### **MUNI MED**

### PATOPHYSIOLOGY OF HEART FAILURE

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# Heart as a pump

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- **France Controllery**<br>
 Heart is a central organ of circulatory system<br>
 Heart ejects blood into systemic and pulmonary arteries<br>
 It removes blood from version at cava superior et inferior as well

**Heart as a pump**<br>
• Heart is a central organ of circulatory system<br>
• Heart ejects blood into systemic and pulmonary arteries<br>
• It removes blood from vena cava superior et inferior as well<br>
as from pulmonary arteries<br> EF  $\lceil % \rceil$  = SV/EDV Cardiac output has to match a venous return (↓CO in the cardiac output of cardiac output of cardiac output has to match a venous return (↓CO in the cardiac output has to match a venous return (↓CO in the cardiac output ha • Heart is a central organ of circulatory system<br>• Heart ejects blood into systemic and pulmonary art<br>• It removes blood from vena cava superior et inferio<br>as from pulmonary arteries<br> $CO = SV$  (stroke volume) × f<br> $SV = EDV$  (endd France Starling mechanism: stretching curve SV/EDV)<br>
The stretching – Starling curve SV (endspt of the stretching – SV (endspt of the stretching – SV (endspt of the starling mechanism: stretching of muscular fibres increa





## **Mechanisms leading into heart failure** Mechanisms leading into heart f **Mechanisms leading into heart faill<br>• Extracardiac causes<br>• ↑ preload<br>• Primary – cardiac causes<br>• systolic dysfunction(↓ inotropy)<br>• diastolic dysfunction (↓ lusitropy, tachycardia)<br>• bradycardia • chanisms leading into heart failure**<br>• Thereload<br>• Thereload<br>• Thereload<br>• systolic dysfunction (↓ lusitropy)<br>• diastolic dysfunction (↓ lusitropy, tachycardia)<br>• bradycardia<br>• Thereload and/or afterload (valvular diso

- - ↑ preload
	- ↑ afterload
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- extracardiac causes<br>• ↑ preload<br>• ↑ afterload<br>• systolic dysfunction(↓ inotropy)<br>• diastolic dysfunction (↓ lusitropy, tachycardia)<br>• bradycardia<br>• ↑ preload and/or afterload (valvular disorders and shunts)

# Preload and afterload in a muscular fibre

- Preload and afterload in a muscular fibre<br>• Preload force needed for keeping of muscle<br>• tension before the start of muscle shortening<br>(isometric phase) Teload and afterload in a muscular fibre<br>Preload - force needed for keeping of muscle<br>tension before the start of muscle shortening<br>(isometric phase)<br>Afterload - force needed for isotonic reload and afterload in a musc<br>Preload - force needed for keeping of muscle<br>tension before the start of muscle shortening<br>(isometric phase)<br>Afterload - force needed for isotonic<br>contraction Preload and afterload in a muscular fibre<br>• Preload – force needed for keeping of muscle<br>• Ension before the start of muscle shortening<br>• Afterload – force needed for isotonic<br>• Compared to skeletal muscle, cardiac muscle
- contraction
- increasing Friedrich phase)<br>
Friedral – force needed for isotonic<br>
traction<br>
mpared to skeletal muscle, cardiac muscle<br>
much higher passive stretch force -<br>
ting overstretching, the active force of<br>
traction decreases, but the passiv
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# Preload and afterload in the heart

- $2h$ , where:
	- P....pressure inside the sphere
	- r....inner radius of the sphere
	- h....sphere wall thickness
- Preload wall tension  $(N.m^{-2} = Pa -$  force per area) before the systole
	- The main factor is venous return  $\rightarrow$  filling of cardiac ventricles
- Afterload increase in wall tension during the systole
	- The main factor is a peripheral resistence, or pulmonary vascular resistence

in the case of the right ventricle

• Preload is higher in the right ventricle, afterload is higher in the left one

# Muscular work of the heart – P-V diagram:





# P-V diagram in the right ventricle





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- $MVO<sub>2</sub>$  (PE + SW)  $\times$  f

volume corresponds to wall stress ( $\sigma = \frac{P \times r}{2h}$ ;  $\frac{1}{2}$ 

## **P-V diagram during changes of preload or all analysis of preload or all all analysis of preload or all all all a**<br>afterload afterload



d or<br>
Limit of Frank-Starling<br>
mechanism (active muscular<br>
force decrease) **dor**<br>
Limit of Frank-Starling<br>
mechanism (active muscular<br>
force decrease)



# Inotropy and lusitropy

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- **Inotropy and lusitropy**<br>
 ↑ inotropy ("ability to contract") of the heart –<br>
shifts the endsystolic P-V curve up<br>
 ↑ lusitropy ("ability to relax") of the heart shifts<br>
the enddiastolic P-V curve down<br>
 In principle
	-
- **INCORPY AND LUSITROPY**<br>
 Thistropy ("ability to contract") of the heart -<br>
shifts the endsystotic P-V curve up<br>
 The usitropy ("ability to relax") of the heart shifts<br>
the enddiastodic P-V curve down<br>
 In principle, Final term of the contract of the heart and the cardiac P-V curve up<br>  $\uparrow$  hustropy ("ability to relax") of the heart - shifts<br>
the enddastolic P-V curve down<br>
the muscular frank Staring mechanism<br>
well as it is enabled  $\uparrow$  inotropy ("ability to contract") of the heart -<br>
shifts the endsystolic P-V curve up<br>
the enddiatolic P-V curve up<br>
the endiatolic P-V curve down<br>
the endiatolic P-V curve down<br>
the endiatolic P-V curve down<br>
the en • Those compensatory processes contribute to heart failure<br>
• Thustropy ("ability to relax") of the heart - shifts<br>
• The enddiatolic P-V curve down<br>
• In principle, the relaxation process is ATP-dependent as<br>
which is, ho
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Passive contraction by elastic fibres (relaxation ability decreases)

# "  $\frac{1}{2}$ , Interests" of the heart and perfused tissues

- Increasing of cardiac work means higher energy needs for the myocardium, however the increase of circulating volume/venous return an peripheral resistance is necessary to ensure the perfusion of key organs (heart, lungs, liver, kidneys…) **• On contrary, systemic hypotension is often associated with lower preload (e.g.**<br>
• Increasing of cardiac work means higher energy needs for the myocardium,<br>
however the increase of circulating volume/venous return an p Interests" of the heart and perfused tissues<br>
Increasing of cardiac work means higher energy needs for the myocardium,<br>
however the increase of circulating volume/venous return an peripheral<br>
resistance is necessary to en
- 
- From the heart's viewpoint,  $\downarrow$  preload and  $\downarrow$  afterload are advantageous, regarding the blood supply to key organs they may be linked to circulatory failure Increasing of cardiac work means higher energy needs for the myocardium,<br>however the increase of circulating volume/venous return an peripheral<br>resistance is necessary to ensure the perfusion of key organs (heart, lungs, Increasing of cardiac work means higher energy needs folowever the increase of circulating volume/venous retur resistance is necessary to ensure the perfusion of key org kidneys...)<br>On contrary, systemic hypotension is of
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## **Regulation of circulating volume (preload)** example of the Community of Contract of Contract of Contract of the Sustainess Shipmon of Contract of the Sustainess Shipmon of the Sustainable of the Sustainable of the Shift to the left and the Shift to the left and the egulation of circulating volume (prel<br>  $\uparrow$  preload in  $\uparrow$  systolic volume<br>
– renal function curve shift<br>
– renal function curve shift<br>
the shift to the left<br>
the shift to the left

volume (preload)<br>• Most substances shifting the renal<br>• Most substances shifting the renal<br>• masoconstriction effects, those promoting<br>• the shift to the left are often vasodilators olume (preload)<br>Most substances shifting the renal<br>function curve to the right have also<br>vasoconstriction effects, those promoting<br>the shift to the left are often vasodilators **olume (preload)**<br>Volume (preload)<br>Wost substances shifting the renal<br>function curve to the right have also<br>vasoconstriction effects, those promoting<br>the shift to the left are often vasodilators **olume (preload)**<br>
Most substances shifting the renal<br>
function curve to the right have also<br>
vasoconstriction effects, those promoting<br>
the shift to the left are often vasodilators

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kininy, ANP

prostacyklin

I.-receptory

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## Regulation of systemic peripheral resistance<br>(afterload) (afterload) tion of systemic peripheral<br>
bad)<br>
sodilatation<br>
• No – produced in the<br>
endothelium by constitutive<br>
(eNOS) and inducible (iNOS)<br>
• synthesizeding<br>
• Synthesizeding<br>
• Synthesizeding

- Vasodilatation
	- endothelium by constitutive endothelium by constitutive (eNOS) and inducible (iNOS) synthase
	- prostacyclins
	- histamine
	- bradykinin
	- $pO_2$ ,  $pCO_2$ ,  $pH$  , and  $P^2$
	- adenosine
	- catecholamines
	- cGMP, cAMP
- Vasoconstriction
	- endothelin
	- ATII
	- ADH
	- catecholamines
	- thromboxane A2
	- $\cdot$  Ca<sup>2+</sup>

 $\begin{array}{ll}\n 0 \text{ - produced in the}\\ \n \text{modthelimit by } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text{ and} \text{ of } & \text{ \cdot } \text$ resistence arterioles

# Dilatation in acute cardiac insufficiency **Dilatation in acute cardiac insuffici**<br>• acute reaction of the heart<br>• a consequence of increased enddiastolic volume<br>• enables the use of Frank-Starling mechanism in the acute cardia

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- **Dilatation in acute cardiac insufficiency**<br>• acute reaction of the heart<br>• a consequence of increased enddiastolic volume<br>• enables the use of Frank-Starling mechanism in the acute cardiac<br>insufficiency, but at the expens
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## Cardiac remodelation (cellular level) Cardiac remodelation (cellula<br>• Triggered by overload<br>• Proliferation factors reach the overloaded cardiomyoc<br>• Expression of fetal genes (protooncogenes)  $\rightarrow$  fetal ph<br>• Expression of fetal genes (protooncogenes)  $\rightarrow$  fet **Provided Control (Cellular Letter action conducts)**<br>Figure and potential operation (Cellular Letter action factors reach the overloaded cardiomyocytes (<br>potersion factors action for the overloaded cardiomyocytes (protosi **Cardiac remodelation (cellular lefture of Same Cardiac remodelation (cellular lefture)**<br>
• Triggered by overload<br>
• Proliferation factors reach the overloaded cardiomyocytes<br>
• Expression of fetal genes (protooncogenes)

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- **Cardiac remodelation (cellular level)<br>• Proliferation factors reach the overloaded cardiomyocytes (catecholamines, angiotesin II,<br>• aldosterone, ADH, endothelin-1…)<br>• Expression of fetal genes (protooncogenes) → fetal ph** Cardiac remodelation (cellular level)<br>• Triggered by overload<br>• Proliferation factors reach the overloaded cardiomyocytes (catecholamines, angiotesin II,<br>• Expression of fetal genes (protooncogenes) → fetal phenotype<br>• Sh extrained the contraction of Cellular level)<br>eigered by overload<br>obsteroine, ADH, enach the overloaded cardiomyocytes (catecholamines, angiotesin II,<br>contraction fectal genes (protooncogenes) → fetal phenotype<br>• shorter a **• Cardiac remodelation (cellular level)**<br>• Triggered by overload<br>• Proliferation factors reach the overloaded cardiomyocytes (catecholamines, angiotesin II,<br>• aldosterone, ADH, endothellin-1...)<br>• Expression of fetal gen **Indiac remodelation (cellular lends)**<br>
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oliferation factors reach the overloaded cardiomyocytes<br>
dosterone, ADH, endothelin-1...)<br>
pression of fetal genes (protooncogenes)  $\rightarrow$  fetal phenoty<br>
• shorte **FIGURE TETTIOUE CALTOFT (CETTURE OF SOME OF SOMETABLY THE SHAPE OF SOMETHER (ISONET ACTION AND SURFACT AND ARRY TO SAMPLE OF SOME CHANGE OF SOMETHING THE SHAPE OF SOMETHING THE SHAPE OF SOMETHING THE SHAPE OF SOMETHING** iggered by overload<br>
oligeration factors reach the overloaded cardiomyocytes (catecholamines, angiotesin II,<br>
closterone, ADH, endothelin-1...)<br>
pression of fetal genes (protooncogenes) → fetal phenotype<br>
• choren action • Triggered by overload<br>• Proliferation factors reach the overloaded cardiomyocytes (catecholamines, angiotesin II,<br>• aldosterone, ADH, endothelin-1...)<br>• Expression of fetal genes (protooncogenes) → fetal phenotype<br>• sh
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- - $\cdot$   $\uparrow$  O<sub>2</sub> consumptions
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## Cardiac remodelation in chronically ↑ preload<br>and ↑ afterload and 个 afterload Example 2014<br>
Cardiac remodelation in chronically 1<br>
and  $\uparrow$  afterload<br>
• Volume overload - eccentric<br>
hypertrophy (e.g. valvular<br>
regurgitation, left-to-right<br>
shunt) ardiac remodelation in chronical<br>
Id  $\uparrow$  afterload<br>
Volume overload - eccentric<br>
Nypertrophy (e.g. valvular<br>
regurgitation, left-to-right<br>
shunt)  $\begin{array}{lllllllllllll} \text{r-adjoint} & \text{r-adjoint} &$ FRA THE CONTROLLATION OF A SERVICE THE PRESENT OF THE PRE Figure 1 and  $\uparrow$  afterload<br>
• Volume overload – eccentric<br>
• regurgitation, left-to-right<br>
• shunt)<br>
• wall tension is high (l

- regurgitation, left-to-right shunt) **Solution Controllery**<br> **hypertrophy (e.g. valvular**<br> **hypertrophy (e.g. valvular**<br> **regurgitation, left-to-right**<br>
shunt)<br>
• wall tension is high (law of<br>
Laplace), but lusitropy increases<br>
Pressure overload - concentric blume overload - eccentric<br>
pertrophy (e.g. valvular<br>
egurgitation, left-to-right<br>
unnt)<br>
• wall tension is high (law of<br>
Laplace), but lusitropy increases<br>
ressure overload - concentric<br>
perthrophy (e.g. valvular<br>
+ ume overload - eccentric<br>
ertrophy (e.g. valvular<br>
urgitation, left-to-right<br>
mt)<br>
wall tension is high (law of<br>
Laplace), but lusitropy increases<br>
SSUITE OVERVIORED TO EXAMPLE EXAMPLE EXAMPLE EXAMPLE EXAMPLE EXAMPLE EXAM
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- stenosis, hypertension)
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# Other causes of cardiac hypertrophy Other causes of cardiac hypertrophy<br>
• eccentric: dilated or inflammatory<br>
• concentric: hypertrophic cardiomyopathy

- cardiomyopathy
- 
- Other causes of cardiac hypertrophy<br>
 eccentric: dilated or inflammatory<br>
 concentric: hypertrophic cardiomyopathy<br>
 mixed: IHD, reactive hypertrophy following<br>
myocardial infarction (eccentric in the<br>
myocardial infarc Other causes of cardiac hypertrophy<br>
eccentric: dilated or inflammatory<br>
cardiomyopathy<br>
concentric: hypertrophic cardiomyopathy<br>
mixed: IHD, reactive hypertrophy following<br>
myocardial infarction (eccentric in the<br>
of the Other causes of cardiac hyper<br>eccentric: dilated or inflammatory<br>cardiomyopathy<br>concentric: hypertrophic cardiomyopathy<br>mixed: IHD, reactive hypertrophy following<br>myocardial infarction (eccentric in the<br>ischemic area, conc eccentric: dilated or inflammatory<br>
cardiomyopathy<br>
concentric: hypertrophic cardiomyopathy<br>
mixed: IHD, reactive hypertrophy following<br>
myocardial infarction (eccentric in the<br>
ischemic area, concentric in unaffected part Examplion in the matter of the matter of the methods of the methods of the methods is a set of the chemic area, concentric in the chemic area, concentric in unaffected part<br>of the heart - i.e. combined systolic and astolic
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## Why (concentric) hypertrophy does not finally decrease myocardial  $O_2$  consumption<br>  $\sigma = P \times r / 2h$ Why (concentric) hypertrophy does not finally decrease myocardial  $O<sub>2</sub>$  consumption

- $\blacksquare$   $\sigma$  =  $\blacksquare$   $\times$  r / 2h
- mcentric) hypertrophy does not finally decrease myoca<br>  $\sigma = P \times r / 2h$ <br>
When wall stress (i.e. necessity to<br>
generate higher pressure during overload)<br>
increases (together with MVO<sub>2</sub>),<br>
MVO<sub>2</sub><br>
MVO<sub>2</sub><br>
Rut as the myocardia  $\overline{)}$ ,  $\overline{$ mcentric) hypertrophy does not finally decreased the present of the When wall stress (i.e. neccessity to generate higher pressure during overload) increases (together with  $MVO<sub>2</sub>$ ), hypertrophy initially compensates w metric) hypertrophy does not finally decrease m<br>  $\sigma = P \times r / 2h$ <br>
When wall stress (i.e. neccessity to<br>
generate higher pressure during overload)<br>
increases (together with MVO<sub>2</sub>),<br>
hypertrophy initially<br>
compensates wall s  $MVO<sub>2</sub>$ <br>But as the myocardial mass increases, MVO<sub>2</sub> metric) hypertrophy does not finally dec<br>  $\sigma = P \times r / 2h$ <br>
When wall stress (i.e. neccessity to<br>
generate higher pressure during overload)<br>
increases (together with MVO<sub>2</sub>),<br>
hypertrophy initially<br>
MVO<sub>2</sub>,<br>
But as the myoca
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## Biochemic changes in heart failure **Biochemic changes in heart**<br>• Tissue hypoxia<br>• Tissue hypoxia<br>• Impaired energetic metabolism ( $\downarrow$ ATP and cr<br>• Decreased utilization of fatty acids, followed l **Biochemic changes in heart failure**<br>• Iissue hypoxia<br>• Impaired energetic metabolism (↓ATP and creatine phosphate)<br>• Decreased utilization of fatty acids, followed by glucose<br>• ↑ROS **Biochemic changes in heart failure**<br>• Tissue hypoxia<br>• Impaired energetic metabolism ( $\downarrow$ ATP and creatine phosphate)<br>• Decreased utilization of fatty acids, followed by glucose<br>•  $\uparrow$ ROS<br>•  $\downarrow$ <sub>D</sub>H **BIOCNEMIC CNANGES IN NEATL TAIL**<br>
• Tissue hypoxia<br>
• Impaired energetic metabolism ( $\downarrow$ ATP and creatin<br>
• Decreased utilization of fatty acids, followed by glu<br>
•  $\uparrow$ ROS<br>
•  $\downarrow$ <sub>P</sub>H<br>
•  $\uparrow$ cytosolic Ca<sup>2+</sup><br>
• Incr

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- Figure 1<br>
Figure 1<br>
increased energetic metabolism ( $\downarrow$  ATP and creatine phosphate)<br>
ecreased utilization of fatty acids, followed by glucose<br>
ROS<br>
pH<br>
cytosolic Ca<sup>2+</sup><br>
 Increases the energy consumption vicious circ
- 
- ↑ROS
- ↓pH
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## **Systolic and diastolic heart failure Systolic and diastolic heart failure**<br>• Systolic (with reduced ejection only and the case valvular diseas<br>• Fraction on the case valvular diseas<br>• Impaired lusitropy betwhere the case of the constant of the constant of th **ystolic and diastolic heart fai**<br> **ystolic (with reduced ejection**  $\cdot$  Diastolia<br>
action)  $\cdot$  Minaired inotropy<br>  $\cdot \downarrow$ EF diagnosed as  $\frac{EDV - ESV}{EDV}$ , most  $\cdot$  Diagnosed as  $\frac{EDV - ESV}{EDV}$ , most at the<br>
commonly using **ystolic and diastolic heart failure**<br>
vstolic (with reduced ejection  $\cdot$  Diastolic (with reduced ejection  $\cdot$  Diastolic (with cave valvular<br>
action)  $\cdot$  Limpaired inotropy  $\cdot \downarrow$  Limpaired inotropy  $\cdot \downarrow$  Limpaired **stolic and diastolic heart failure**<br>
tolic (with reduced ejection  $\cdot$  Diastolic (with<br>
tolic (with reduced ejection  $\cdot$  Diastolic (with<br>
limpaired inotropy  $\cdot$  Unapaired lus<br>
limpaired inotropy  $\cdot$  Unapaired lus<br>
Lim **ystolic and diastolic heart failure**<br>
version the matrice of the commonly using USG<br>
• More common in men, younger<br>
• More comm

- fraction)  $\begin{array}{lll} \text{tolic (with reduced ejection} & \bullet \text{ Dias} \ \text{cave} \ \text{impaired entropy} & \bullet \ \text{l} \ \text{lmpained entropy} & \bullet \ \text{l} \ \text{l}} \ \downarrow \text{EF diagonal as} \frac{\textit{EDV}-\textit{ESV}}{\textit{EDV}}, \ \text{most} \ \text{commonly using USG} & \begin{array}{lll} \text{d} & \bullet \ \text{R} \ \text{More common in men, younger} \ \text{patients, DCM} & \bullet \ \text{l} \ \text{More often leads into the terminal} \ \text{heart failure} & \bullet \ \text{f} \ \text{f} \ \text{f} \ \text{f} \$ 
	-
	- $EDV$ ,  $1.1222$ ,  $2.1222$ ,  $2.1222$ most
	- patients, DCM
	-
- Diastolic (with preseved ejection fraction t failure<br>
Diastolic (with preseved ejection fraction -<br>
Ciastolic (with preseved ejection fraction -<br>
cave valvular disease)<br>
• Impaired lusitropy<br>
• Diagnosed using Doppler USG: ↑E/e' (flow<br>
through mitral valve/ mitral
	-
- **FIGUIC AND CONDUC MEAT TAILUTE**<br>
stolic (with reduced ejection orbital control of the terminal<br>  $\cdot$  Moreover action)<br>  $\cdot$  Moreover all the terminal<br>  $\cdot$  More commonly using USG<br>  $\cdot$  More common in men, younger<br>
More • Impaired lusitropy † **ailure**<br>
• **ailure**<br>
• **a**istolic (with preseved ejection fraction -<br>
• Impaired lusitropy<br>
• Diagnosed using Doppler USG: ↑E/e' (flow<br>
• through mitral valve/ mitral anulus movement<br>
• at the beginning of the diastole Sailure<br>
Sailure<br>
Sailure<br>
Sailure<br>
Sailure<br>
Sailure<br>
Container (September 1986: The September 2014)<br>
Container and Sailure<br>
Container and Sailure<br>
Container (September 2014)<br>
Container and Sailure<br>
Container and Sailure<br> Example:<br>
Fig. (1) The diastocology of the diastocology of the valvular disease)<br>
Impaired lusitropy<br>
Diagnosed using Doppler USG:  $\uparrow$  F/e' (flow<br>
through mitral valve/ mitral anulus movement<br>
at the beginning of the di  $\begin{array}{l} \textbf{failure} \\ \textbf{static (with preserved ejection fraction =\newline & \textbf{valvular disease}) \\ \textbf{Impaired lustropy} \\ \textbf{Diagnosed using Doppler USC: } \Upsilon \text{Fe}^\prime \text{ (flow } \text{through mitral value / mitral anulus movement} \\ \text{at the beginning of the diastole) - blood is \text{``presentrices} \\ \textbf{More common in women, older patients,} \\ \textbf{hypertension, HCM, RCM, tachycardia} \end{array}$ ventricles **failure**<br> **Failure**<br> **Exercise Common in the set of the diastole) - blood is<br>
" pressed" rather than "sucked" into the ventricles<br>
• Example 18**<br> **Example 18**<br> **Example 19**<br> **Example 19**<br> **Constrained Unitypy**<br>
• Diagnosed using Doppler USG:  $\text{TE/e}$  (flow<br>
through mitral valve/ mitral anulus movement<br>
at the beginning of the diastole) - blood is<br>
"pr stolic (with preseved ejection fraction -<br>e valvular disease)<br>Impaired lusitropy<br>Diagnosed using Doppler USG: ↑E/e′ (flow<br>through mitral valve/ mitral anulus movement<br>at the beginning of the diastole) - blood is<br>"pressed" stolic (with preseved ejection fraction -<br>e valvular disease)<br>Impaired lusitropy<br>Diagnosed using Doppler USG: ↑E/e′ (flow<br>through mitral valve/ mitral anulus movement<br>at the beginning of the diastole) - blood is<br>"pressed"
	- More common in women, older patients,<br>hypertension, HCM, RCM, tachycardia
	-

## Heart failure - systemic effects<br>
ided failure de des Right-sided failure **CONFIDENT FAILUTE - SYSTEMIC Effece**<br>
Fight-sided failure<br>
ackward 
• Right-side<br>
• Thydrostatic pressure in pulmonary<br>
• Thydrostatic pressure in pulmonary<br>
• Thydrostatic pressure in pulmonary<br>
• Thydrostatic effusion<br> **• Heart failure • systemic effect**<br>
• Right-side<br>
• Right-side<br>
• Cackward<br>
• Thydrostatic pressure in pulmonary<br>
• Thydrostatic pressure in pulmonary<br>
• Thydr<br>
• Thydr<br>
• Thydr<br>
• Thydr<br>
• Thydr<br>
• Thydr<br>
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### •Left-sided failure

### • backward

- ↑hydrostatic pressure in pulmonary • systemic hypotension→ shock • organ failure (liver, kidneys, GIT, • muscular weakness, fatigue, cachexia<br>• Thydrostatic pressure in pulmonary<br>• respiratory failure, pleural effusion<br>• respiratory failure, pleural effusion<br>• pulmonary hypertension  $\rightarrow$ <br>• secondary right-sided failure<br>• sy
- (transudate)
- secondary right-sided failure **Constanting Constanting Constanting Constanting Constanting Constanting Constanting**

### • forward

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- brain)
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# mic effects<br>• Right-sided failure<br>• backward<br>• ^ Ahydrostatic pressure at the venous

- backward
- Thydrostatic pressure in pullitionary<br>capillaries → pulmonary oedema<br>end of systemic **effects**<br> **t-sided failure**<br> **ackward**<br>
• ↑hydrostatic pressure at the venous<br>
• oedemas and effusions in systemic<br>
• oedemas and effusions in systemic<br>
circulation (incl. pleural effusion) Ffects<br>
Fided failure<br>
Sided failure<br>
Sided failure<br>
Ahydrostatic pressure at the venous<br>
Anydrostatic pressure at the venous<br>
Condemas and effusions in systemic<br>
Circulation (incl. pleural effusion)<br>
Anasarca (systemic oe **effects**<br> **t**-sided failure<br>
ackward<br>
• Thydrostatic pressure at the venous<br>
• oedemas and effusions in systemic<br>
• oedemas and effusions in systemic<br>
• anasarca (systemic oedema)<br>
• hepatomegaly, ascites FFECTS<br>
Sided failure<br>
Sided failure<br>
Sided failure<br>
Ahydrostatic pressure at the venous<br>
end of systemic<br>
oedemas and effusions in systemic<br>
circulation (incl. pleural effusion)<br>
anasarca (systemic oedema)<br>
hepatomegaly, **effects**<br> **t**-sided failure<br>
ackward<br>
• Thydrostatic pressure at the venous<br>
• oedemas and effusions in systemic<br>
• oedemas and effusions in systemic<br>
• anasarca (systemic oedema)<br>
• hepatomegaly, ascites<br>
• Nepatomegaly, • hepatomegaly, ascites t-sided tailure<br>
ackward<br>
• ↑hydrostatic pressure at the venous<br>
end of systemic<br>
• oedemas and effusions in systemic<br>
circulation (incl. pleural effusion)<br>
• anasarca (systemic oedema)<br>
• hepatomegaly, ascites<br>
prward<br>
• experiences<br>
• Ahydrostatic pressure at the venous<br>
• oedemas and effusions in systemic<br>
• oedemas and effusions in systemic<br>
• into anasarca (systemic oedema)<br>
• hepatomegaly, ascites<br>
• isolated is a rarity<br>
• leads int  $\bigcap$  hydrostatic pressure at the venous<br>end of systemic<br>oedemas and effusions in systemic<br>circulation (incl. pleural effusion)<br>anasarca (systemic oedema)<br>hepatomegaly, ascites<br>**Ward**<br>isolated is a rarity<br>leads into  $\bigcup$ 
	-
	-
	-
	- forward
		-
		-

# **Heart failure and renal function**

- hypervolemia
- 
- hypervolemia



## Etiology of left-sided and right-sided failure **iology of left-sided and right**<br> **iology of left-sided and right**<br>
• Usually primary<br>
• LHD, MI<br>
• Cardiomyopathies<br>
• Left-sided valvular disease iology of left-sided and right-sided<br>
• Example 1990<br>
• Example 1990<br>
• Example 1990<br>
• Lind, Microsofters and Corp, respected<br>
• Cardiomyopathies<br>
• Left-sided valvular disease<br>
• Severe hypertension<br>
• Outflow tract obs iology of left-sided and right-sided<br>
• Usually primary<br>
• Consully primary<br>
• Consully primary<br>
• Consulty primary<br>
• Cardiomyopathies<br>
• Left-sided valvular disease<br>
• Severe hypertension<br>
• Other causes<br>
• Other causes

- Left-sided
	-
	- IHD, MI
	- Cardiomyopathies
	-
	- Severe hypertension
	- - -
- Copp.<br>• Copp.<br>• Copp.<br>• Copp.<br>• Copp.<br>• Copp.<br>• Copp.<br>• Conservation<br>• Copp.<br>• Copp. • Right-sided<br>• Usually secondar<br>• COPD, pulmonar<br>• COPD, pulmonar<br>• Wellet-to-right shunt<br>• Pulmonary hyper<br>• Sided heart failur<br>• MI in RCA area<br>• Right-sided valvu<br>• Right-sided valvu<br>• Right-sided valvu<br>• Pericardial e pericarditis
	- Right-sided
		-
- Sided failure<br> **Sided failure**<br> **sht-sided**<br>
 Usually secondary<br>
 COPD, pulmonary arterial<br>
hypertension, pulmonary<br>
 Pulmonary hypertension in left. • Sided failure<br> **Sided failure**<br> **Sided**<br>
• Usually secondary<br>
• COPD, pulmonary arterial<br> **hypertension**, pulmonary<br>
• Pulmonary hypertension in left-<br>
• Pulmonary hypertension in left-<br>
• sided heart failure hypertension, pulmonary<br>embolism  $\rightarrow$  cor pulmonale ided failure<br>
ent-sided<br>
Usually secondary<br>
COPD, pulmonary arterial<br>
hypertension, pulmonary<br>
embolism→ cor pulmonale<br>
Pulmonary hypertension in left-<br>
sided heart failure<br>
MI in RCA area • Sided failure<br>
• Usually secondary<br>
• Usually secondary<br>
• COPD, pulmonary arterial<br>
• Pulmonary hypertension, pulmonary<br>
• Pulmonary hypertension in left-<br>
• MI in RCA area<br>
• Right-sided valvular disease<br>
• Right-sided **ided failure**<br> **ided failure**<br> **out-sided**<br> **Usually secondary**<br> **COPD, pulmonary arterial**<br> **hypertension, pulmonary<br>
embolism**  $\rightarrow$  **cor pulmonale**<br> **Pulmonary hypertension in left-<br>
Sided heart failure**<br> **MI in RCA area** • Sided failure<br>
ent-sided<br>
• Usually secondary<br>
• COPD, pulmonary arterial<br>
• My pertension, pulmonary<br>
• Pulmonary hypertension in left-<br>
• MI in RCA area<br>
• Right-sided valvular disease<br>
• Right-sided valvular disease
	-
	- MI in RCA area

No pulmonary

congestion

# Starling forces and edema Starling forces and edem<br>
ures, or pressure gradients<br>
– P<sub>t</sub>) – σ(π<sub>v</sub> – π<sub>t</sub>)], where:<br>
n<br>
an area<br>
ane permeability coefficient (for

- Actually pressures, or pressure gradients
- $F = A \cdot K \cdot [(P_v P_t) \sigma(\pi_v \pi_t)],$  where:
	- F…filtration
	- A…filtration area
	- K…membrane permeability coefficient (for water)
	- σ...membrane reflection coefficient (for all allow proteins)
- The pressure gradient is directed outside at the arterial end and inside at the venous end of a capillary
- 
- Actually pressures, or pressure gradients<br>
 F = A . K .  $[(P_v P_t) \sigma(\pi_v \pi_t)]$ , where:<br>
 F...filtration<br>
 A...filtration area<br>
 K...membrane permeability coefficient (for<br>
water)<br>
 σ...membrane reflection coefficien • Actually pressures, or pressure gradients<br>
• F = A . K . [(P<sub>v</sub> - P<sub>t</sub>) - σ(π<sub>v</sub> - π<sub>t</sub>)], where:<br>
• F...filtration area<br>
• K...membrane permeability coefficient (for<br>
<sup>blood</sup> water)<br>
• o...membrane reflection coeffici
- But the excessive water is either drained by lymphatic vessels or breathed out, the lungs stay<br>"dry"

![](_page_23_Figure_11.jpeg)

The flow from the capillary little exceeds the

- Pulmonary edema and pleural effusion<br>• Pulmonary edema: fluid accumulation in the lung tissue • Pulmonary oedema: fluid accumulation in the lung tissue  $($ "swamp") **• both fluid filtration and pleural effusion**<br> **• both fluid filtration and resorption from/to pulmonary circulation**<br>
• both fluid filtration and resorption from/to pulmonary circulation<br>
• treatment: medication<br>
• crea
	- interstitial
	- alveolar
	-
	- treatment: medication
	- Pulmonary edema and pleural effusion<br>• Pulmonary oedema: fluid accumulation in the lung tissue<br>• interstitial<br>• interstitial<br>• both fluid filtration and resorption from/to pulmonary circulation<br>• Pleural effusion: fluid pleura ("lake") Fractionary edemated mainds and pleural effusion<br>
	intensity of the system of the fluid filtration and resorption from to pulmonary circulation<br>  $\cdot$  treatment: medica rearly Cucristian and production in the lung tissue<br>
	wamp")<br>
	• interstitial<br>
	both fluid filtration and resorption from/to pulmonary circulation<br>
	treatment: medication<br>
	ural effusion: fluid between the parietal and visceral ulmonary oedema: fluid accumulation in the lung tissue<br>
	swamp")<br>
	• interstitial<br>
	• alveolar<br>
	• both fluid filtration and resorption from/to pulmonary circulation<br>
	• treatment: medication<br>
	eural effusion: fluid between the values)<br>
	• Interstitial<br>
	• Interstitial<br>
	• In transmender the particular of the particular of the particular<br>
	• Interstment: medication<br>
	• Interstituted mainly from the systemic circulation<br>
	• It is filtrated mainly from t
		-
		-
		- effusion

### X-ray

![](_page_25_Picture_1.jpeg)

![](_page_25_Picture_2.jpeg)

### **Heart failure according to rapidity of the set of the s**<br>development development Part failure according to rapidity of<br>
expecting to the property of the property of the property of through<br>
decompensation of chronic decompensation of chronic<br>
decompensation of chronic deconsiliation Nillin LIV<br>
Classif decompensation of chronic art failure according to rapi<br>
relopment<br>
respectively<br>
the surface of chronic<br>
the decompensation of chronic<br>
the distribution Killip I-IV<br>
Classification Killip I-IV ant failure according to rapidity of<br>
• Velopment<br>
• Chronic<br>
• De novo origin or through<br>
• Chronic<br>
• Classification Aillip I-IV<br>
• Classification Killip I-IV<br>
• Classification Killip I-IV<br>
• Classification Killip I-IV Propidity of<br>
Propidity of<br>
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Propicty development<br>
Propicty Classification NYHA I-IV<br>
Propicty of the Subset of Subset Prapidity of<br>
Phronic<br>
Classification NYHA I-IV<br>
Prophetication NYHA I-IV

### • Acute

- 
- 

### • Chronic

- 
- 

# **Heart failure treatment Example 12**<br> **Example 12**

#### • Acute

- 
- Rest in bed, hospitalization Nild physical load
- $\cdot$  O<sub>2</sub>
- Diuretics
- **Example 18 September 19 Se** and the transformation of the transformation of the transformation of the transformation of the transformation – i.e. "warm and Wasopressors (in cardiogenic shock - "cold and wet") . Reads inhibition (prevention - i.e. and wet" failure) Part failure treatment<br>
• Treatment of initiating cause<br>
• Rest in bed, hospitalization<br>
• C<sub>2</sub><br>
• Univertics<br>
• Vasodilators (if not severe<br>
• Vasodilators (if not severe<br>
• Vasopressors (in cardiogenic<br>
• Vasopressors (i the transition of initiating cause<br>
Treatment of initiating cause<br>
Rest in bed, hospitalization (Rest in bed, hospitalization (Rest in bed, hospitalization (Rest in bed, hospitalization of the Mid physics of the Conditioni **Chronic**<br>
• Treatment of initiating cause<br>
• Rest in bed, hospitalization<br>
• O<sub>2</sub><br>
• Diuretics<br>
• Vasodilators (if not severe<br>
• Vasodilators (if not severe<br>
• Vasodilators (if not severe<br>
• Vasopressors (in cardiogenic<br> **•** Chronic<br>
• Treatment of initiating cause<br>
• Rest in bed, hospitalization<br>
• O<sub>2</sub><br>
• Diuretics<br>
• Vasodilators (if not severe **a**<br>
• Vasodilators (if not severe **a**<br>
• Vasopressors (in cardiogenic **a**<br>
• Meart wet "fai  $\begin{tabular}{lllllllllllllllllll} \multicolumn{3}{l}{{\small \begin{tabular}{l} \multicolumn{3}{l}{}{\textbf{c}}{\small \end{tabular} \end{$
- 
- 
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- 

#### • Chronic

- 
- 
- **hronic**<br>• Treatment of initiating cause<br>• Mild physical load<br>• Conditioning training 3-5 times<br>• Para Week 20-30 min **hronic<br>• Treatment of initiating cause<br>• Mild physical load<br>• Conditioning training 3-5 times<br>• Piuretics<br>• Diuretics hronic<br>• Treatment of initiating cause**<br>• Mild physical load<br>• Conditioning training 3-5 times<br>• Piuretics<br>• Heart rate reduction (B-blockers **Solution Control Con**
- Diuretics
- **hronic**<br>• Treatment of initiating cause<br>• Mild physical load<br>• Conditioning training 3-5 times<br>per a week 20-30 min<br>• Diuretics<br>• Heart rate reduction (β-blockers, digoxin, ivabradine)<br>• RAAS inhibition (prevents<br>remodel digoxin, ivabradine) • Treatment of initiating cause<br>• Mild physical load<br>• Conditioning training 3-5 times<br>per a week 20-30 min<br>• Diuretics<br>• Heart rate reduction (β-blockers, digoxin, ivabradine)<br>• RAAS inhibition (prevents<br>• remodelling)<br>• hronic<br>
• Treatment of initiating cause<br>
• Mild physical load<br>
• Conditioning training 3-5 times<br>
per a week 20-30 min<br>
• Diuretics<br>
• Heart rate reduction (β-blockers,<br>
digoxin, ivabradine)<br>
• RAAS inhibition (prevents<br> • Treatment of initiating cause<br>• Mild physical load<br>• Conditioning training 3-5 times<br>per a week 20-30 min<br>• Diuretics<br>• Heart rate reduction (β-blockers,<br>digoxin, ivabradine)<br>• RAAS inhibition (prevents<br>remodelling)<br>• I
- remodelling)
- (arrhythmia)
-