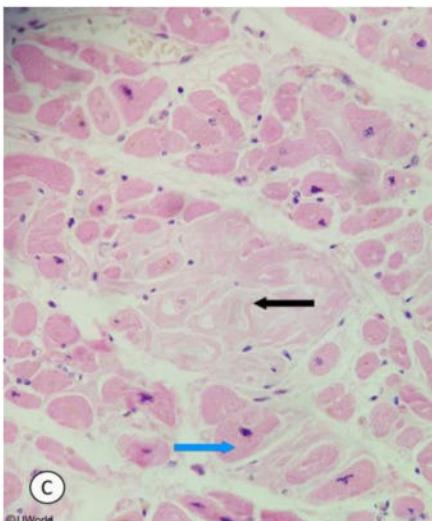
black arrow -->

amorphous  
acellular  
extra-cellular insoluble pink material deposition.

seen in Cardiac Amyloidosis.

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### UWORLD IMAGES

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Blue Arrow "normal cardiac cells"

Black Arrow

"acellular" + "amorphous" + "extracellular" deposition of insoluble pink materials which are insoluble proteins :-

1. monoclonal light chain (AL Amyloidosis)
2. mutated transthyretin (familial ATTR Amyloidosis)
3. wild-type transthyretin (senile systemic Amyloidosis)

Amyloidosis causes Restrictive/ Infiltrative Cardiomyopathy.

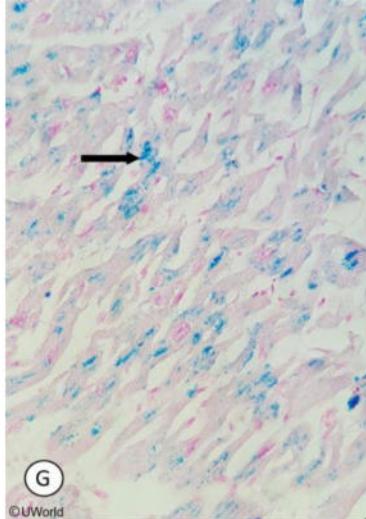
normally in this type --> LV thickness is normal.

BUT

in amyloidosis --> LV Thickness is INCREASED

### UWORLD IMAGES

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### UWORLD IMAGES

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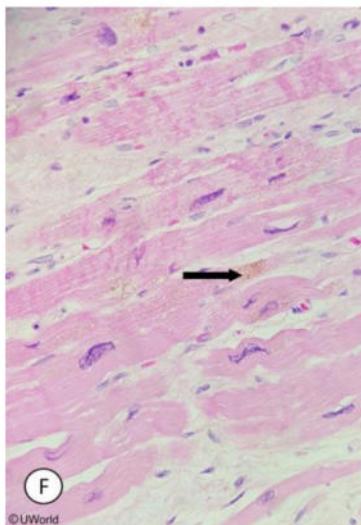
Prussian Blue Staining of Cardiac Myocytes showing excessive iron deposition in hemochromatosis

causes dilated > restrictive cardiomyopathy.

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excessive iron deposition in myocardial fibers in Hemochromatosis.

(H&E Stain)

can be confirmed by Prussian Blue Stain.

classically causes Dilated >> Restrictive Cardiomyopathy.

DD:-

lipofuscin --> lipo + fuscin (normal wear and tear/aging)

insoluble pigment composed of

"lipid polymers and protein-complexed phospholipids"

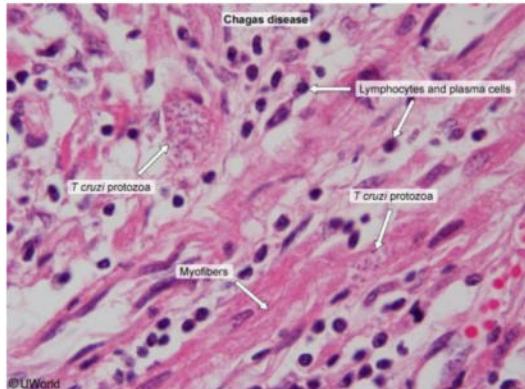
can be seen in aging heart and liver / in cachectic, malnourished pt. as

"intracytoplasmic granules that are tinged yellowish-brown"

due to free radicle injury and lipid peroxidation.

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07:53



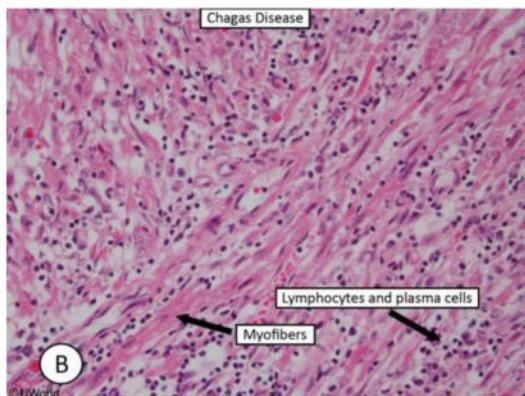
**UWORLD IMAGES**

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causes myocarditis and dilated cardiomyopathy in Latin America.

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07:54



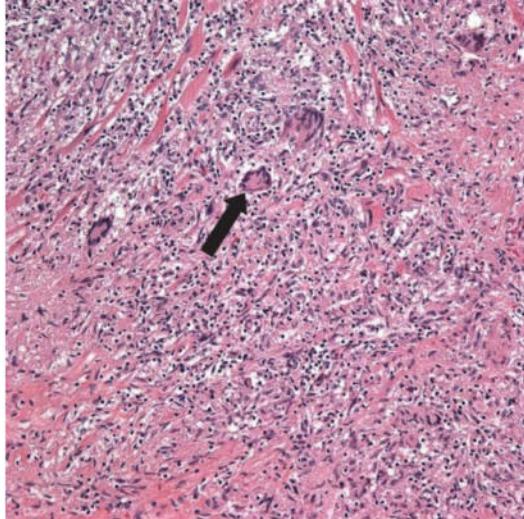
**UWORLD IMAGES**

07:54

causes myopericarditis and "Dilated Cardiomyopathy".

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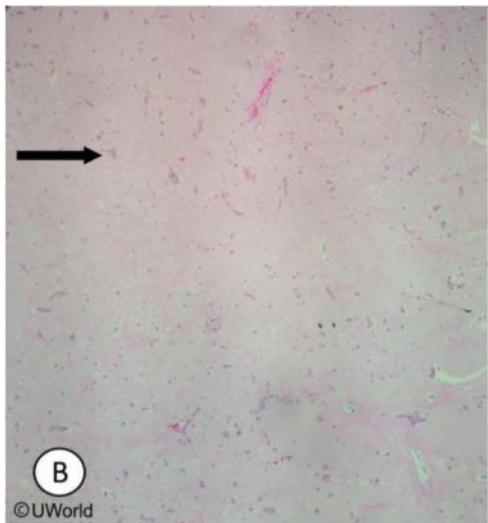
"non-caseating granuloma" seen in Cardiac Sarcoidosis.

classically causes :-

Restrictive/Infiltrative Cardiomyopathy

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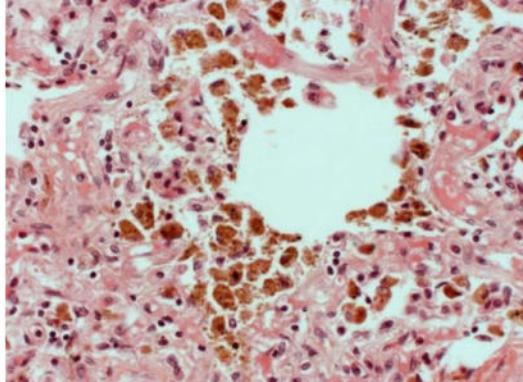
"bland stellate cells" in a background

of myxoid (mucopolysaccharide) ground substance.

seen in Cardiac Myxoma.

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### UWORLD IMAGES

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"abundance of alveolar macrophages filled with brown pigment in the lung parenchyma"

Heart Failure Cells.

due to chronic LV dysfunction --> elevated pulmonary venous pressure  
-->

transudation of fluid across the alveolar–capillary membrane -->  
Pulmonary Edema -->

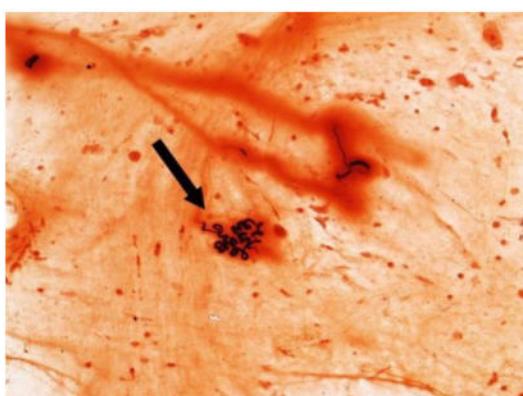
breaks in the endothelium and extravasation of the RBC into the alveoli and the lung parenchyma.

Pt. with severe left ventricular diastolic dysfunction is associated with increased risk of Ventricular Arrhythmia (V. Tac, V. Fibrillation)

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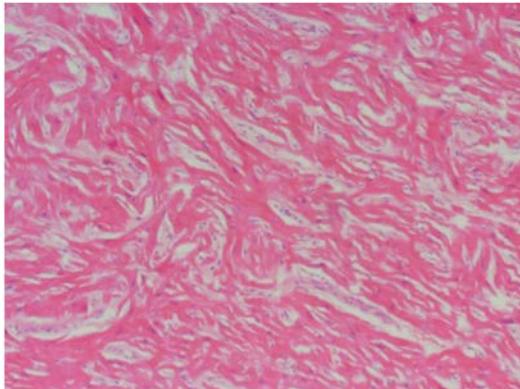
Curschmann spirals  
(spiral shaped mucus plugs)

seen in asthma  
(hyperreactivity of airways to environmental allergens)

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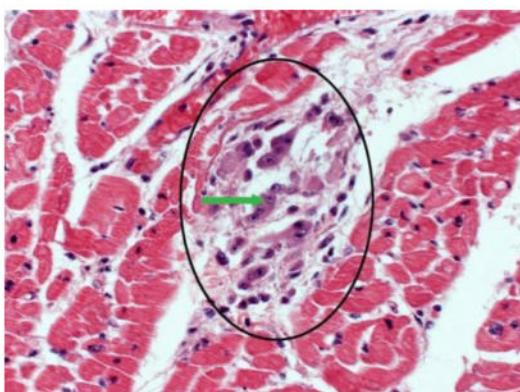
"disorganised, hypertrophic myocytes" seen in HCM.

genetic mutation involving Sarcomere genes.

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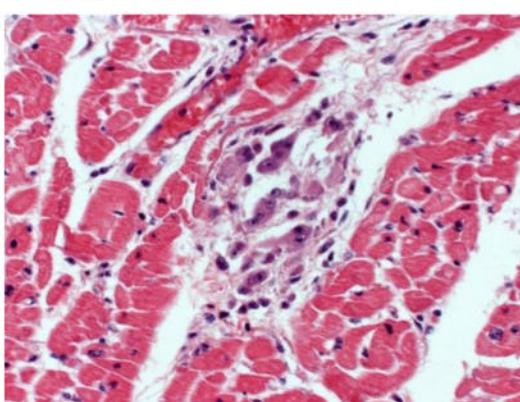
"interstitial fibrosis with central lymphocytes and macrophages as well as multinucleate giant cells (green arrow)"

Aschoff Body or Interstitial Myocardial granuloma.

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"interstitial fibrosis with lymphocytes and macrophages as well as

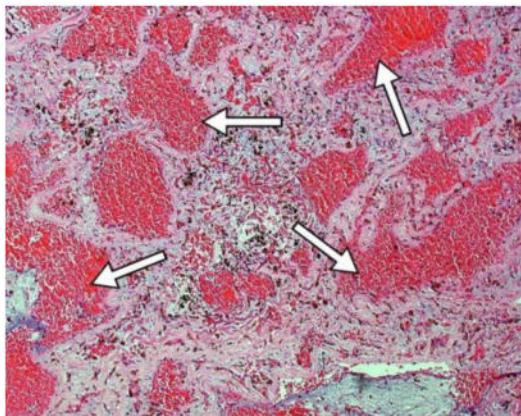
scattered MNG cells (in the centre)"

Aschoff Body are pathognomonic of ARF-myocarditis.

they may also contain specialised macrophages --> Anitschkow cells.

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07:56



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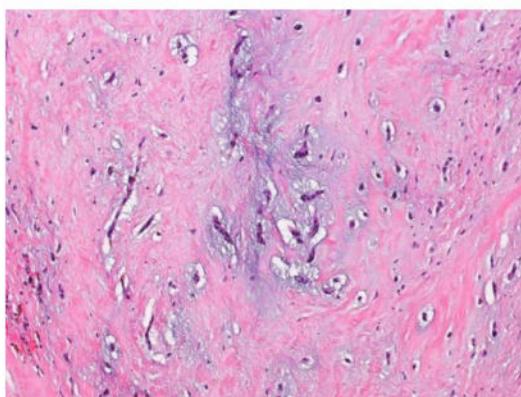
"regions of angiogenesis" in Atrial Myxoma

due to excess secretion of VEGF

may hemorrhage.

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07:56

"scattered cells within a mucopolysaccharide stroma"

seen in Atrial Myxoma

M/C tumor of the heart. 80% from the LA.

tumor secretes :-

1. VEGF (large amounts) --> angiogenesis -->hemorrhaging and friability

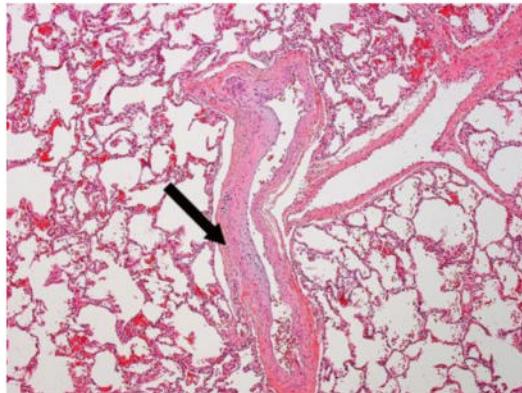
2. Cytokines (IL-6 ; small amounts) -->  
Constitutional symptoms (Fever and weight loss)

mimicks Murmur of MS

Mobile Mass --> hence, transient, positional obstructive symptoms (i.e.  
Upright > Lying), dyspnea, lightheadedness or syncope

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"media and intimal hyperplasia"

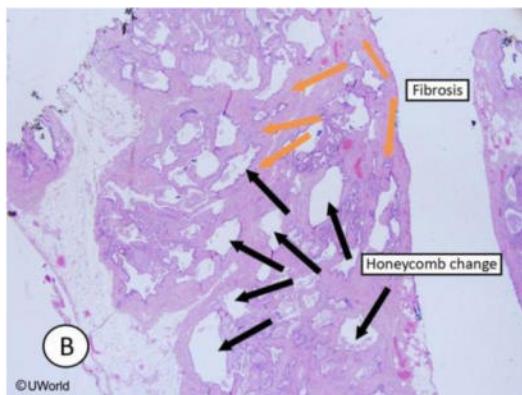
seen in Pulmonary vascular hypertension

treatment with –

1. Endothelin Receptor antagonist – Bosentan, ambrisentan
2. PDE 5 Inhibitor --> inc. cGMP --> smooth muscle relaxation
3. PGI2 analogue – epoprostenol

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idiopathic pulmonary fibrosis

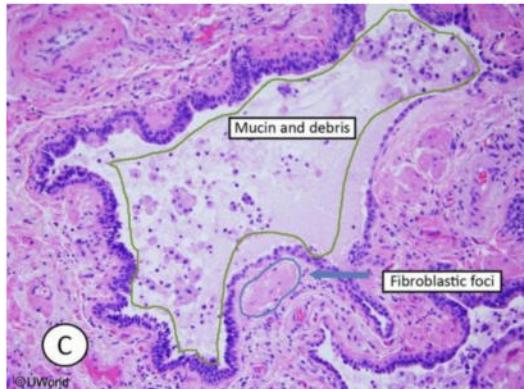
therapy for slowing down progression :-

Pirfenidone (TGF-beta)

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07:57



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idiopathic pulmonary fibrosis

treatment therapy include

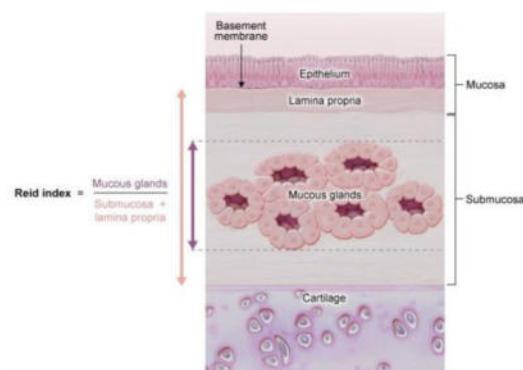
Perfenidone inhibits TGF-beta

Nintedanib (tyrosine kinase inhibitor) against VEGF, PDGF, FGF

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pathological tool.

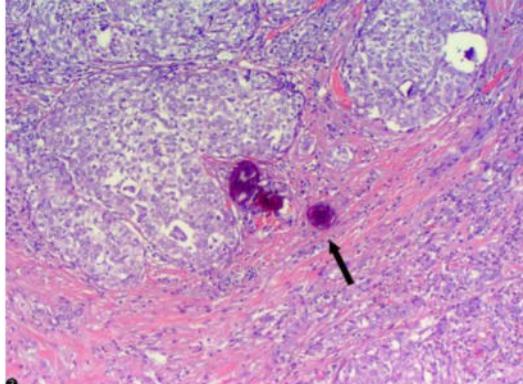
thickness of the sub-mucosal glands to the thickness of the bronchial wall B/W the epithelial BM and the bronchial cartilage.

normal Reid's index is 0.4.

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### UWORLD IMAGES

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"laminated calcum deposits" Psammoma Bodies

seen in :-

1. Papillary thyroid cancer
2. Serous cystadenoma of ovary
3. Mesothelioma
4. Meningioma

PTC :-

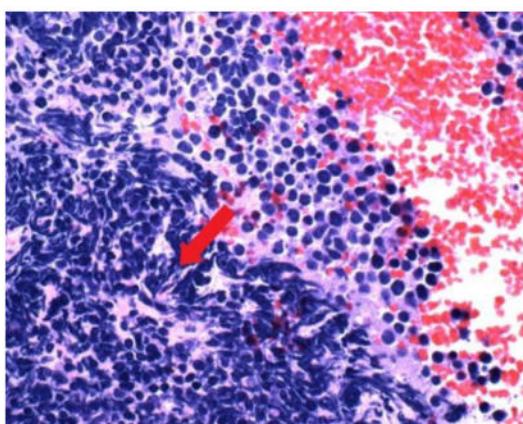
pr. with thyroid nodule, cervical LAD but can also as anterior Mediastinal mass

[DD:-

Hodgkin's / Central Lung tumor (Sq./Small – cell)]

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07:58



### UWORLD IMAGES

07:58

"small round/ovoid cells with scant cytoplasm" seen in

SMALL cell / OAT cell Cancer of Lung.

[neuroendocrine origin;  
hence, cell marker :-

NCAM (CD 56) +ve

NSE

chromogranin

synaptophysin]

SIADH, Lambert–Eaton Syndrome, ACTH

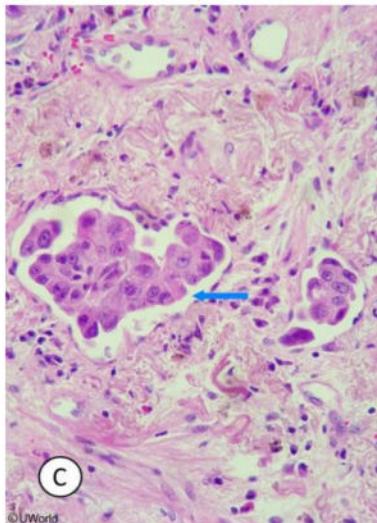
Central Location (DD)  
(mediastinal mass)

Oat cell cancer of lung/ Sq cell cancer  
Hodgkin's Lymphoma  
Papillary thyroid cancer.

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"glandular type cells" seen in adenocarcinoma of the lung.

cells with abundant cytoplasm and eccentrically placed nuclei.

often stain positive for mucin.

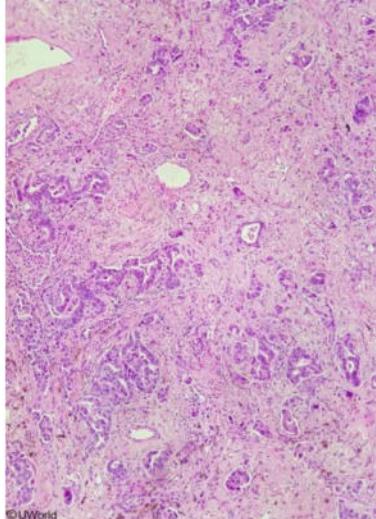
typically arises in the periphery of the lung.

M/C type of cancer in non-smoker and in total population.

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07:58



### UWORLD IMAGES

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"glandular pattern" of growth seen in adenocarcinoma of Lung.

M/C type of Lung cancer in USA.

arises from the alveolar epithelium.

characterised by invasive cells with abundant cytoplasm and eccentrically placed nuclei that form irregular glandular elements.

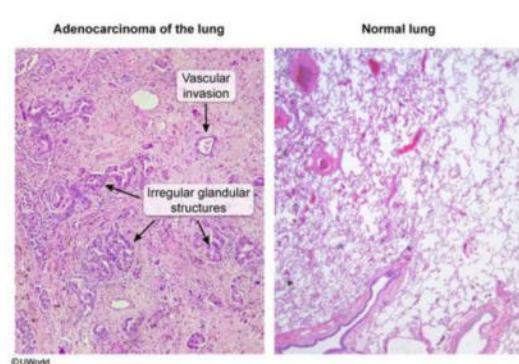
mucin production is common, hence pt. may present with copious amount of watery sputum (bronchorrhea)

usually in the peripheral region;

"discrete mass" or "pneumonia like consolidation"

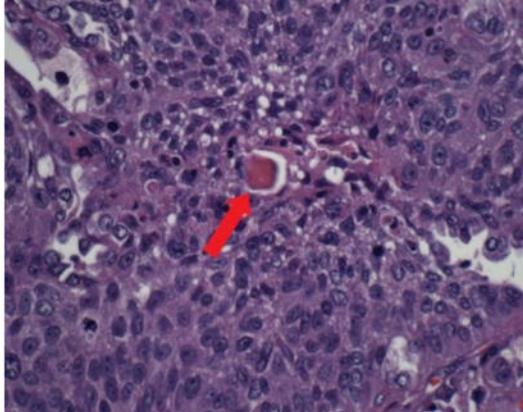
### UWORLD IMAGES

07:59



### UWORLD IMAGES

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#### UWORLD IMAGES

07:59

keratin pearls" characteristic features of Squamous Cell cancer

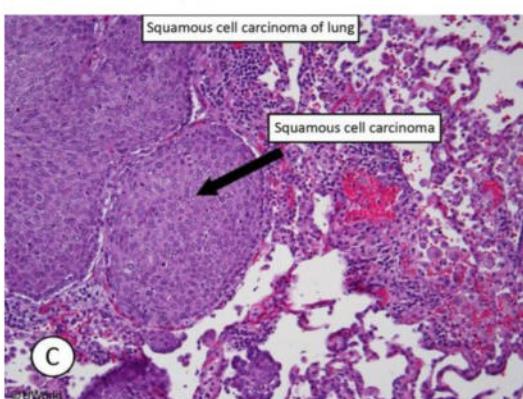
"intercellular bridges" are also seen

ass. with smoking.

usually arising centrally, form the main bronchus.

#### UWORLD IMAGES

07:59



#### UWORLD IMAGES

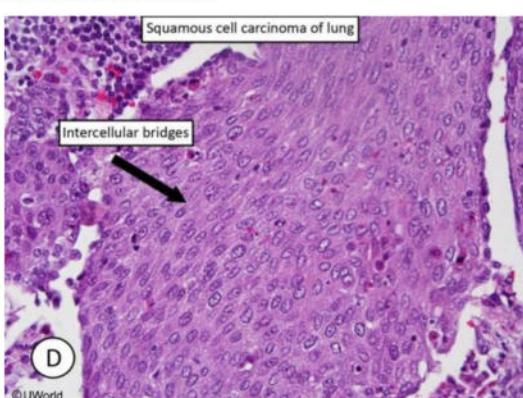
07:59

typically arises centrally within a bronchial lumen.

squamous cells with "intercellular bridges" and "keratin pearls"

#### UWORLD IMAGES

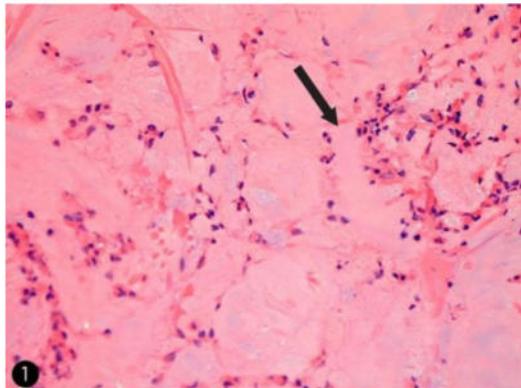
07:59



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07:59



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**UWORLD IMAGES**

08:00

Charcot Leyden crystals (bipyramidal – shaped accumulation of the eosinophil membrane protein)

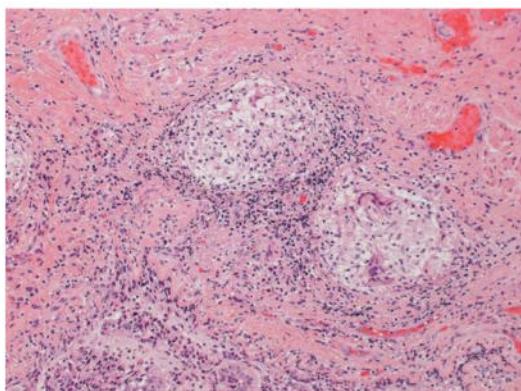
or

Crystalloid Masses on sputum microscopy

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**UWORLD IMAGES**

08:00



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**UWORLD IMAGES**

08:00

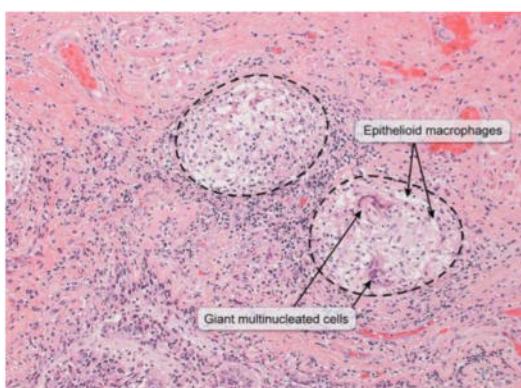
Non-Caseating Granuloma seen in Sarcoidosis.

(made up of epithelioid macrophages and MN giant cells)

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**UWORLD IMAGES**

08:00



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## UWORLD IMAGES

seen in

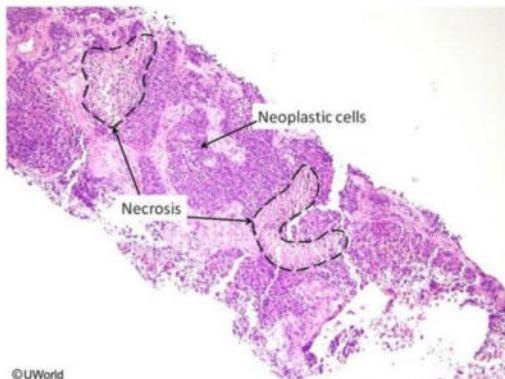
08:00

Sarcoidosis

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## UWORLD IMAGES

08:00



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## UWORLD IMAGES

08:01

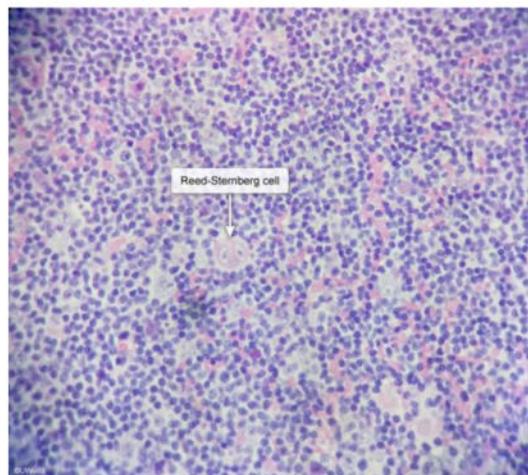
Small Cell Carcinoma (ass with smoking)

"small cells with scant cytoplasm"

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## UWORLD IMAGES

08:01



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## UWORLD IMAGES

08:01

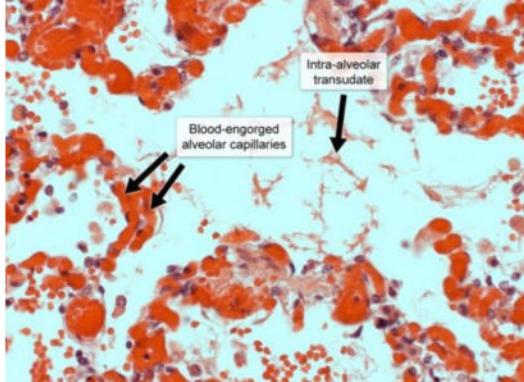
presents with Cervical LAD

+ B symptoms (fever, night sweats, wt. loss)

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## UWORLD IMAGES

08:01



**UWORLD IMAGES**

08:01

in acute pulmonary edema -->

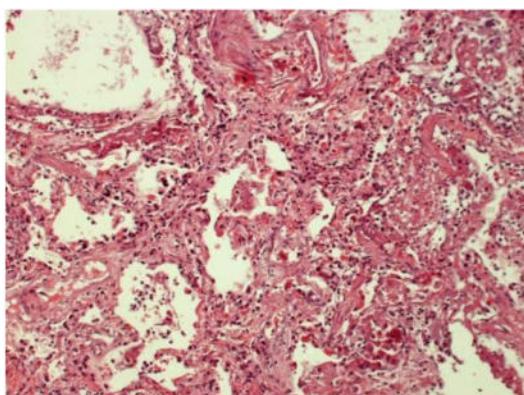
blood engorged alveolar capillaries -->

increased hydrostatic pressure -->

intra alveolar transudate appearing as acellular pink material.

**UWORLD IMAGES**

08:01



**UWORLD IMAGES**

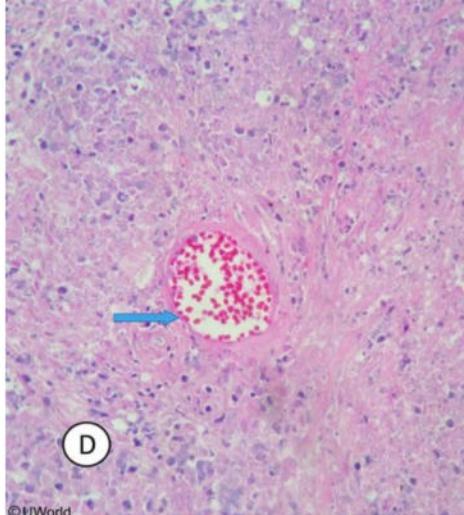
08:01

patchy interstitial fibrosis seen in

Idiopathic Pulmonary fibrosis

**UWORLD IMAGES**

08:01



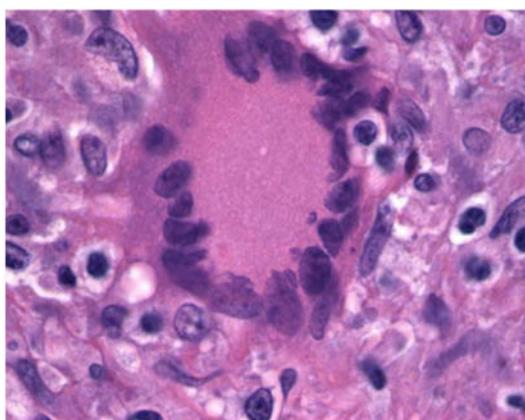
**UWORLD IMAGES**

08:01

Necrotizing arteritis with  
adjacent palisading epitheloid histiocytes  
(granulomatosis with polyangiitis)

**UWORLD IMAGES**

08:02



**UWORLD IMAGES**

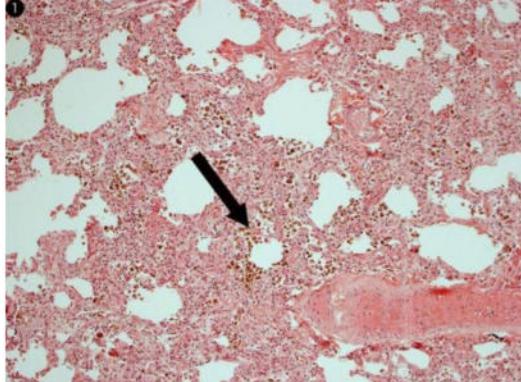
08:02

multi-nucleated giant cells surrounding caseating granulomas.  
characteristic of :-

TB, Leprosy,  
Fungal (histoplasmosis)

**UWORLD IMAGES**

08:02



**UWORLD IMAGES**

08:02

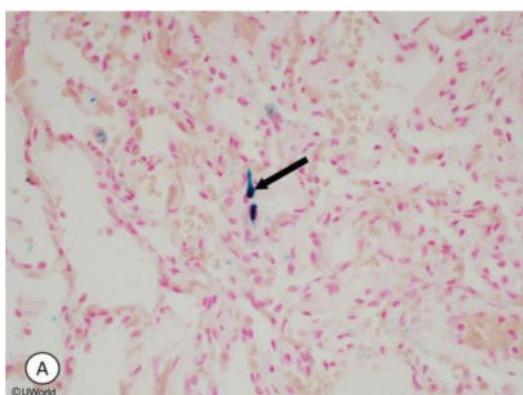
Heart Failure cells (hemosiderin laden macrophages)

macrophages digest RBCS that leak from alveolar capillaries damaged by high intravascular pressure.

sign of Chronic Lung Congestion.

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08:02



**UWORLD IMAGES**

08:02

Asbestos body (ferruginous bodies)

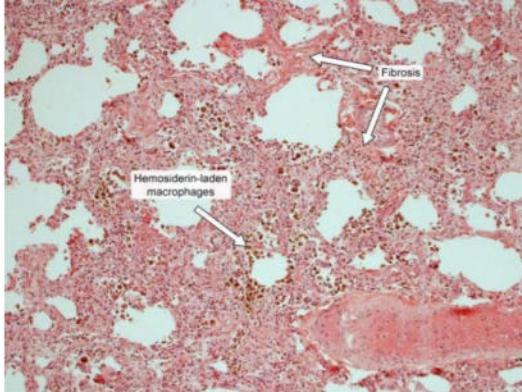
embedded within interstitial fibrous tissue.

represent foreign (hydrated Mg silicate) particles enclosed in Iron-rich material and typically appear as brown "barbell" shaped on Prussian Blue staining.

**UWORLD IMAGES**

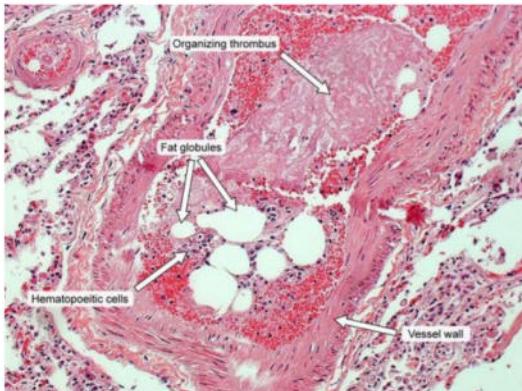
08:03

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### UWORLD IMAGES

08:03



### UWORLD IMAGES

08:03

seen in alveoli of a pt. with FAT EMBOLISM SYNDROME.

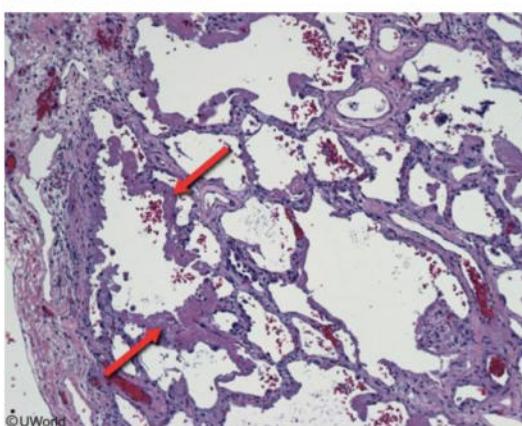
bone fracture --> bone marrow cells enter systemic circulation -->  
aggregate with platelets --> formation of microthrombi -->

damage alveolar functioning,

enter systemic circulation (rash, confusion)

### UWORLD IMAGES

08:03



### UWORLD IMAGES

08:03

"diffuse waxy hyaline membrane lining the alveoli"

in ARDS.

(consisting of :-

1. fibrin exudate
2. inspissated (thick) protein-rich edema fluid
3. remnants of necrotic epithelial cells)

pancreatitis --> interstitial and intra-alveolar edema --> alveolar inflammation --> damage due to neutrophils.

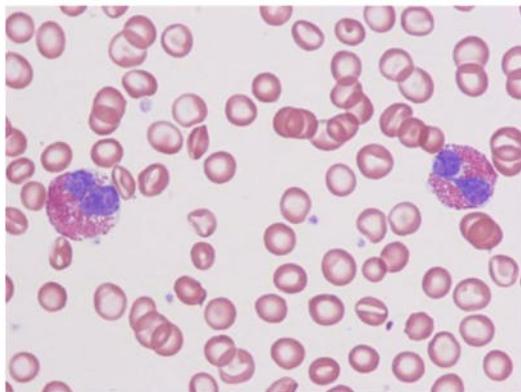
in Decompensated HF --> cardiogenic pul. edema --> Transudative edema (due to increased hydrostatic pressure)

in DIC & ARDS --> Alveolar hemorrhage --> hemoptysis.

caused by pancreatitis

**UWORLD IMAGES**

08:03



**UWORLD IMAGES**

08:03

" cells bilobed nuclei and containing eosinophilic granules in the cytoplasm"

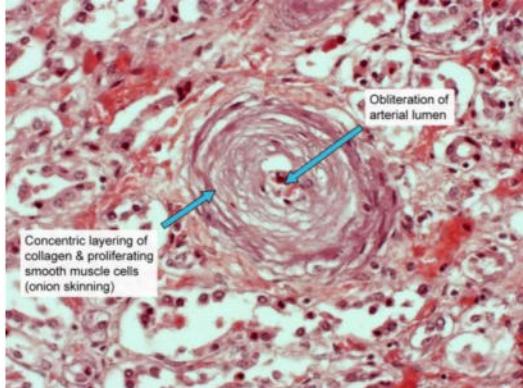
**EOSINOPHILS**

these granules predominantly contain MBP (Major Basic Protein) which is toxic for

helminth (Ascaris) and  
damages epithelial and endothelial cells in the lungs (hence,  
responsible for chronic lung diseases like asthma)

**UWORLD IMAGES**

08:04



Next messages

# UWORLD IMAGES

## Previous messages

6 April 2020



**UWORLD IMAGES**

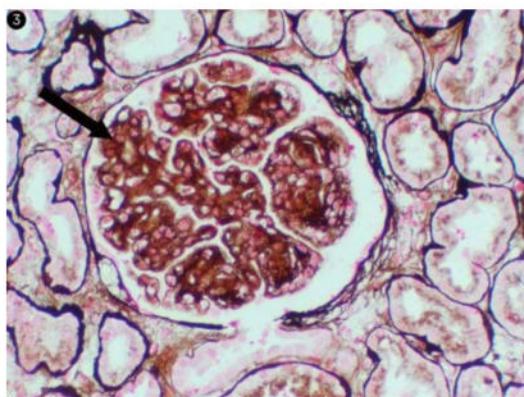
08:04

seen in malignant HTN.



**UWORLD IMAGES**

08:04



**UWORLD IMAGES**

08:04

"hypercellular glomeruli with thickening of GBM"

seen in Membrano Proliferative GN

due to subendothelial immune complex deposition.

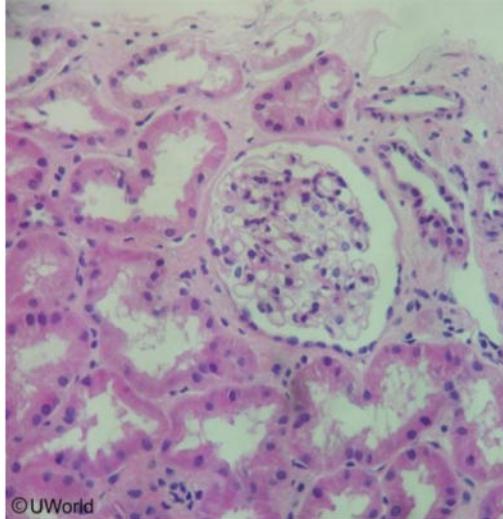
common causes :-

hepatitis B, C



**UWORLD IMAGES**

08:04



#### UWORLD IMAGES

08:04

"re-epithelialisation of tubular epithelium"

corresponds to phase 3 (recovery) of AKI.

During this phase,

the GFR returns to normal as soon as the tubular casts (obstructing the tubules) are cleared.

But Tubular epithelialisation which takes time (occurs gradually).

hence, the patient is at risk of el. abnormalities and transient polyuria.

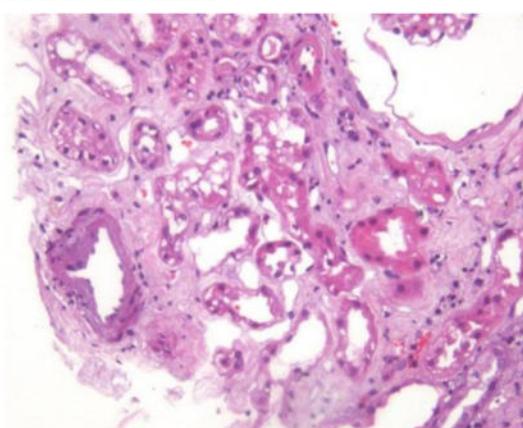
a small percentage of pts. where re-epithelialisation does not occur,

then,

foci of interstitial fibrosis causing "medullary scarring and atrophy" can be seen on LM.

#### UWORLD IMAGES

08:04



#### UWORLD IMAGES

08:05

renal tubular damage in acute kidney injury.

"interstitial edema"

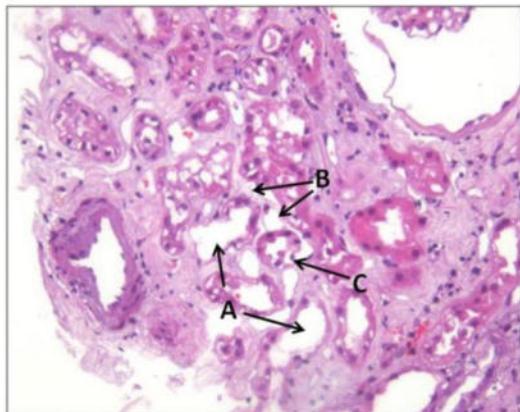
"epithelial cell vacuolisation"

"loss of tubular columnar epithelium and damaged basement membrane"

U

### UWORLD IMAGES

08:05



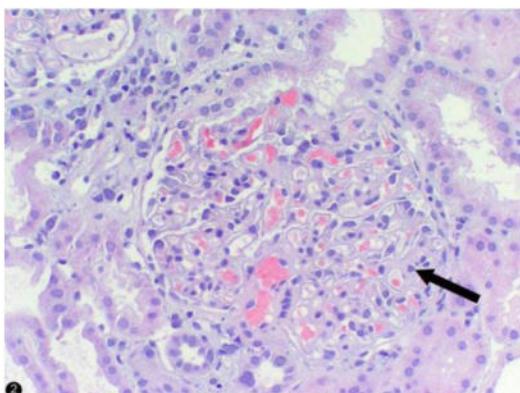
Acute tubular necrosis, H&E stain.

(A) Loss of columnar epithelium, denudation of basement membrane; (B) interstitial edema; (C) epithelial cell vacuolization.

U

### UWORLD IMAGES

08:05



U

### UWORLD IMAGES

08:05

"note the uniformly diffuse thickening of glomerular capillary walls" seen in Membranous nephropathy.

common cause of nephrotic syndrome in adults.

Primarily due to :-

idiopathic (anti-phospholipase A2 Ab)

Secondarily due to :-

Drugs :- NSAIDs, penicillamine, gold

Auto-Immune :- SLE, RA

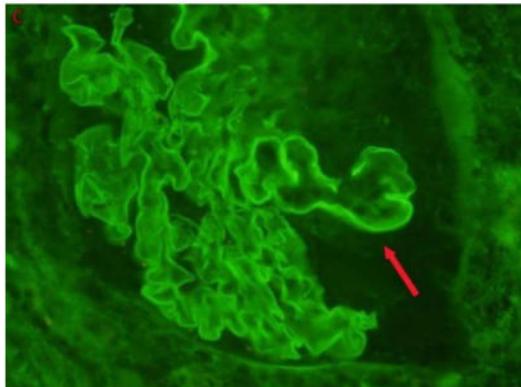
Infections :- hepatitis B, C

Solid tumors :- colon ca, melanoma, lung ca.

U

UWORLD IMAGES

08:05



U

UWORLD IMAGES

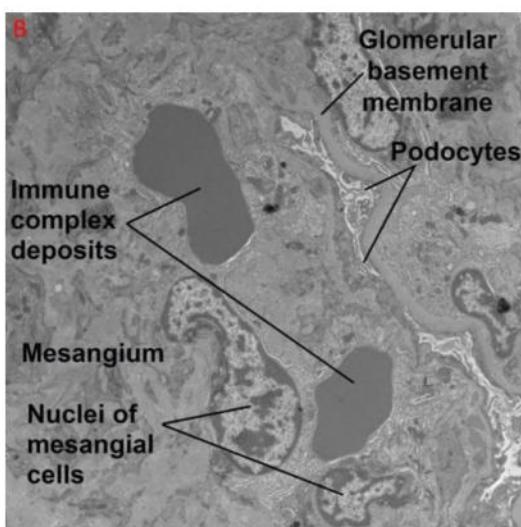
08:05

"note the linear IF due to deposition of C3 and IgG" seen in anti-GBM disease.

U

UWORLD IMAGES

08:05



U

UWORLD IMAGES

08:06

"IgA deposition in the mesangium"

IgA Nephropathy (LOCALISED VASCULITIES) presents as :-

recurrent painless spontaneous HEMATURIA following URTI

additionally,

HSP

in children b/w 2-10 years, (SYSTEMIC VASCULITIS)

nonthrombocytopenic palpable purpura (classically in buttocks and lower limb)

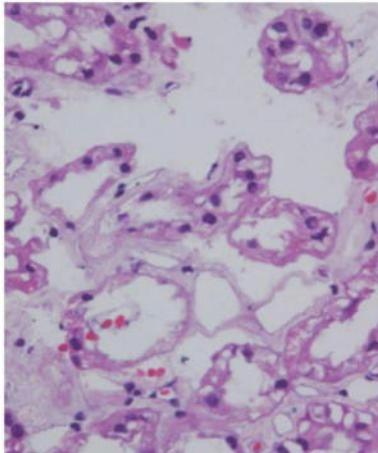
arthralgias

abdominal pain (increased risk of intussusception)

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UWORLD IMAGES

08:06



U

UWORLD IMAGES

08:06

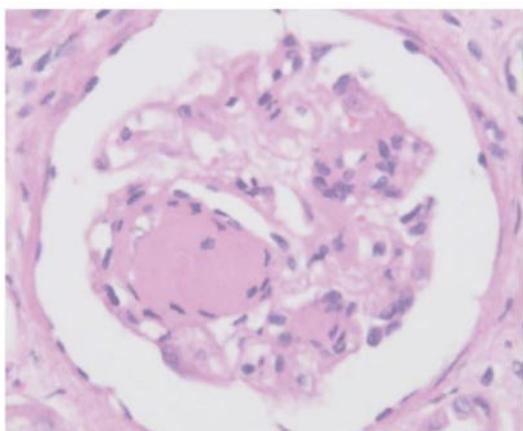
note the damaged tubules

found in AKI.

U

UWORLD IMAGES

08:06



U

UWORLD IMAGES

08:06

"kimmelstein wilson nodule" seen in Diabetic Nephropathy

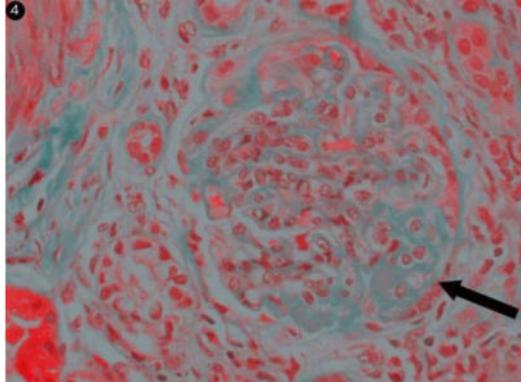
progressive GBM thickening and mesangial proliferation -->

capillary compression --> decreased GFR

U

UWORLD IMAGES

08:06



U

### UWORLD IMAGES

08:06

"segmental sclerosis" (stained with Jones Silver) FSGS

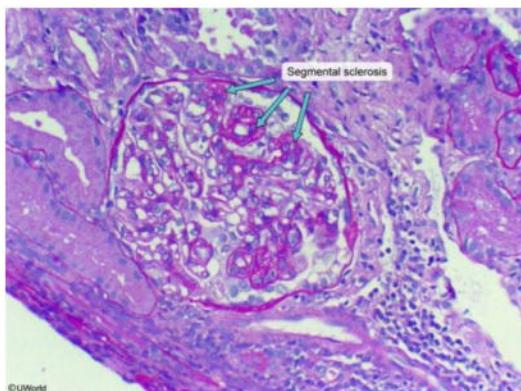
EM :-

effacement of foot processes

U

### UWORLD IMAGES

08:07



U

### UWORLD IMAGES

08:07

LM :- (diagnostic)

"segmental glomerulosclerosis" (stained with PAS)

FSGS :-

is usually found in  
HIV pt,  
heroin abuse,  
massive obesity,  
IFN Rx,

IF :-

-ve

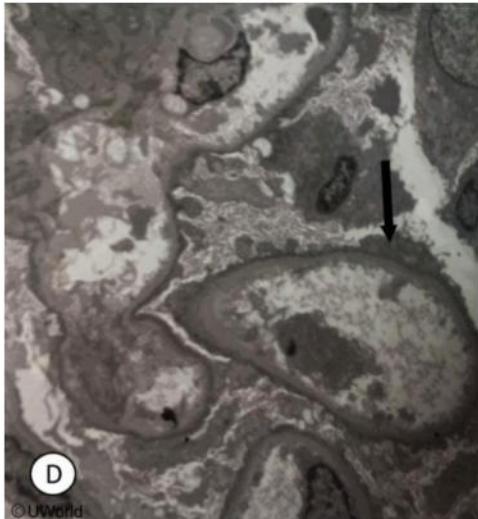
EM :-

effacement of foot processes

### UWORLD IMAGES

08:07

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**UWORLD IMAGES**

08:07

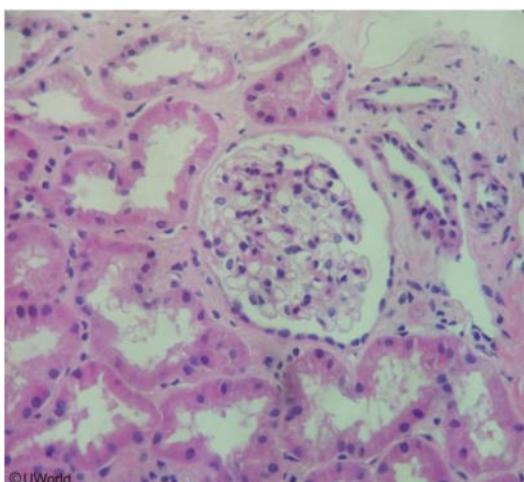
"fusion and effacement of Podocyte Foot Processes"

seen in MCD

U

**UWORLD IMAGES**

08:07



U

**UWORLD IMAGES**

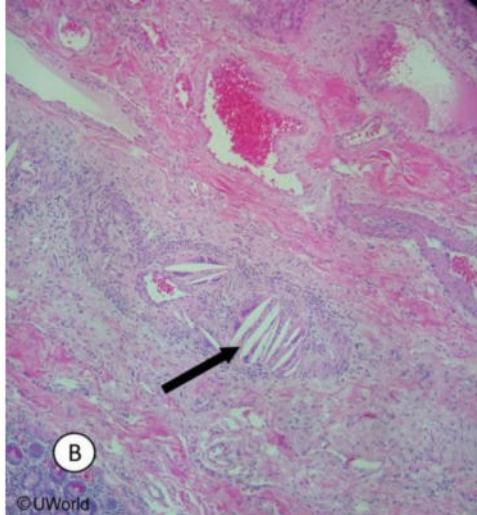
08:07

normal glomerulus

U

**UWORLD IMAGES**

08:07



U

## UWORLD IMAGES

08:07

"needle shaped cholesterol clefts"

pt. with h/o invasive surgery/ procedure -->

mechanical disruption of atherosclerotic cholesterol plaques in large blood vessels -->

embolic shower into circulation -->

1-3 week later presentation with S/S of obstruction (typically in) :-

SMALL INTESTINE – small bowel ischemia/ obstruction

SKIN/ LL – livido reticularis/ blue toe syndrome

CNS – stroke/TIA

EYES – amaurosis fugax/ Retinal Ischemia

KIDNEY – AKI

U

## UWORLD IMAGES

08:07



U

## UWORLD IMAGES

08:07

PYURIA – UTI

**WHITE BLOOD CELL CASTS – AIN/ acute pyelonephritis  
(characteristic of intra-renal Inflammation/ Infection)**

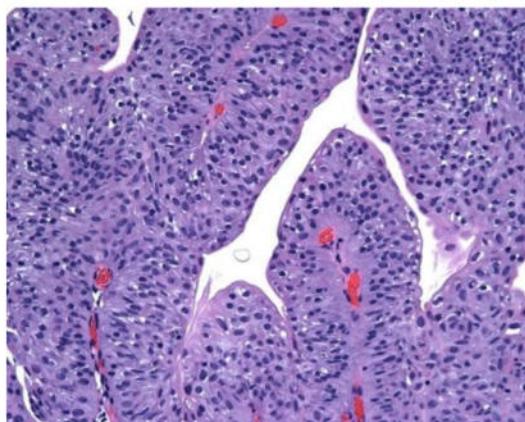
**UWORLD IMAGES**

08:08

Nephrolithiasis					
Content	Frequency	Radiograph opacity	pH	Microscopic appearance	
Calcium oxalate	70%-80%	++	-	 • Octahedron (square with an "X" in the center)	
Calcium phosphate			>7.0	• Elongated, wedge-shaped • Forms rosilles	
Magnesium ammonium phosphate (struvite or triple phosphate)	15%	+	>7.0	 • Rectangular prism ("coffee-bean")	
Uric acid	5%	-	<7.0	 • Yellow or red-brown, diamond or rhombus	
Cystine	1%	+	<7.0	 • Flat, yellow, hexagonal	

**UWORLD IMAGES**

08:08



**UWORLD IMAGES**

08:08

"papillary organisation with central fibrovascular stalk"

originating from epithelium of renal pelvis/ureter/bladder.

**UROTHELIAL CARCINOMA**

**UWORLD IMAGES**

08:08



### UWORLD IMAGES

08:08

Waxy Cast in urine (sign of advanced / CKD) :-

decreased renal mass -->

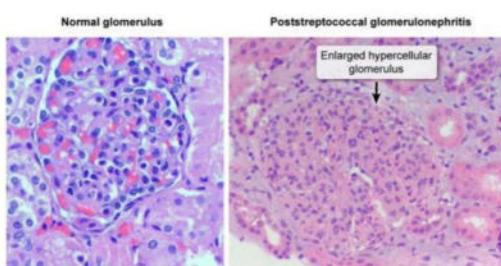
compensatory hypertrophy of the normal nephrons -->

dilated tubules of enlarged nephrons -->

SHINY/ Translucent Cast

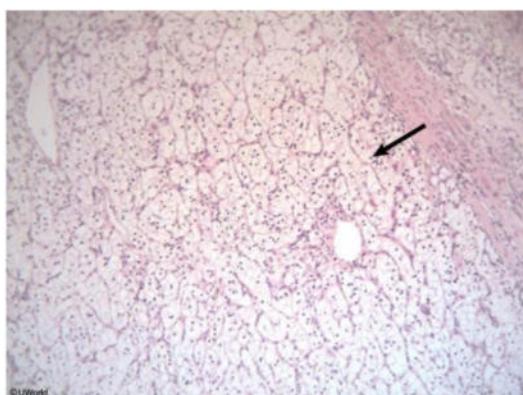
### UWORLD IMAGES

08:09



### UWORLD IMAGES

08:09



### UWORLD IMAGES

08:09

"round/polygonal cells containing abundant glycogen and lipids"

when treated during tissue processing gets removed/cleared.

Clear Cell Variant of RCC.

(these cells originate from Proximal Tubular Epithelial Cells)

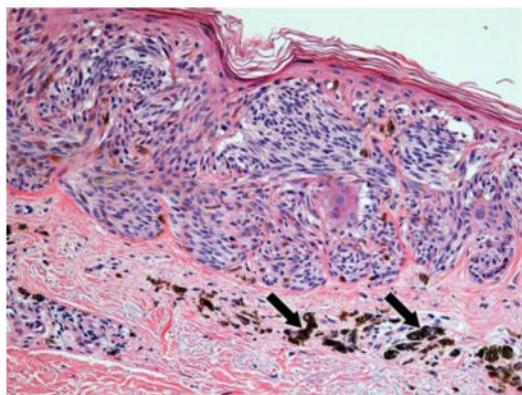
any elderly patient presenting with Gross Painless Hematuria,

initially must be suspected with Renal Cell Carcinoma (Urothelial/RCC)

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UWORLD IMAGES

08:09



UWORLD IMAGES

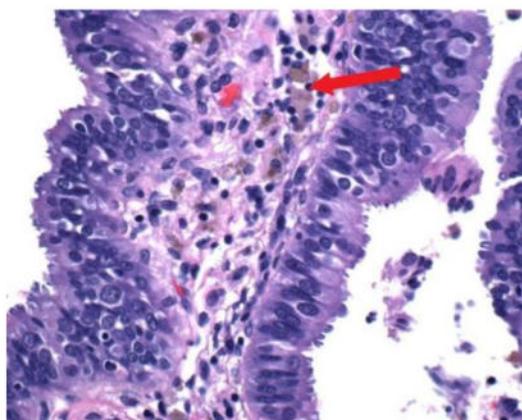
08:09

Melanin Pigments

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08:09



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08:09

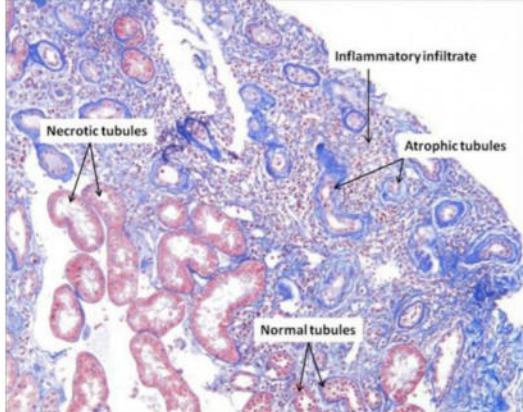
"hemosiderin pigment"

Iron Oxide containing Pigment.

U

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08:09



### UWORLD IMAGES

08:09

NSAIDs mediated Chronic Interstitial Nephritis

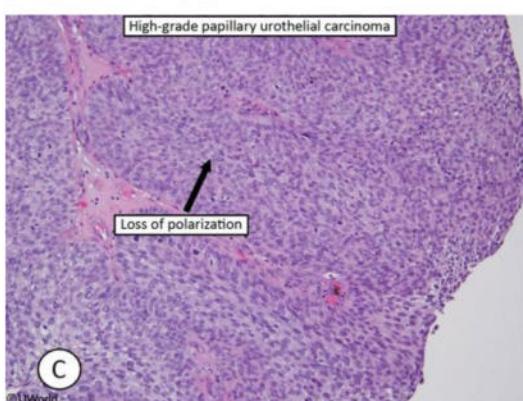
Gross,

B/L shrunken and irregular kidneys

(similar finding also seen in Chronic Pyelonephritis)

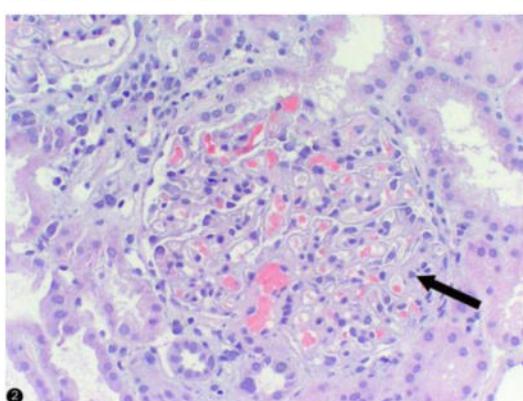
### UWORLD IMAGES

08:10



### UWORLD IMAGES

08:10



### UWORLD IMAGES

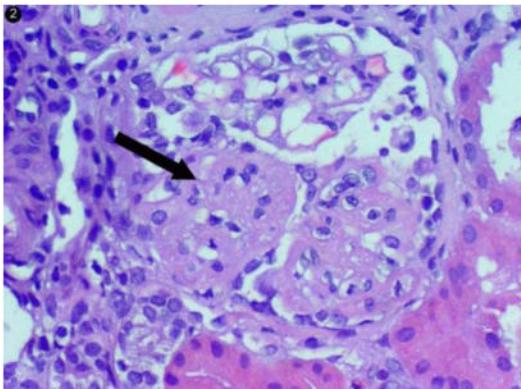
08:10

"diffuse GBM thickening" in Membranous Nephropathy.

### UWORLD IMAGES

08:10

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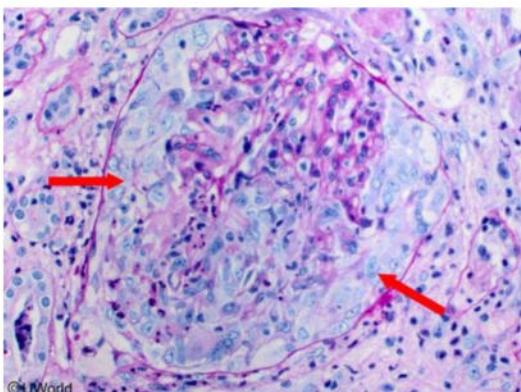
08:10

FSGN

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UWORLD IMAGES

08:11



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08:11

"crescentic Glomerulus"

anti-GBM

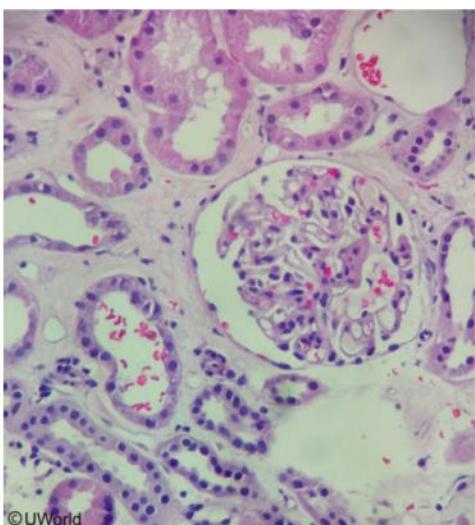
or

ANCA diseases

U

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08:11



©UWorld

## normal Glomerulus

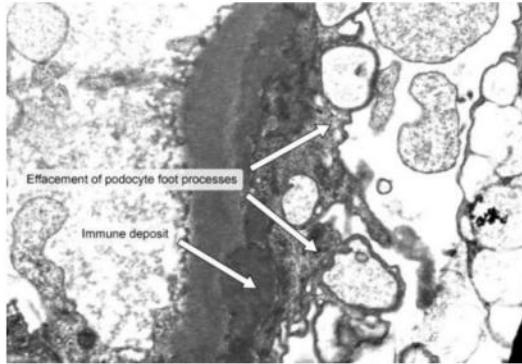
seen in MCD (M/C cause of GN in Children)

can be caused due to :-

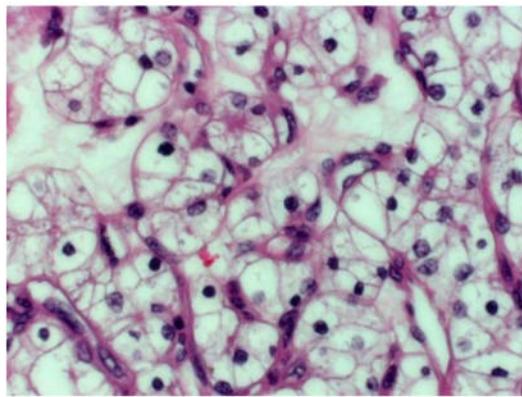
defective T-Cell function --> Glomerular Permeability Factor synthesis  
--> Damage to Podocyte Foot Processes --> loss of -ve charge -->  
selective proteinuria

Primary :- Idiopathic

Secondary :- Infection, Immune Stimulus, Vaccination, NSAIDs



## Membranous Nephropathy



## (Clear) type of RCC :-

"note the clear cytoplasm of the cells"

this is due to abundant lipid and glycogen accumulating in the cytoplasm

(during histopath treatment these content are treated resulting in empty appearing cellular cytoplasm)

**GROSS :-**

"yellow appearing fungating tumor mass"

P – PTHrp

E – EPO

A – ACTH

R – Renin

## NEOPLASTIC SYNDROMES

**S/S :-**

asymptomatic (90%)

Symptomatic (<10% ; that too in advanced cancer)

Triad of :- Painless hematuria + Flank Pain + Palpable Mass

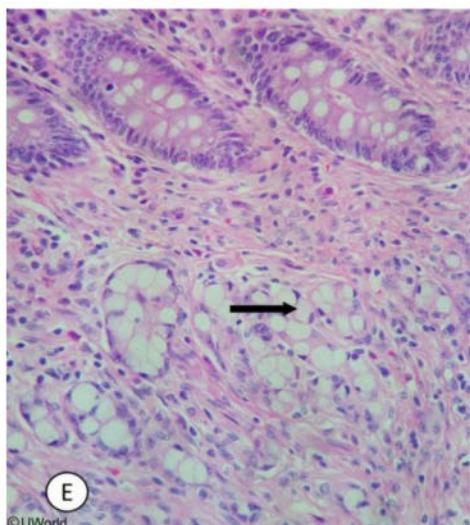
**METASTASIZING to :-**

Lung (canonball)

BONE (osteolytic)

### UWORLD IMAGES

08:12



### UWORLD IMAGES

08:12

Signet Ring Cell Cancer of Stomach (eccentrically placed nucleus)

normally metastasizing to Lung

vs

Clear cytoplasm in RCC

### UWORLD IMAGES

08:13

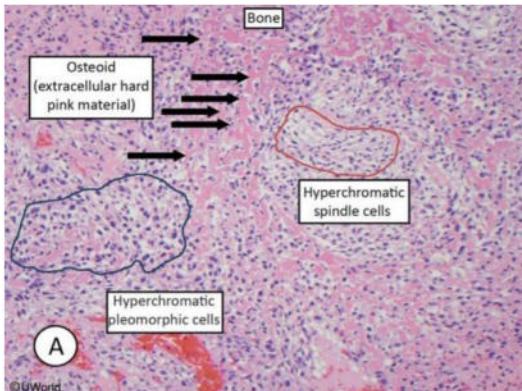
OSTEOSARCOMA

metastasizes to Lung.

### UWORLD IMAGES

08:13

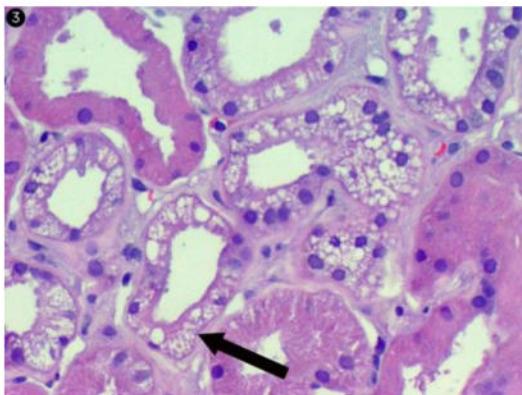
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### UWORLD IMAGES

08:13



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### UWORLD IMAGES

08:13

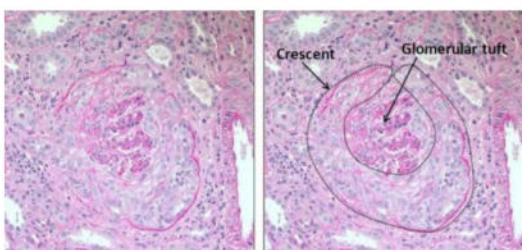
"tubular vacuolization" and arteriolar hyalinization due to direct renal toxicities by

CALCINEURIN inhibitors (cyclosporin and tacrolimus)

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### UWORLD IMAGES

08:39



U

### UWORLD IMAGES

08:39

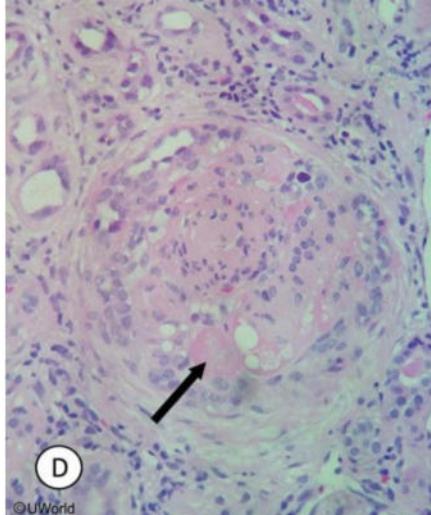
RPGN

glomerular CRESCENT formations (due to parietal cell proliferation, deposition of fibrin, lymphocytes and macrophages)

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### UWORLD IMAGES

08:39



**UWORLD IMAGES**

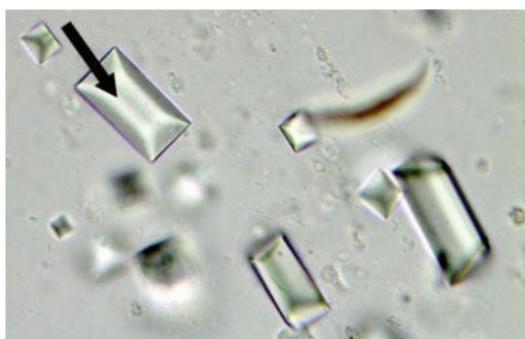
08:39

Kimmelstiel-Wilson lesion/

(eosinophilic) nodular glomerulosclerosis.

**UWORLD IMAGES**

08:40



**UWORLD IMAGES**

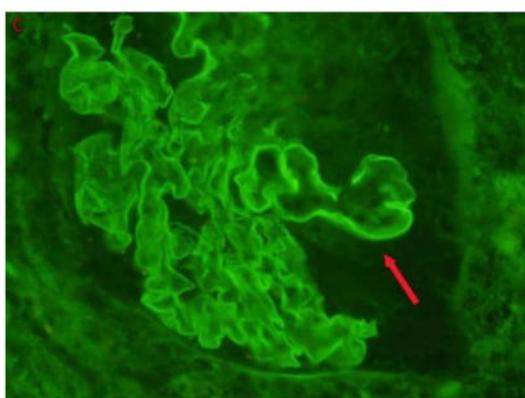
08:40

"Struvite" ( $Mg\ NH_4^+\ PO_4$ )

due to recurrent upper UTI with Urease producing organisms  
(*Klebsiella*, *Proteus*)

**UWORLD IMAGES**

08:40



**UWORLD IMAGES**

08:40

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## Good Pasture Syndrome

IF ma "linear staining" due to anti-GBM Ab against

"alpha 3 chain of Type 4 collagen"

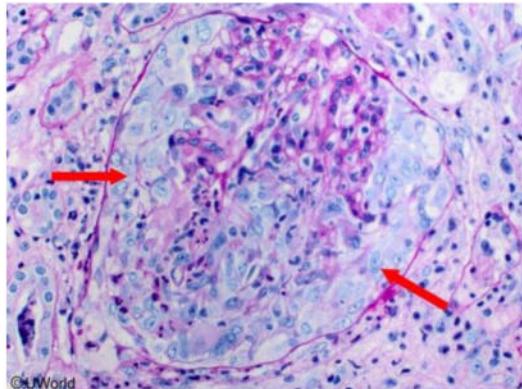
found in :-

1. Glomerular Basement Membrane
2. Alveolar Basement Membrane

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### UWORLD IMAGES

08:40



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### UWORLD IMAGES

08:40

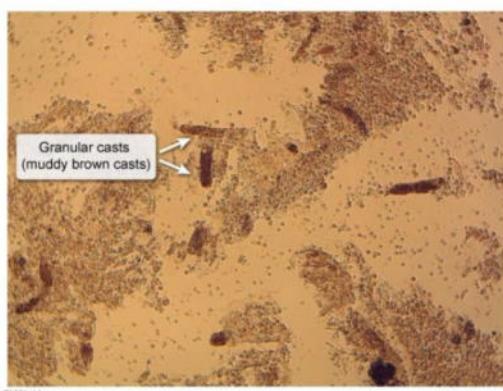
"crescent consisting of fibrin and macrophages" in RPGN

U

### UWORLD IMAGES

08:40

Acute tubular necrosis



U

### UWORLD IMAGES

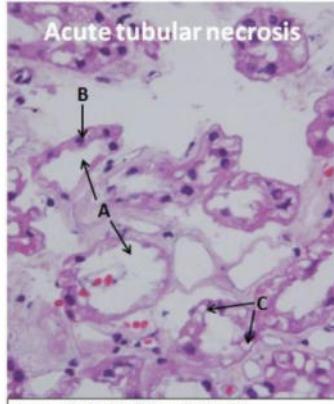
08:40

"Muddy Brown Cast" – ATN

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### UWORLD IMAGES

08:40



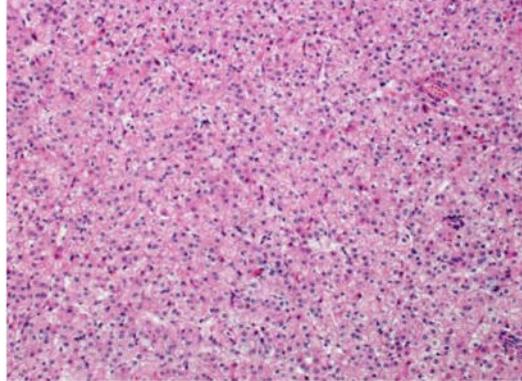
Outer medulla of the kidney (H&E stain)

(a) Patchy loss of proximal tubular epithelial cells with tubular dilation; (b) regenerating epithelial cells with hyperchromatic nuclei; (c) epithelial cell vacuolization.

08:41

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### UWORLD IMAGES



U

### UWORLD IMAGES

08:41

HEPATIC ADENOMA (in females ass. with inc estrogen levels)

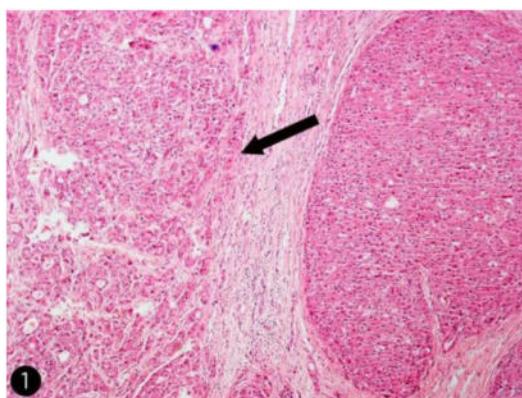
"sheets of normal hepatocytes with bubbly vacuolated cytoplasm".

Reticulin Scaffold Preserved (vs HCC)

U

### UWORLD IMAGES

08:41



U

### UWORLD IMAGES

08:41

HCC

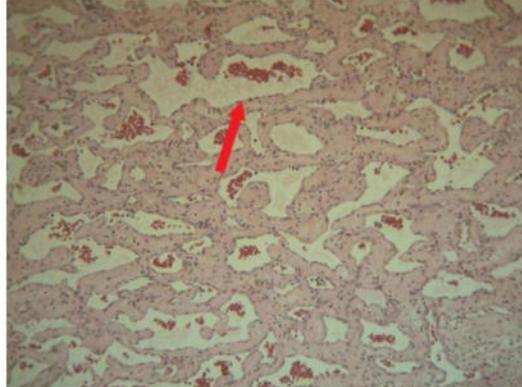
note the irregularly seeming hepatocytes on the left surrounded by

Dense Fibrous Bands (arrow)

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UWORLD IMAGES

08:41



U

UWORLD IMAGES

08:41

CAVERNOUS HEMANGIOMA

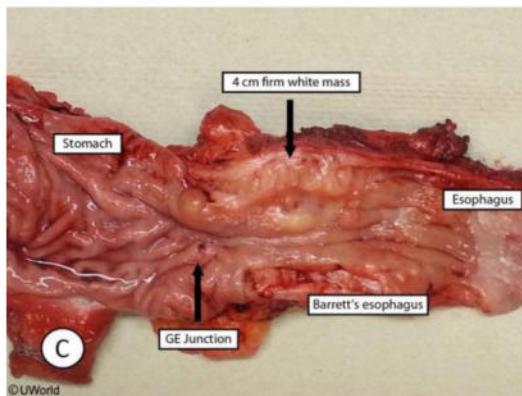
M/C Benign Tumor of Liver

"blood filled cavernous space lined by a single layer of epithelium"  
thought to be congenital malformations which increases in size by  
ECTASIA (or dilation) NOT by hyperplasia/hypertrophy.

U

UWORLD IMAGES

08:41



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UWORLD IMAGES

08:41

"firm 4 cm white mass surrounded by a large segment of discoloured mucosa in the lower esophagus"

Adenocarcinoma of Esophagus.

U

UWORLD IMAGES

08:41



U

## UWORLD IMAGES

08:42

in FAP, the second-hit in wild type (normal) allele of APC gene, occurs in 2nd-3rd Decade of life -->

accumulation of beta catenin -->

activation of transcriptional factors -->

cells in the intestinal crypts grows into numerous polyps -->

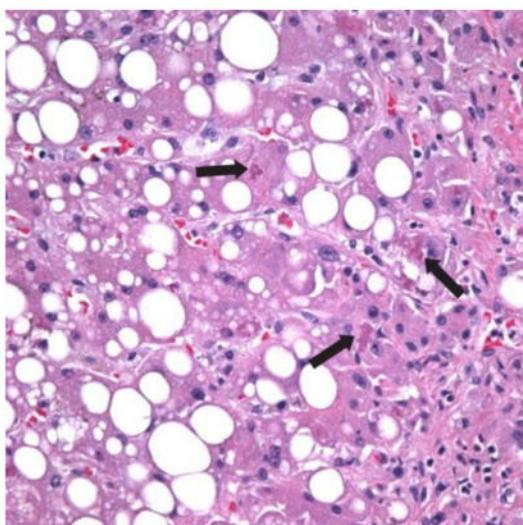
and over a lifetime there is nearly 100% chance of one of these polyps turning carcinomatous (by Adenoma – Carcinoma Sequence AK-53 !!)

HENCE, prophylactic removal.

U

## UWORLD IMAGES

08:42



U

## UWORLD IMAGES

08:42

"Mallory Bodies"

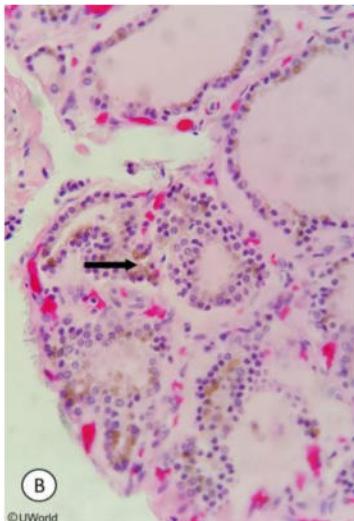
are "eosinophilic cytoplasmic inclusions"  
which represents

"damaged cytokeratin filaments" in alcoholic hepatitis.

## UWORLD IMAGES

08:42

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**UWORLD IMAGES**

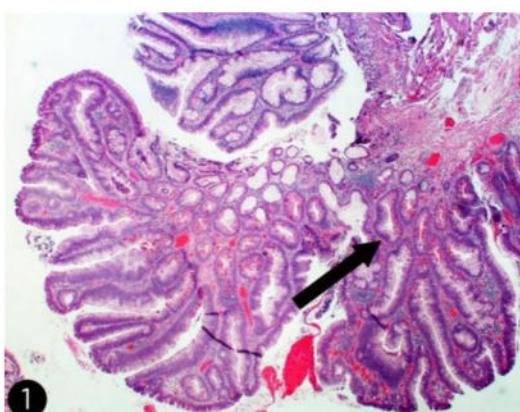
08:42

Hemochromatosis ma liver cells are pigmented due to excess iron deposition (on Prussian blue staining)

U

**UWORLD IMAGES**

08:42



U

**UWORLD IMAGES**

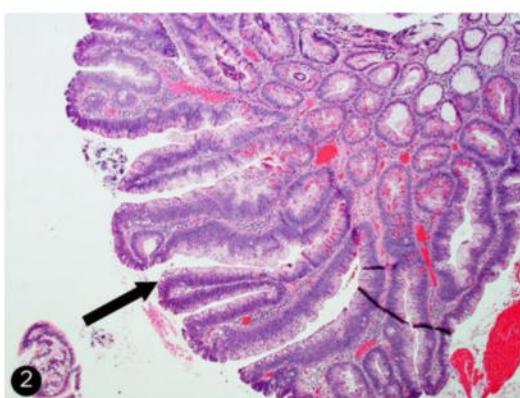
08:42

"tubular glandular" component of a Tubovillous Polyp

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**UWORLD IMAGES**

08:42



U

**UWORLD IMAGES**

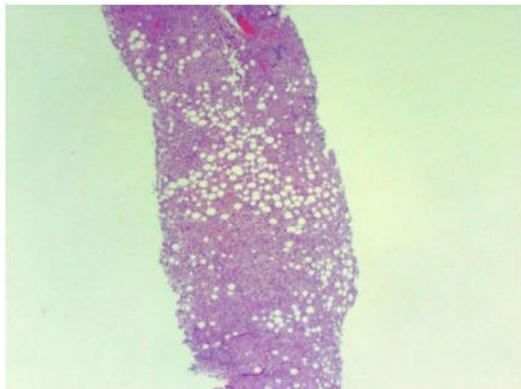
08:42

"villous component" of a tubovillous polyp.

U

**UWORLD IMAGES**

08:43



U

**UWORLD IMAGES**

08:43

"Cytoplasmic Vacuoles" seen in Hepatic Steatosis.

usually in Alcoholic Liver disease. Primarily due to, excess NADH production by the two alcohol metabolising enzymes,

and subsequently,

reduced fatty acid oxidation.

**HISTOLOGY PRINCIPLE :-**

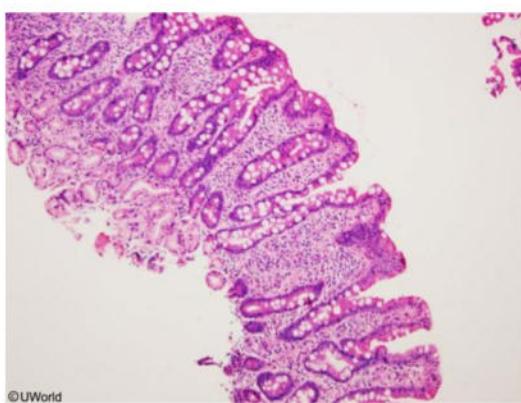
On routine Microscopy, lipid stored in hepatic cytoplasm gets washed away during tissue processing and hence, empty cytoplasmic vacuoles can be seen.

On Frozen section, using stains such as "Sudan Black" and "Oil Red O", lipids can be visualised.

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Celiac Disease :-

Histopathological Findings :-

1. Blunting of Villi/ Atrophy

2. Crypt Hyperplasia  
3. Intra epithelial Lymphocytosis

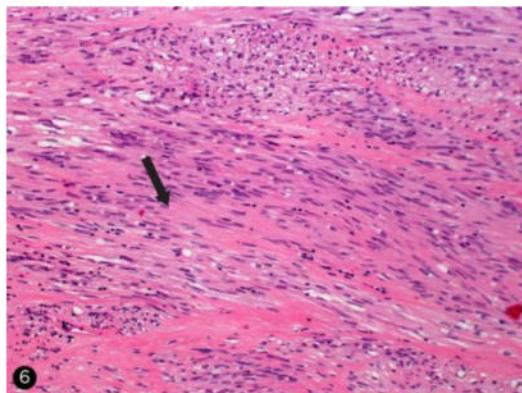
causing S/S of Malabsorption.

example of Secondary lactase Deficiency (temporary)

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"note Spindle shaped eosinophilic cells arranged in fascicles"

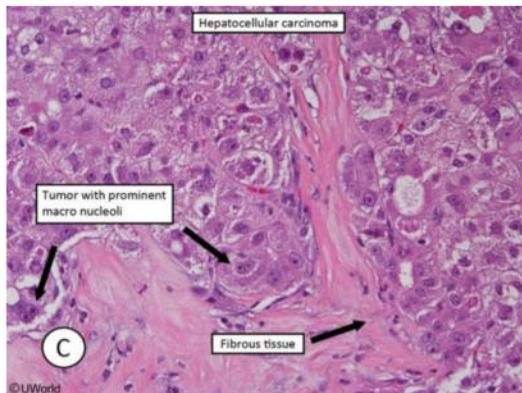
these cells overexpress CD 117.

characteristic of GIST (which are commonly found in Stomach and Intestine)

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Significant Hepatic fibrosis with presence of cluster of

Pleomorphic Cells, high N:C Ratio and prominent nucleoli.

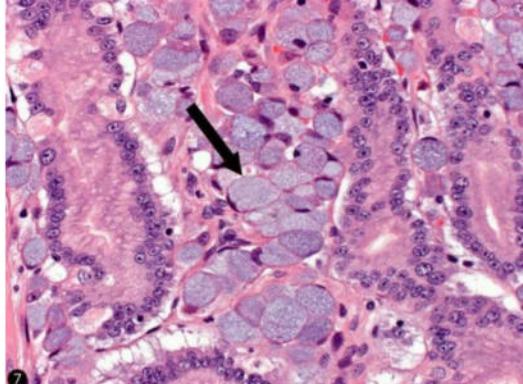
HCC.

chronic HCV with sudden worsening of S/S OF Liver Dysfunction.

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"Signet Ring" cells found in adenocarcinoma of Stomach.

cells produces abundant mucin which pushes the nucleus to the periphery.

this variant of the adenocarcinoma doesn't produce glandular structure like the

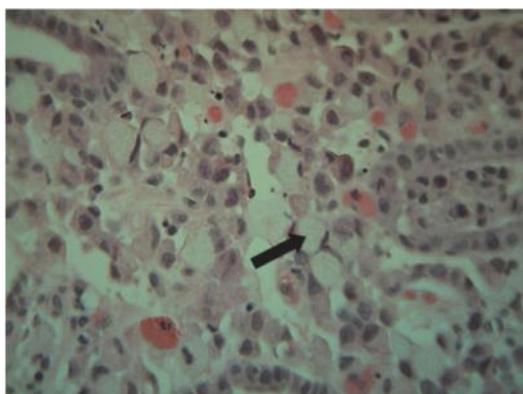
Intestinal Variant. (Grows out as a nodular, polypoid and well demarcated Mass into the Lumen of the Stomach)

Prognosis of Stomach Adenocarcinoma depends mainly on :-

1. Depth of Gastric Wall involvement.
2. Regional Lymph Node Spread.

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"Signet Ring" AdenoCarcinoma of the Stomach. (which does not forms glandular structure)

"abundant mucin producing cells which push the nucleus to the periphery".

there is rapid stomach wall involvement due to Loss of cell adhesion protein E-CADHERIN

1 of 2 variants of Gastric adenocarcinoma.

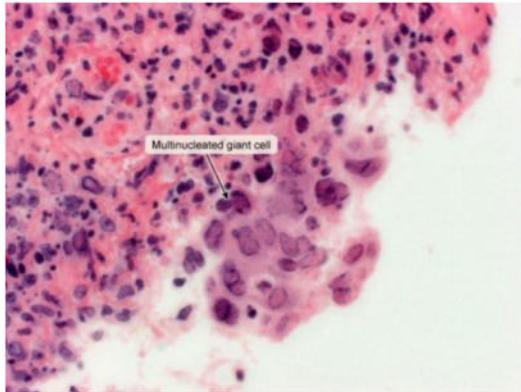
2nd Type is the "Intestinal" variant of adenocarcinoma.

where there is cuboidal/columnar growth of cells into a well-demarcated polypoid mass into the lumen of the stomach.

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"Multi Nucleate Epithelial Giant cells with eosinophilic intranuclear inclusions (cowdry type A)" seen in HSV Esophagitis among Immunocompromised/HIV pts.

"Punched Out Ulcers" on Endoscopy

Rx with Acyclovir

vs

"Basophilic Cytoplasmic/Nuclear Inclusions" seen in CMV Esophagitis

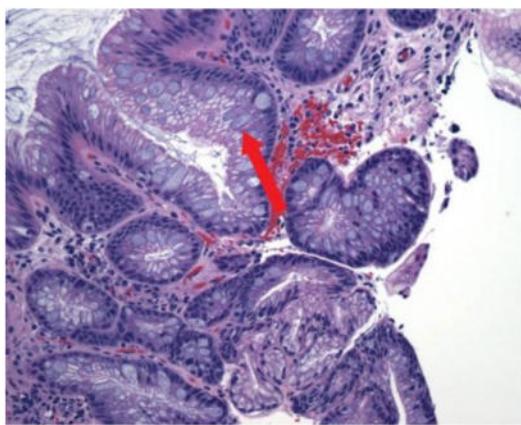
"Linear Ulcers" on Endoscopy.

Rx with Ganciclovir

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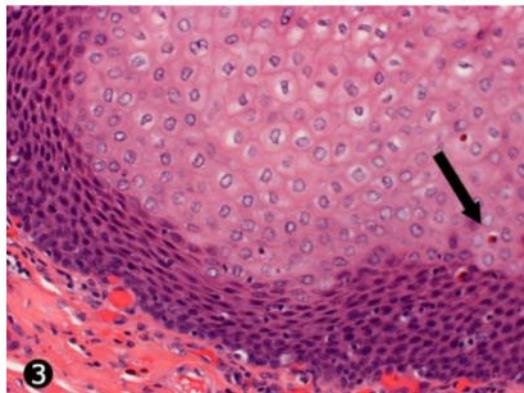
"Goblet cells" (filled with mucus) along with Intestinal Metaplasia of Esophagus.

seen in ADENO Carcinoma variant

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DEEP "Intra Epithelial Eosinophils" seen in Reflux esophagitis

1 Of 3 features

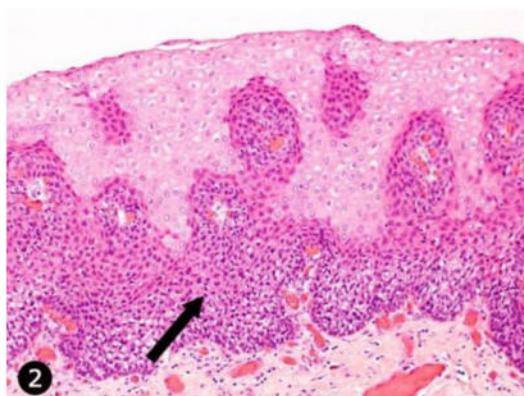
which may progress to Adeno Carcinoma of Esophagus.  
(marked by presence of Goblet Cells filled with Mucus)

SUPERFICIAL "Intra Epithelial Eosinophils" are seen in Eosinophilic Esophagitis.

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"Basal Cell Hypertrophy"

1 of 3 features

seen in REFLUX ESOPHAGITIS (with chronic GERD)

along with

2. Elongation of (lamina propria) Papillae
3. Intra – Epithelial Eosinophils

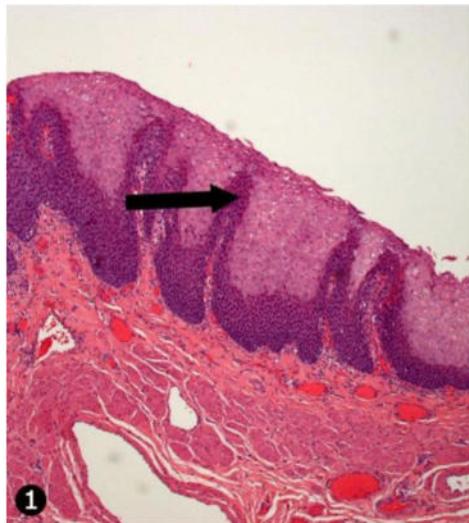
this condition may progress to

Adeno Carcinoma of Esophagus (marked presence of Goblet cells)

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"Elongation of (lamina propria) Papillae"

1 of the 3 features

seen in Reflux Esophagitis (due to chronic GERD)

along with

2. Basal Cell Hypertrophy
3. Intra Epithelial Eosinophils.

GERD may have 2 variants :-

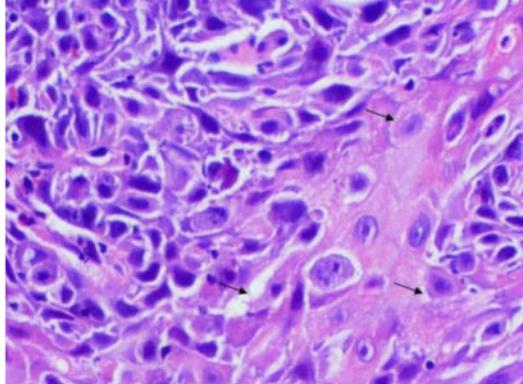
1. NORMAL with Heartburn, Regurgitation and Dysphagia
2. SILENT GERD with none of the above symptoms but with

"Chronic Nocturnal Cough" + "Hoarseness"

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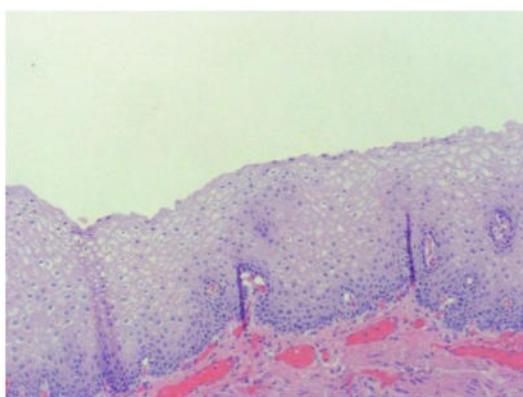
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"Intercellular Bridges" which are found in Cells of Squamous variant of Esophageal Carcinoma.

along with Nests of KERATIN PEARLS.

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Noraml Esophageal Epithelium i.e.

NON-KERATINIZED STRATIFIED SQUAMOUS EPITHELIUM.

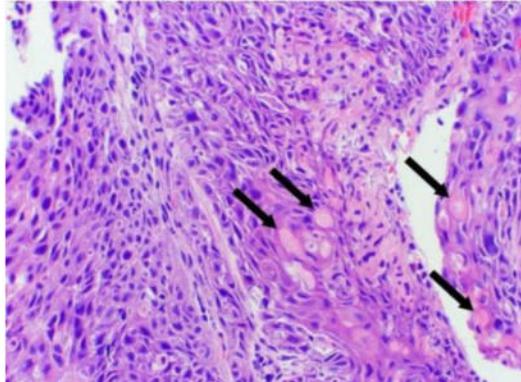
(note the cells of the epithelium does not have distinct cell borders

vs

KERATINIZED Cells having Distinct Cell borders with abundant Eosinophilic Cytoplasm and Nests of Keratin Peals)

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"Sheets of pleomorphic cells"

+

"Nests of Keratin Pearls with abundant eosinophilic cytoplasm"

found in Squamous cell Carcinoma of Esophagus.

Normally the esophagus is lined by NON-KERATINIZED Stratified Squamous Epithelium.

Risk Factors:- Smoking, Alcohol, Nitrosamine containing food, Obesity, Caustic Dye,

may undergo malignant transformation to

1. KERATINIZED Squamous cells which have distinct borders.

2. Inter - Cellular Bridges

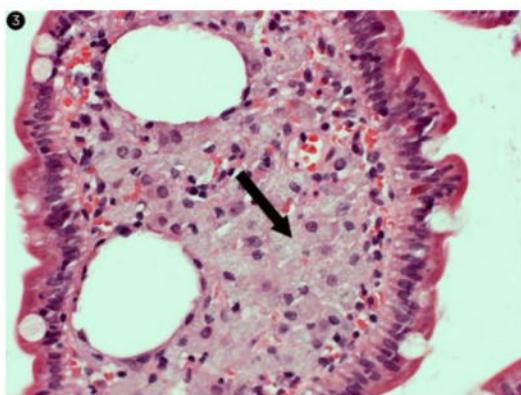
Prognosis is generally poor due to Metastatic/ advanced tumor @ presentation.

commonly in the upper 2/3rd Esophagus.

Spreads to Mediastinal/Brecho Pulmonary Lymph Node.

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"distended macrophages in the lamina propria of Intestine"

seen in Whipple's Disease

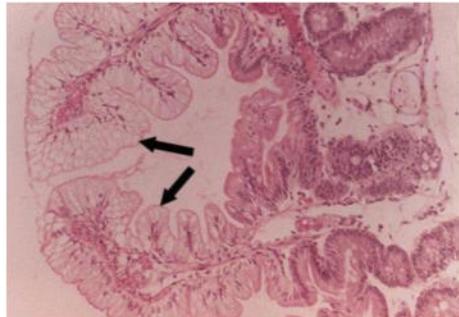
along with PAS +ve + Diastase resistant (magenta coloured) glycoprotein granules along with rod shaped Tropheryma Whipplei Bacteria.

responsible for MALABSORPTION SYNDROME.

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Normal Intestinal (Jejunal) Histology except that the Enterocytes at the tips of Villi are

"distended/filled with lipids (note the clear cytoplasm of the cells) especially at the tips of the villous cells"

this is seen in A-beta-lipoproteinemia

which presents with Steatorrhea and other malabsorption symptoms in an Infant within the first year of life.

Due to AR ; Loss of Function Mutation in MTP Gene (Microsomal Triglyceride Transfer Protein)

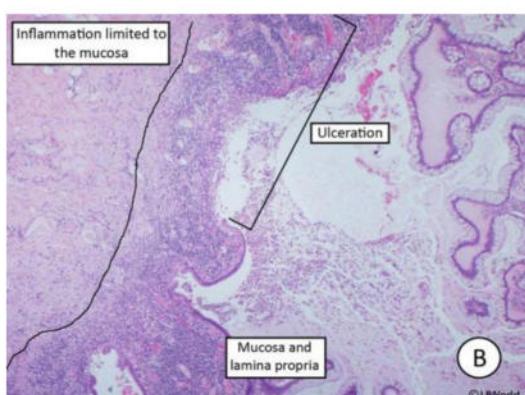
which is a "chaperone Protein" i.e. it helps in

"Proper Folding of apo-B lipoprotein" and in "transfer of TGs in newly synthesised Chylomicrons and VLDL".

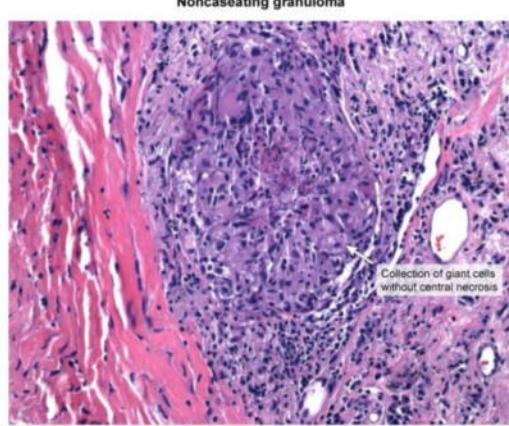
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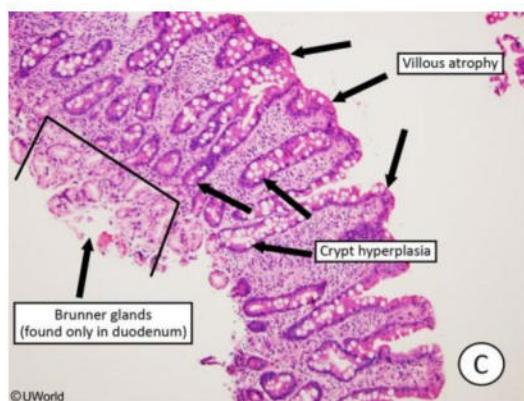


Notice the Superficial Extent of lymphocytic infiltration in Ulcerative Colitis.



"Non Caseating Granuloma" found in any layer of the Intestine in Crohn's Disease.

NCGs consists of Epitheloid Macrophages which may form multi-nucleated Giant Cell surrounded by a rim of lymphocytes.



following findings are seen in CELIAC DISEASE.

which classically presents in a child between age 6–24 months,

with S/S of Malabsorption.

- non-bloody diarrhea
- vomiting
- aversion to feed
- weight loss