CONGESTIVE HEART FAILURE

Pathophysiology

- Normal filling capacity of left ventricle is about 130 ml, out of which about 70ml undergoes ejection, while the remaining volume persist in the ventricles.
- The volume of blood ejected from the left ventricle reduces to about 55ml

  In condition of left ventricular systolic dysfunctioning.
- Any factor that tends to increase the stress on the heart or lead to myocardial infraction results in left ventricular systolic dysfunction.
  (LVSD).
- The eventual consequences is an impairment in the systolic contraction or diastolic relaxation or both.
- Imapariment in the contracting ability of the heart results in **systolic dysfunction**,due to **this ejection faction tends to get lowered.**
- The diastolic function is concerned with the filling of the ventricles, such filling is governed by the venous return and adequate dilation of the ventricles.
- In case of diastolic dysfunction, the ventricles do not dilate properly resulting in relatively less filling.
- If the diastolic dysfunction persists for longer periods, it result in systolic dysfunction and remodelling of the ventricles.

COMPENSATORY MECHANISM

In order to maintain normal cardiac output, several compensatory mechanisms play a role. Compensatory enlargement in the form of *cardiac hypertrophy, cardiac dilatation, or both.*

Tachycardia (i.e. increased heart rate) due to activation of neurohumoral system e.g. release of norepinephrine and atrial natrouretic peptide, activation of renin-angiotensinaldosterone mechanism.

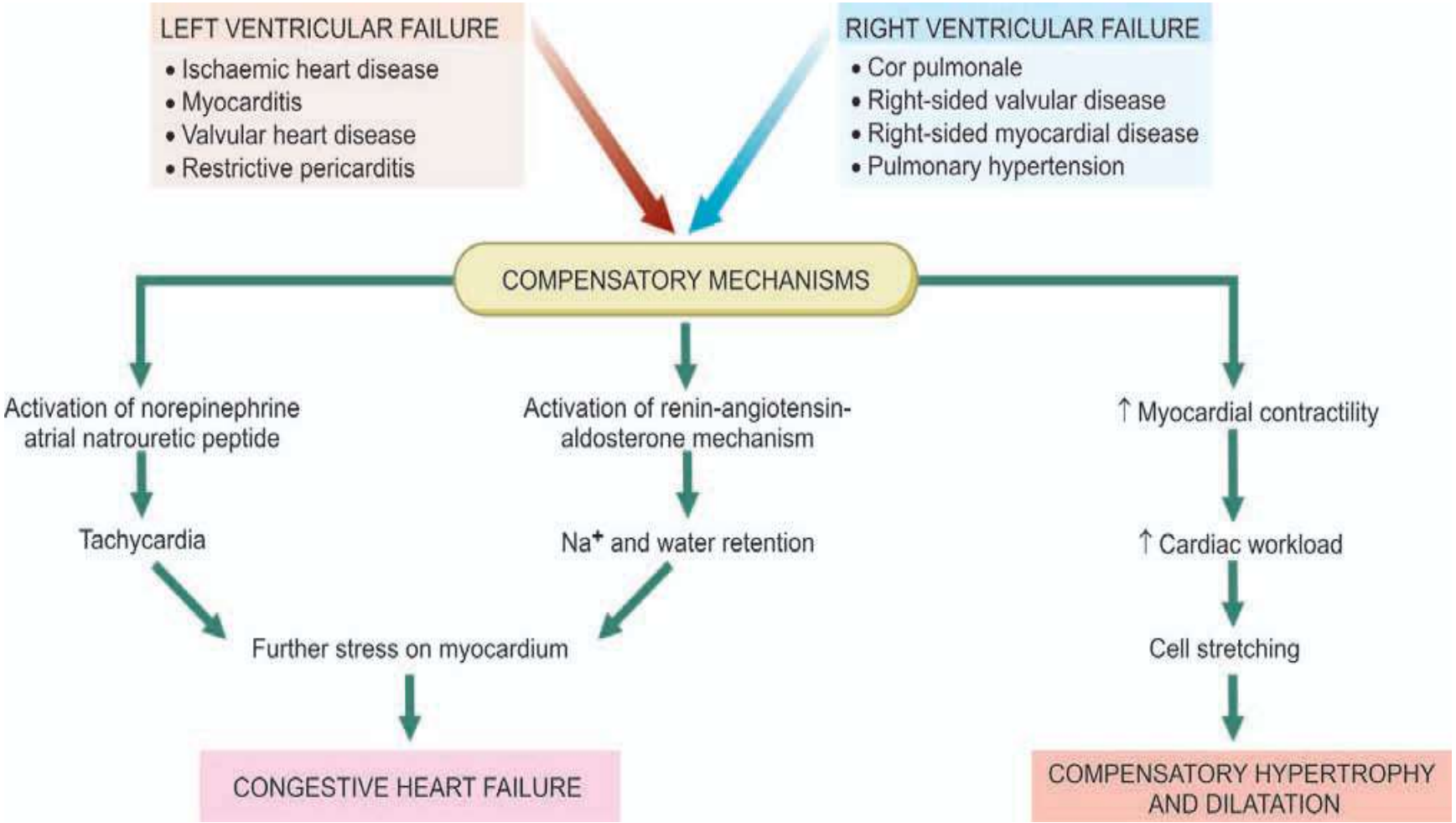
According to *Starling's law* on pathophysiology of heart, the failing dilated heart, in order to maintain cardiac performance, increases the myocardial contractility and thereby attempts to maintain stroke volume. This is achieved by increasing the length of sarcomeres in dilated heart.

Ultimately, however, dilatation decreases the force of contraction and leads to residual volume in the cardiacchambers causing volume overload resulting in cardiac failure that ends in death

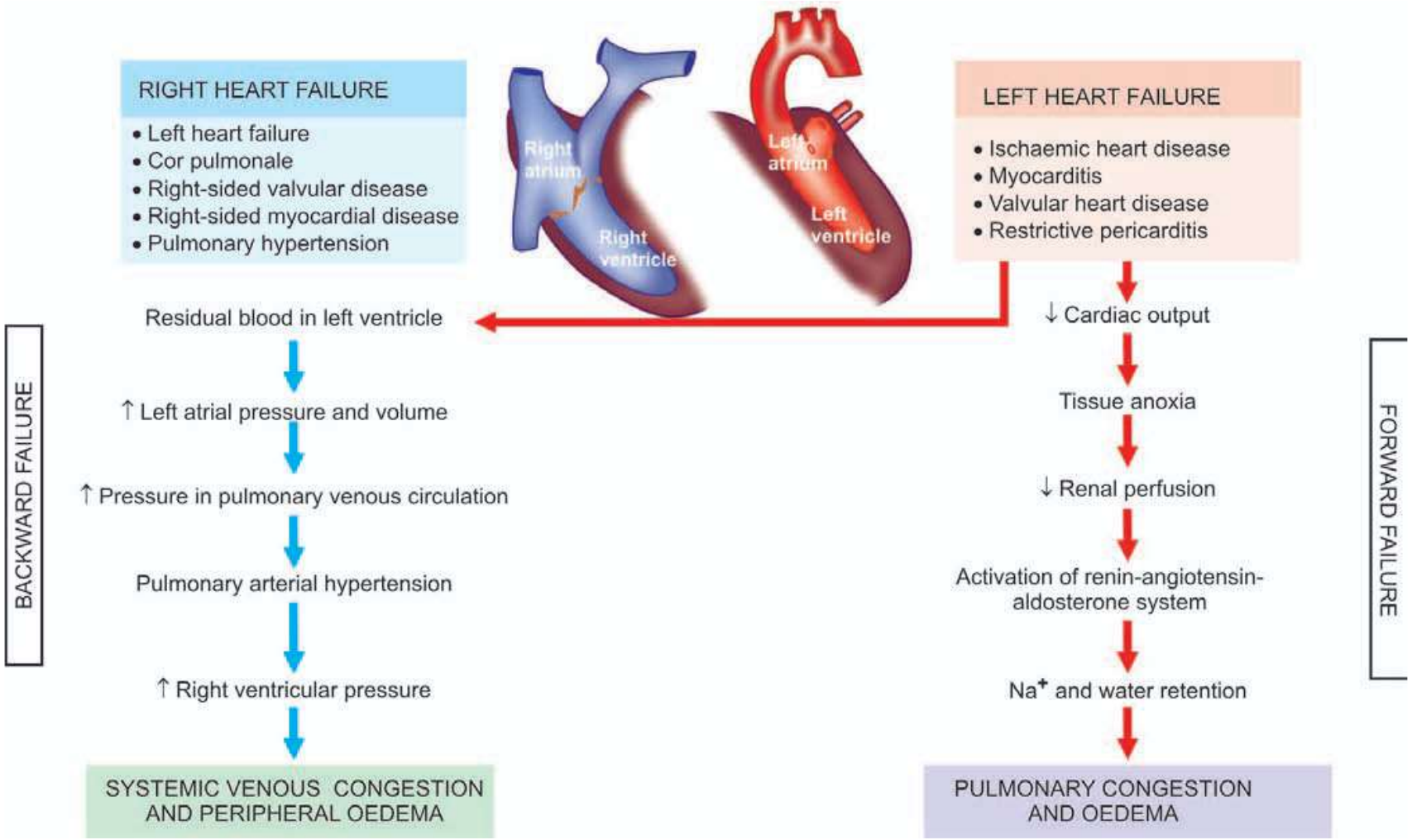
Clinical condition that results in systolic and systolic dysfunctioning include:

- Hypertension
- Aortic stenosis
- Valvular defects
- Acute myocardial infraction
- Cardiomyopathies
- Thus the ventricles fails to fill in the required volume of blood and also fails in its subsequent ejection.
- This results in in the drastic reduction in the stroke volume and cardiac output.
- A decrease in cardiac ouput leads to reduced perfusion of essential organs.
- The body tries to compensate for the reduced cardiac output by stimulating the functioning of various compensatory mechanisims

COMPENSATORY MECHANISMS



EVOLUTION OF CONGESTIVE HEART FAILURE



Increased sympathetic discharge

To compensate for the decreased B.P**, baroreceptors** located in the **arch of aorta** carotid sinuses and walls of the heart get stimulated and **causes activation of beta-adrenergic receptors** leading to an increase in rate and force of contraction of heart.

- An increase in venous return (preload) is also seen due to the **activation of alpha-adrenergic receptors.**
- Increased **rate and force of contraction** together with the increased **preload** results in an initial increase in the **cardiac output**.
- Vasoconstriction of the arteries due to alpha stimulation also causes an increase in after load, leading to fall in ejection fraction.
- As a result the cardiac output decreases.

Activation of RAAS

- Fall in the cardiac output decreases the renal perfusion rate, as a result the RAA system gets activated .
- Angiotensin 2 causes vasoconstriction and an increase in the peripheral vascular resistance(PVR).
- while aldosterone leads to increased retention of sodium and water, there by increasing the blood volume.
- PVR effects the after load during which the heart is unable to pump the extra blood volume.
- This leads to the development of back up pressure causing pulmonary congestion and peripheral oedema.

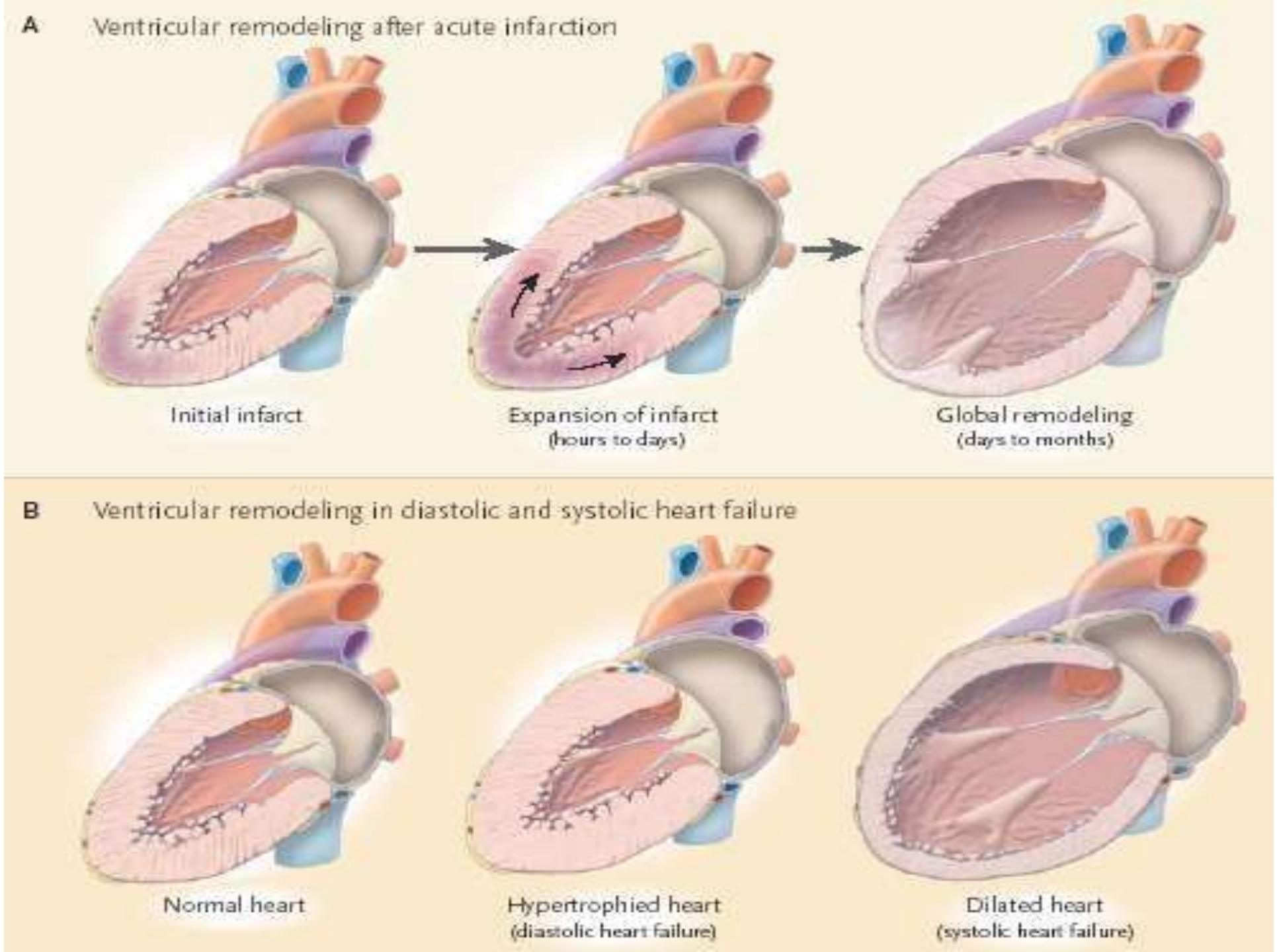
Cardiac Remodeling

- It is most important mechanisum by which body compensated for the intrinsic cardiac effects.
- It involves changes in the shape of the heart (from normal to spherical) due to myocardial hypertrophyt.
- During cardiac remodelling, the connective tissue cells as well as the abnormal myocardial cells undergoes proliferation and dilation instead of steching under the influences of angiotensin 2.
- In the early stages, the remodelled heart maintains the cardiac performances.
- But later on ,hypertrophy may exert certain adverse effects like ischaemic changes, decrease in the rate and force of contraction of heart.

- After certain period of time the compensatory mechanisms get exhausted and worsen the cardiac performances.
- The stress on heart increase and a stage is reached where these mechanisms fails to maintain the adequate cardiac output.

The heart produce signs &symptoms like:

- Dyspnoes, hypoxia
- Pulmonary & peripheral oedema
- Hepatic congestion and enlarged liver
- Enlarged heart
- Decreased exercise tolerance due to decrease in cardiac output
- Decreased urine formation due to decrease in renal perfusion rate.


A
Ventricular remodeling after acute infarction
Initial infarct
Expansion of infarct
(hours to days)
Global remodeling
(days to months)
B
Ventricular remodeling in diastolic and systolic heart failure
Normal heart
Hypertrophied heart
(diastolic heart failure)
Dilated heart
(systolic heart failure)

Clinical manifestations/signs and symptoms

- Fluid retention
- Pulmonary congestion
- Dyspnoea & orthopnoea

CVS manifestations

- Resting tachycardia
- Ventricular arrhythmias
- Enlargement of heart

Renal manifestations

- Nocturia
- Oliguria

Other manifestations

- Reduced cardiac output lead to poor perfusion of skeletal muscle resulting in Fatigue.
- Reduced perfusion to brain results in altered mental states & confusion.
- Reduced perfusion may also causes the patient to appear pale with cold and sweaty hands.

DIAGNOSIS

- Patient history
- Physical examination
- ECHO
- ECG
- CT
- Angiography

THANK YOU

HAVE A WONDERFUL DAY