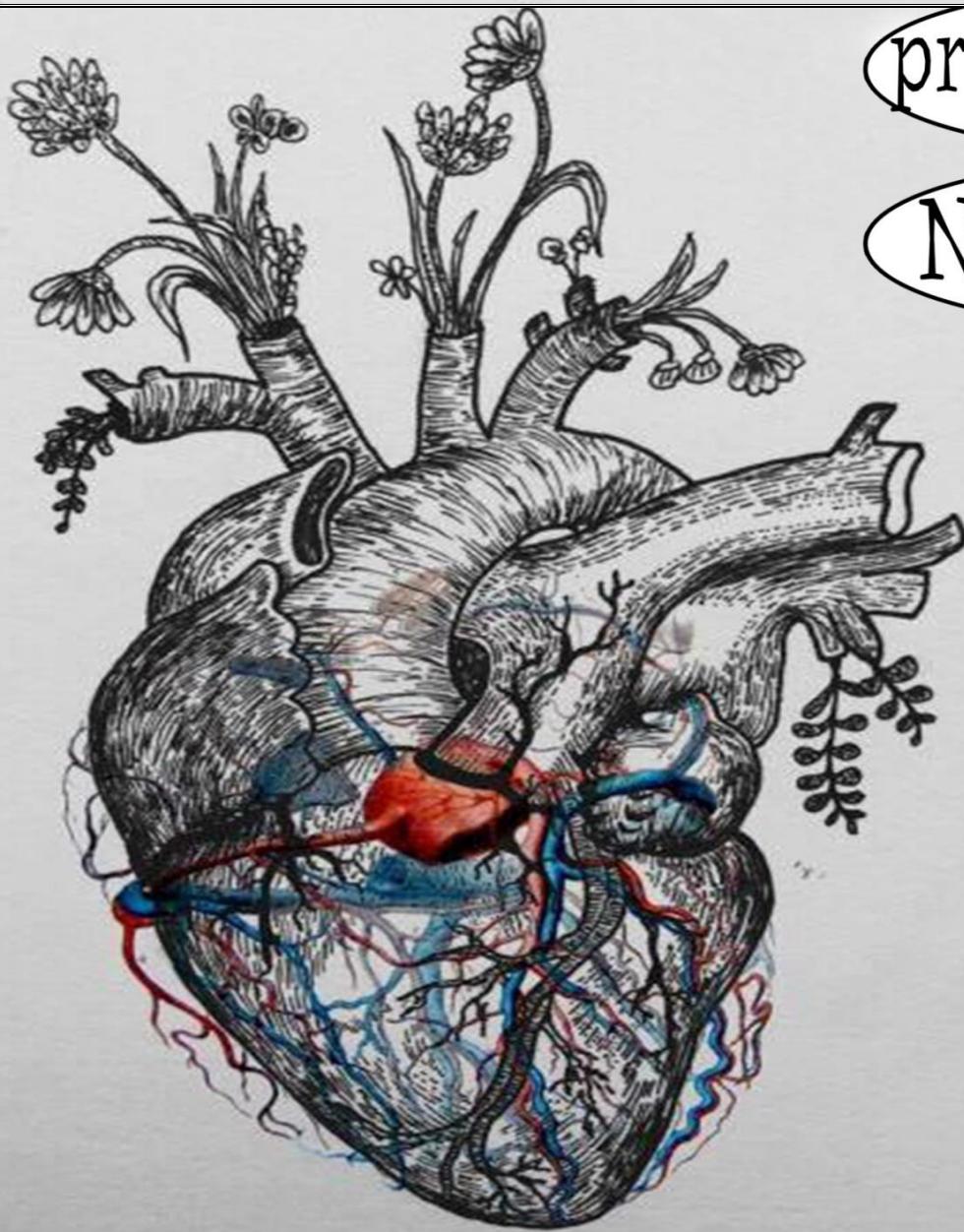


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Physiology #14

lecture

Heart Failure & Circulatory Shock

Doctor

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Date

Tuesday 5/4/2016

Done by

Turquoise Team



Heart Failure:

It's a condition in which the heart can't perform its function properly; it doesn't pump a sufficient cardiac output that meets the tissues' demands.

❖ How do we diagnose the heart failure and its severity?

Based on **the ejection fraction (EF)**.

✓ Recall that:

End diastolic volume (EDV) – End systolic volume (ESV) = Stroke volume (SV)

➔ The ejection fraction (EF) = $\frac{SV}{EDV}$

✓ So, depending on the value ejection fraction:

- EF > 50% ➔ Normal

- EF < 50% ➔ Heart failure (HF), it's further classified into:

* Mild HF: 40% to 49%

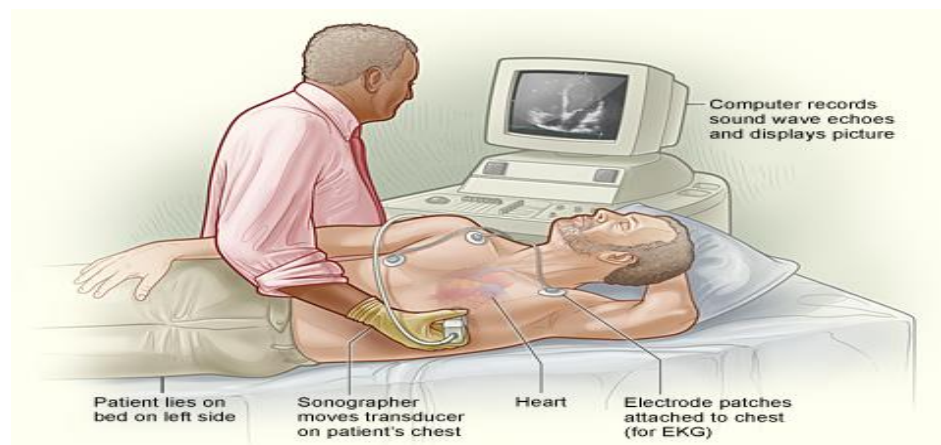
** Moderate HF: 30 to 39%

*** Severe HF: less than 30%

❖ The main test used for the diagnosis of heart failure is the **Echocardiograph**.


This test is based on the ultrasound waves (the sonar!) that are directed against the body and then reflected back to the device to give an image of any organ in the body, and of the fetus in the uterus of the pregnant lady as well. This technique enables us to see the motion of the heart during systole and diastole.

*This image is from Google.



❖ Causes of Heart failure:

Anything that decreases the heart contractility or causes the heart to be out of Frank-Starling law (i.e. the heart is stretched or dilated beyond the physiological limits) is considered to be a cause of heart failure, like:

- **Cardiomyopathies**
 - **Ischemic heart diseases including Myocardial Infarction**
 - Infection
 - Toxins
 - Arrhythmias
 - Others
- 
- The most common

❖ Classifications (Types) of Heart Failure:

1- **Acute or Chronic HF;**

- Acute: when HF develops suddenly as in the case of MI, infection, arrhythmias ... etc.
- Chronic: when HF develops gradually as in the case of Hypertension, valvular diseases ... etc. In hypertension, there will be a gradual myocardial hypertrophy & dilation so that the heart can pump against the very high systemic pressure, and then the dilation will go beyond the physiological limits causing heart failure.

2- **Left- or Right- sided** heart (ventricular) failure, or it may be a failure of both of them called **Congestive heart failure**.

3- **Systolic** (forward) **or Diastolic** (backward) **Failure;**

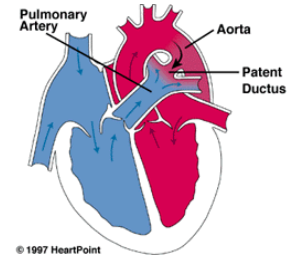
- Systolic: there's a dysfunctional contraction
- Diastolic: there's an impaired blood filling into the ventricles.

4- **High output failure:** this is in which the cardiac output is **normal** or even **higher** than normal but still not enough for the tissues demands.

→ Examples on this condition are:

- Anemia; the oxygen carrying capacity of the blood is reduced, so as a result, the cardiac output will be either normal or will increase to supply the tissues and to compensate for the oxygen reduction but still not enough.

- Patent ductus arteriosus; in which a considerable portion of the blood goes from the aorta into the pulmonary trunk through this opening instead of going to the tissues, so it will keep circulating in this manner. Patent ductus arteriosus is a type of **Arteriovenous (AV) fistulas**.
(Fistula is an abnormal opening between a vein and an artery)



- **Beriberi disease**, a nutritional disorder caused by a deficiency of thiamin (vitamin B1)
- Thyrotoxicosis, increased tissues metabolism

** Remember that whenever we say systole, diastole or angina, we are talking about the left ventricle unless we specify.

Now let's talk a little bit more about types of HF:

Left-sided heart failure

Pathophysiology:

- ⇒ When there is a left heart failure, the left ventricle can't pump blood → this will lead to accumulation of blood in the left ventricle increasing the preload (EDV).
- ⇒ Thus, the left ventricle can't receive more blood.

But where will the blood be?

- ⇒ The blood will be accumulated in the left atrium leading to its dilation and an increase in the left atrial pressure.
- ⇒ As a result, the left atrium won't be able to receive blood from lungs (the left atrium can't pump so it can't receive).
- ⇒ This in turn will lead to the accumulation of the blood in the lungs increasing the pulmonary pressure → filtration of some fluids out of the capillaries into the alveoli affecting oxygen exchange there. What is the impact of this on the patient?

The patient will complain from **Dyspnea (shortness of breath)**.

❖ Symptoms of Left-sided heart failure: In order:

1- **Dyspnea (shortness of breath);** which is the earliest and most significant symptom of left-sided heart failure. This Dyspnea is related to the effort; meaning it's most prominent when the patient makes some sort of effort. The more the fluid in the alveoli the more the severity of dyspnea.

2- **Orthopnea:**

Normally, the upper parts of the lungs are the most ventilated portions. In case of left HF, and when more and more fluid accumulates in the alveolar spaces, the patient will not be able to sleep in supine position because the fluid –when the patient is recumbent- will be distributed all over the lungs including the upper parts. So, to avoid this, the patient usually sleeps in a semi-seated position and he puts many pillows to keep him in this position. The more the fluid in the lungs, the more the pillows he uses.

Note: The doctor in the lecture said that the effect of the gravity in the supine position will be equal all over the body and the venous return will be decreased as a result of increased right atrial pressure, but this is wrong! → **Orthopnea also occurs because the supine position increases venous return from the lower extremities and also elevates the diaphragm.** This is from Robin's pathology book and what Dr. Sukainah said in the medicine lecture. It's true that the effect of the gravity in supine position will be equal all over the body so the blood will not be pooled in the lower extremities and its return to the heart **increases**. This will lead to an increase in the fluids in the lungs.

*If the accumulation of fluid is even more, this will lead the patient to have:

3- **Paroxysmal nocturnal dyspnea:**

Paroxysmal: something repeated ... nocturnal: nightly ... dyspnea: shortness of breath.

In this case, the patient goes to sleep, but after about 2 hours, the fluid will be accumulated in his lungs awakening the patient from sleep with extreme dyspnea and feeling of **suffocation**. The patient then stands up, takes a walk, takes a deep breath until he feels better (when the fluid goes to the lower parts of the lungs) and then goes back to sleep and wakes up again with the same feeling of suffocation after hours and the cycle repeats again and again, hence the name paroxysmal.

4- **Pulmonary edema:** the most serious and severe complication, in which all the lung is filled with fluid, it's **an acute** (emergent) stage so we have to deal with the patient within 5 to 10 minutes, if we don't we will lose the patient. This is a real problem!!

Right-sided heart failure:

Pathophysiology:

Nearly the same thing applies here:

- ⇒ Accumulation of blood in the right ventricle as a result of its failure.
- ⇒ More pressure in the right ventricle → more pressure in the right atrium → more pressure in the peripheral veins → more pressure in the capillaries → filtration of fluid into the interstitium → **Peripheral edema**.

The difference between the pulmonary and the peripheral edema is that the pulmonary edema is something **sudden** (acute, emergent) whereas peripheral edema takes **years** to develop.

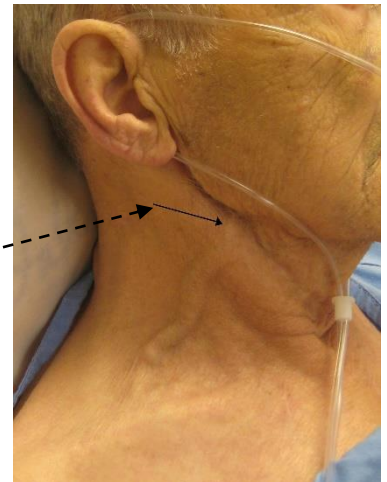
❖ Symptoms of Right heart failure:

1- **Peripheral edema**, which is an accumulation of fluid that may be:

- In the peritoneal cavity → **Ascites**.
- In the spleen → Splenomegaly
- In the liver → Hepatomegaly

2- **Dilated jugular vein**

3- **Jugular vein pulsation:**

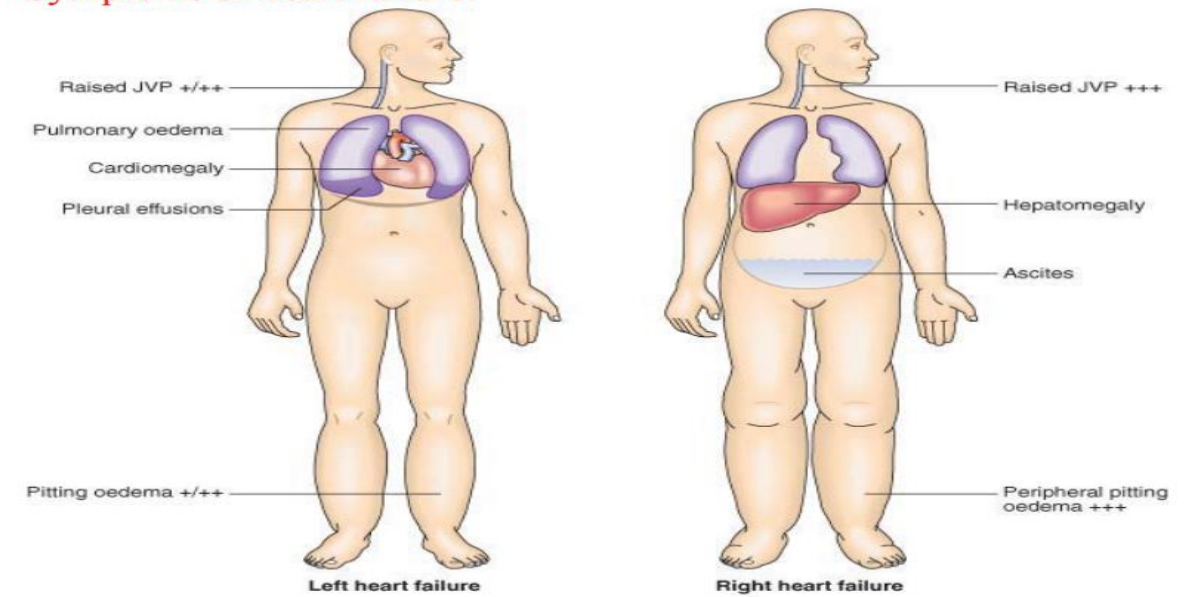


The jugular vein drains blood into the superior vena cava which is connected to the right atrium. So as we said, in case of right-sided HF, when the pressure in the right atrium is sufficiently high, blood flows back into the internal jugular vein. This can be observed as a pulsation.

<http://www.nursingtimes.net/clinical-subjects/cardiology/cardiovascular-examination-part-one-of-a-four-part-series-measuring-jugular-venous-pressure/199369.fullarticle>

**Dilation & pulsation of the jugular vein make it easy to diagnose Right-sided heart failure;
→ the more the pulsation the more severe is the right-sided heart failure.**

Symptoms of heart failure:



❖ Sometimes, both, right-sided and left-sided heart failure, are developed together ... in this case it's called **congestive heart failure**:

- ⇒ It starts with left ventricular failure → high pressure in the left atrium → more blood staying in the lungs → increase the pressure in the lungs (↑ pulmonary pressure)
- ⇒ This increased pulmonary pressure can't be overcome by the right ventricle → the right ventricle won't pump blood to the lungs.
- ⇒ In congestive heart failure you will see the findings of right-sided and left-sided heart failure.

❖ Investigation of heart failure:

You have to do several tests including ECG and chest X-ray but the best test to assess the severity of heart failure is the Echo that gives you exactly the ejection fraction and the cardiac output.

So the best **test** is Echo ... and the best **criteria** to classify the severity of heart failure is the ejection fraction.

We will talk more about these tests in our medicine lectures.

- ⇒ As heart failure develops, the low cardiac output results in hypotension. Now the body has to compensate for this by several compensatory mechanisms.
- ⇒ It starts by inducing vasoconstriction which will lead to an increased circulatory filling pressure thereby increasing venous return and heart rate.
- ⇒ If vasoconstriction is not sufficient enough to balance the blood pressure, the RAAS will come in hand by stimulating urinary retention and further vasoconstriction (with the help of angiotensin) to increase blood volume as well as cardiac output.
- ⇒ If the CO and BP are now within a somewhat normal range, we can call this **compensated heart failure**.
- ⇒ If the body compensatory mechanisms couldn't correct the situation, the blood pressure stays low leading to **decompensated heart failure**.

This figure shows the cardiac output vs. the right arterial pressure in a **compensated heart failure**:

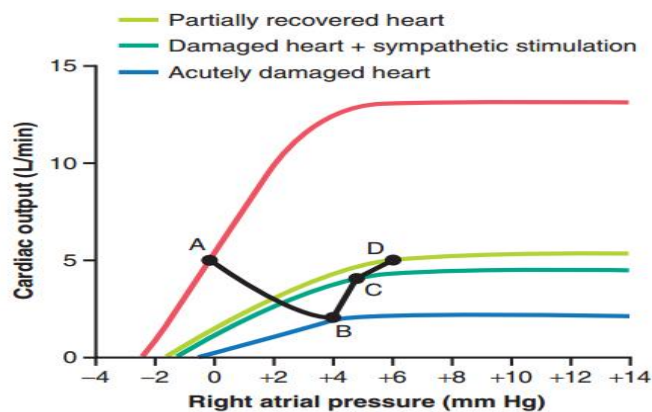
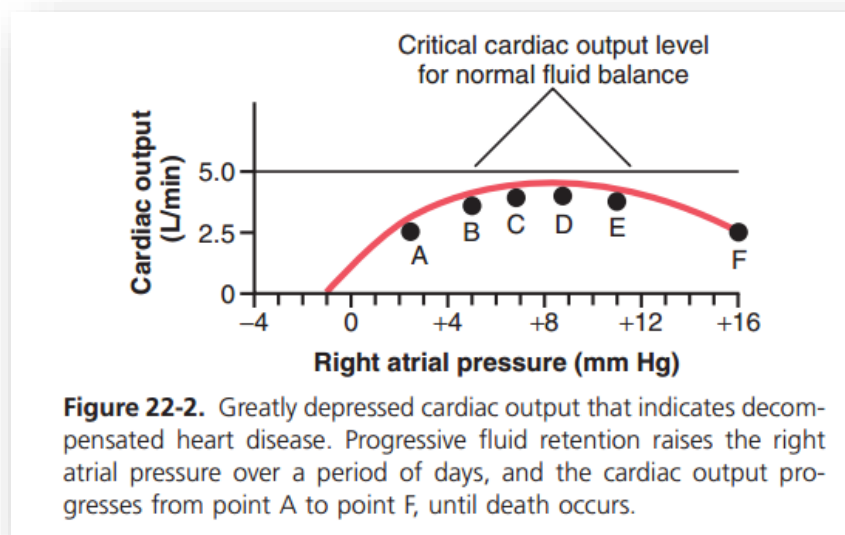


Figure 22-1. Progressive changes in the cardiac output curve after acute myocardial infarction. Both the cardiac output and right atrial pressure change progressively from point A to point D (illustrated by the black line) over a period of seconds, minutes, days, and weeks.

- The uppermost red curve represents the normal heart which has a normal right atrial pressure of 0 (point A) ... and that gives the normal cardiac output of 5L/min
- The lowermost blue curve represents the acutely damaged heart; a failing heart with its **sudden** response:
The right atrial pressure is not zero anymore, it's 4 mm Hg here, and the cardiac about is decreased to about 2 L/min (point B)

- The green curve: After that, the body will respond by compensatory mechanism: The right atrial pressure will be increased a little bit more to about 5 mm Hg and this will increase the cardiac output to about 3 L/min. This is a partially recovered heart (point C). → Early compensatory stage
 - The yellow curve: Later on, with more compensation (sympathetic stimulation), the cardiac output is nearly returned back to the normal on the expense of right atrial pressure increase. (point D) → Late compensatory stage
- At rest, this cardiac output is enough for the patient. But when the patient starts to do exercise it won't be enough anymore.

The following diagram represents a **decompensated heart failure**; it shows that further increase in the right atrial pressure leads to a **DECREASE** in the cardiac output :



Circulatory Shock:

A clinical syndrome characterized inadequate tissue perfusion due to decreases cardiac output and decreased arterial blood pressure (**Hypotension**) and that causes **tachycardia** as a compensatory mechanism.

Symptoms of Shock:

- 1- Hypotension
- 2- Tachycardia
- 3- Cold skin (for the hypovolemic and cardiogenic shock)
- 4- Warm skin (for septic shock)
- 5- Usually the patient is unconscious or semiconscious because of the significant reduction in the blood flow to the brain.

→ To assess the consciousness of the patient just prickle him with a pin. In case of shock, the patient won't respond or will respond a little if he's semiconscious, whereas a normal person's response will be very fast.

Types of shock:

A) Hypovolemic shock

Caused by reduction in blood volume which results in a reduction in blood flow.

Possible Causes:

- 1- Severe dehydration (diarrhea, vomiting, sweating)
- 2- Hemorrhage:
 - Bleeding could be internal or external.
 - An example of internal bleeding is when there's a femoral injury in which there will be no blood going outside the body but it will be in the interstitium and the thigh will be swollen.
- 3- Anaphylactic shock: it's an antigen-antibody reaction; as in the case penicillin hypersensitivity, more histamine will be released increasing the vascular permeability → the fluid will be filtrated to the outside of the capillaries which results in decreased blood volume.
- 4- Burns, in which the patient loses plasma.
- 5- Trauma

**** Burns and Trauma** in addition to hypovolemia will cause severe pain that affect the central nervous system. (I don't know what the point here is but this is what the doctor said).

B) Cardiogenic shock:

Caused by reduction in the contractility of the heart

Anything that causes severe heart failure will lead to cardiogenic shock (e.g. valvular diseases, arrhythmias, MI, cardiomyopathies... etc.)

C) Septic shock (toxic shock, septicemia):

- This is different from the previous two types in which the skin here is **warm** not cold because of the fever caused by the infection.
- The cardiac output may be normal at the beginning
- Blood pressure can be normal
- The patient is unconscious because of the toxic effect of the microorganism

➔ So here the blood volume and pressure are normal at the beginning, but if this septicemia continues, the toxic effect of the microorganism will destroy the elasticity and the contractility of the cardiac muscles which will lead to heart failure.

• General effect of shock:

- Decreased perfusion leading to hypoxia of all tissues and in severe states it may lead to necrosis of some tissues.
- Severe heart failure and significant reduction of the blood to kidneys lead to **renal failure (renal shutdown)**

-
- Note that shock is something beyond the physiological limits. For example Frank starling law is not applied here because it's only applied on healthy cardiac muscles.

Stages of shock:

1) Compensated Shock (stage 1):

When there's hypotension, the body tries to bring the blood pressure back to the normal by several **compensatory mechanisms**.

The compensatory mechanisms for hypotension is **sympathetic stimulation** that causes:

- Tachycardia
- Increased contractility
- Vasoconstriction
- Later on, Angiotensin production to increase water reabsorption and sometimes, if it's severe, the absorption increases to a degree at which the kidneys fails to produce urine (**anuria**) (no or so little fluid is excreted).

So, if a patient loses 1 L of blood, these compensatory mechanisms can bring back the normal blood pressure (nearly to the normal). This condition is called **Compensated heart failure** or compensated shock, in which the response of the body is enough to bring everything back nearly to the normal parameters.

2) Decompensated Shock (stage 2):

If the bleeding results in the loss of a large amount of blood, and the response of the body is not enough in bringing back the blood pressure to the normal, this condition is called **Decompensated (Non-compensated) heart failure or shock**. The patient here needs immediate medical intervention.

Again, in general, in heart failure or in shock, if the body's compensatory mechanisms are enough to bring back everything to normal parameters → Compensated heart failure (or Compensated shock).

If not enough → Decompensated heart failure (or Decompensated shock)

3) Irreversible shock (stage 3):

Sometimes, when there's a severe reduction in blood volume and there is a time until the patient reaches the hospital; this will lead to tissue necrosis. If this happens, then whatever we do to the patient we will lose him because of the

severe hypoxia and the severe tissue damage & necrosis it caused → This is called **irreversible shock** (it's a severe and the last stage of shock).

However, you're NOT allowed to stop treating the patient just because you knew that he has irreversible shock, you have to continue treating him because still the mercy of god is there and anything is possible.

And we're done with the physiology course of the cardiovascular system ... Good luck

The End

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