



# THE MODULE: CARDIOVASCULAR SYSTEM

SESSION: 5, LECTURE: 2

DURATION: 1hr

## PRESSURES AND FLOW IN SYSTEMIC CIRCULATION

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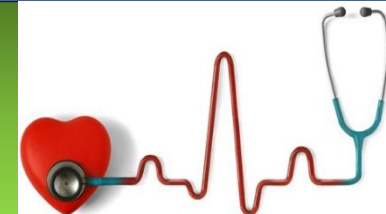
**Dr Mohammed Taha**



As in work book



For more discussion, questions or cases need help please post to the session group



# Learning outcomes (LO)

(LO1)

- Define the terms '**Systolic**' and '**Diastolic**' arterial pressure and '**Pulse Pressure**'.

(LO2)

- Define the term '**Total Peripheral Resistance**'.

(LO3)

- Describe how the elastic nature of arteries acts to reduce arterial pressure fluctuation between systole and diastole.



# Learning outcomes (LO)

(LO4)

- Draw the typical **arterial pressure wave** form.

(LO5)

- Describe the **pulse wave**.

(LO6)

- Describe the role of arterioles as **resistance vessels**.



# Learning outcomes (LO)

(LO7)

- Define the terms **vasoconstriction** and **vasodilatation**.

(LO8)

- Describe what is meant by '**vasomotor tone**' and list the main factors which affect it.

(LO9)

- Describe how '**vasodilator metabolites**' modify vasomotor activity to permit local control of blood flow.



# Learning outcomes (LO)

(LO10)

- Describe **reactive hyperaemia**.

(LO11)

- Describe **autoregulation**.

(LO12)

- Define the terms '**central venous pressure**' and '**venous return**'.



**Systolic Pressure**

**The maximum arterial pressure, 120 mmHg.**

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**Diastolic Pressure**

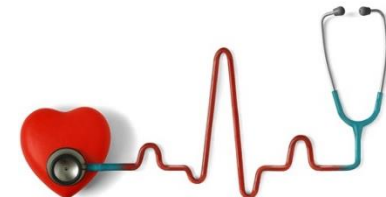
**The minimum arterial pressure, 80 mmHg.**

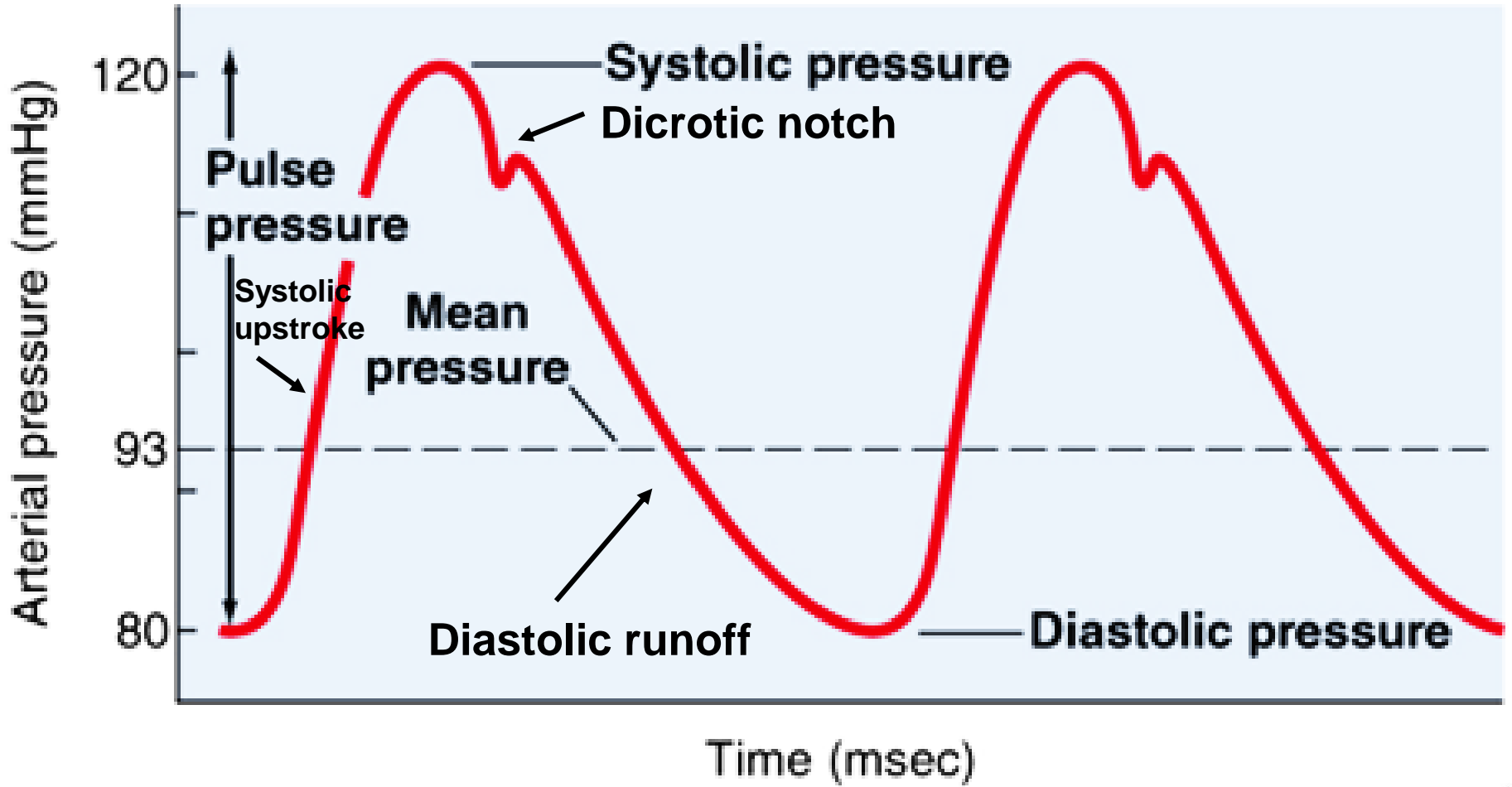
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**Pulse Pressure**

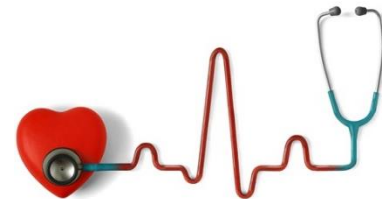
**The difference between systolic and diastolic pressure, 40 mmHg.**

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<https://biology.stackexchange.com/questions/31286/what-causes-a-small-bump-in-the-arterial-pressure-plot?rq=1>



# Total peripheral resistance (TPR)




-  Total peripheral resistance is the sum of the resistance of all of the peripheral vasculature in the systemic circulation.

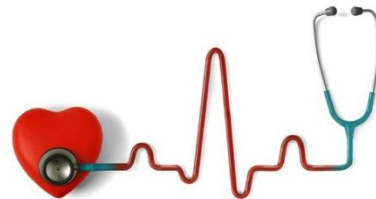
**What are the factors that influence TPR?**

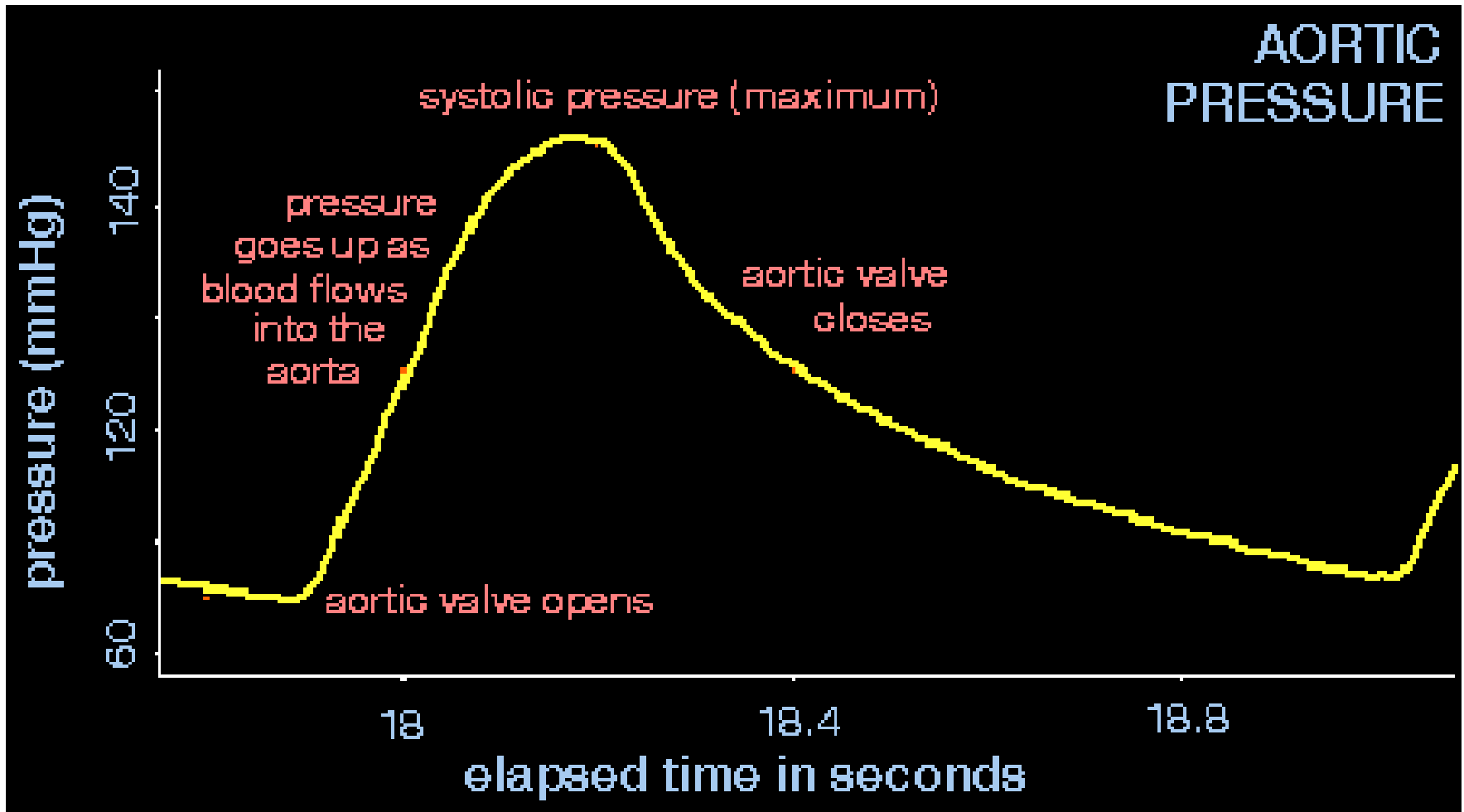




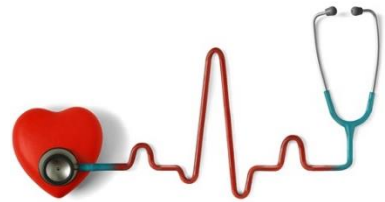
# Pressure fluctuation

-  Ventricle ejects blood intermittently.
-  Arteries have distensible walls, allowing them to stretch in systole. More blood flows in than out, so pressure does not rise so much.
-  The arteries recoil in diastole and flow continues through the arterioles.

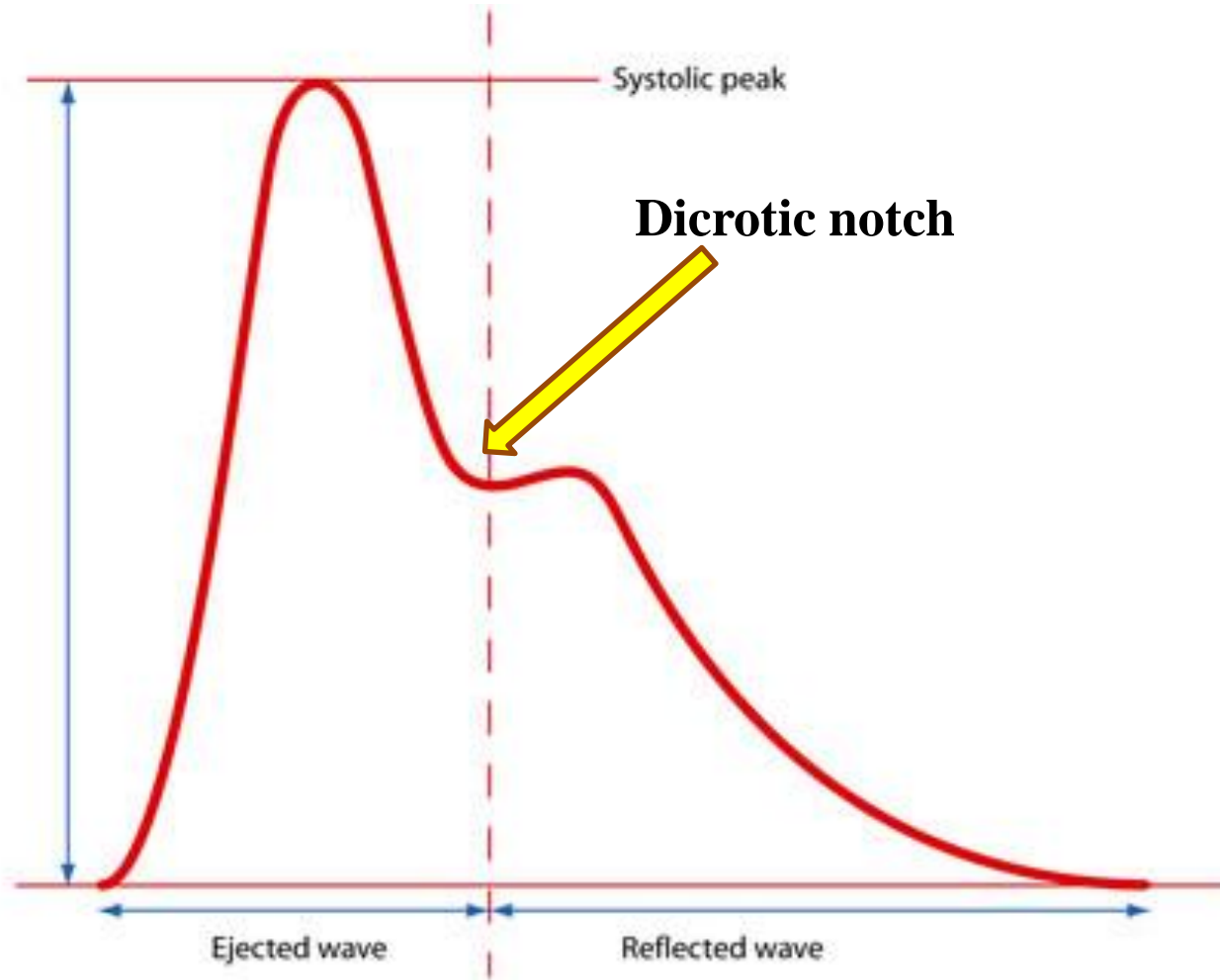




[http://int-prop.lf2.cuni.cz/heart\\_sounds/ekg5/cham2.htm](http://int-prop.lf2.cuni.cz/heart_sounds/ekg5/cham2.htm)



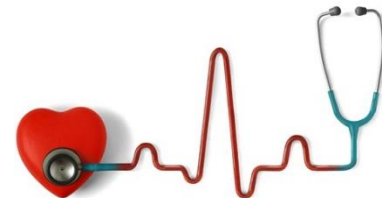
# Arterial pulse wave



**(Systolic phase)**

**(Diastolic phase)**

[http://www.centerforhearts.com/PULSE\\_WAVE.html](http://www.centerforhearts.com/PULSE_WAVE.html)



# pulse wave


❗ Contraction of the ventricle generates a pulse wave.

❗ Propagates along the arteries faster than blood.

❗ felt at a variety of locations where arteries come close to the surface and can be pushed against a reasonably hard surface.



# Resistance vessels



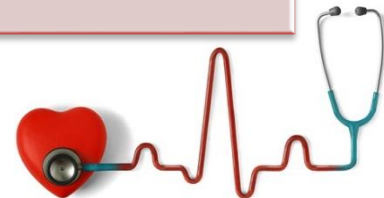
🩺 Arterioles control blood flow to tissues by variable flow restriction.

🩺 Walls of arterioles contain much SM.

🩺 State of contraction determines lumen diameter → flow resistance.

🩺 Vasoconstriction leads to ↓ in flow.

🩺 Vasodilatation leads to ↑ in flow.

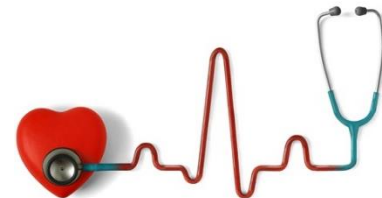


## Vasoconstriction

❖ Narrowing of blood vessels that results from contraction of the muscular walls of the vessels.

## Vasodilatation

❖ widening of blood vessels that results from relaxation of the muscular walls of the vessels.



# Vasodilatation and Vasoconstriction



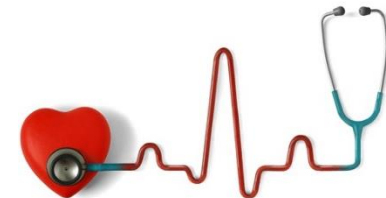
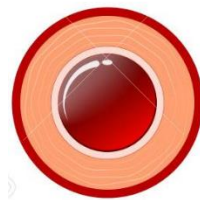
**Normal  
artery**






**Constricted  
artery**

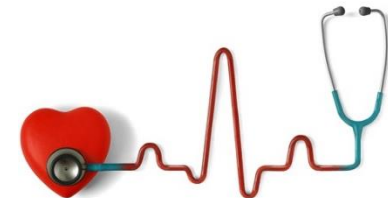


**Dilated  
artery**



# Vasomotor tone

-  Muscles do not actively relax, so except under maximum flow conditions there must always be some vasoconstriction.
-  Continuous contraction of the VSMCs is known as **vasomotor tone**.
-  Vasomotor tone is mainly produced by the sympathetic branch of the ANS ( $\alpha$  adrenergic receptors).





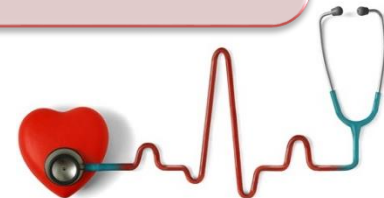
# Vasodilator metabolites

Metabolically active tissues produce vasodilator metabolites ( $H^+$ ,  $K^+$ , Adenosine)

relaxation of local smooth muscle

lowering resistance

increasing blood flow



# Vasodilator metabolites

Nitric oxide  
(endothelial cells)

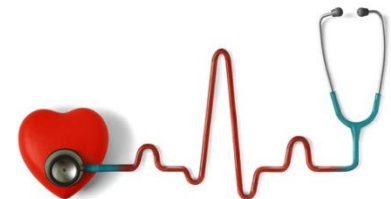
Adenosine (heart,  
SkM, brain)

Hypercapnia (CO<sub>2</sub>) &  
hypoxia

Lactic acid

Hyperosmolarity

Others ( bradykinin  
prostaglandin,  
prostacyclin, histamine)

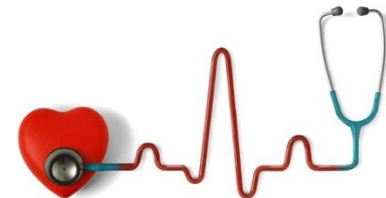


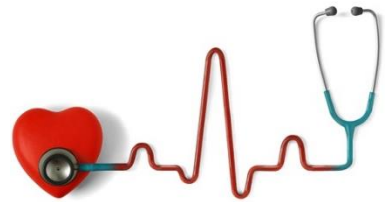
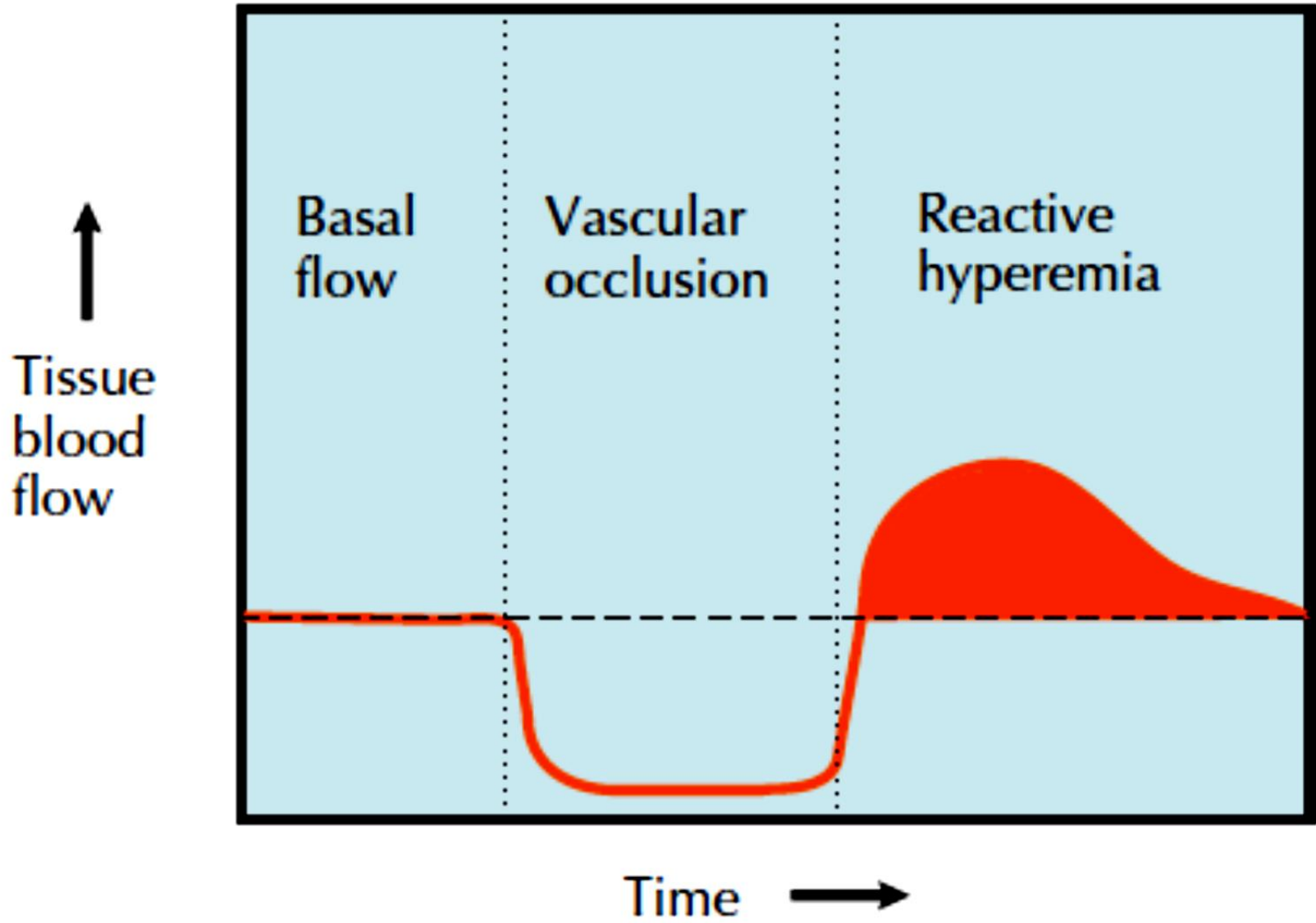
# Reactive hyperaemia

**Circulation to an organ or tissue is cut off (1-2) minutes. The organ or tissue continues metabolising & producing vasodilators (no circulation).**







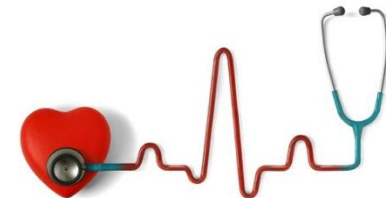
**When circulation is restored, the local arterioles dilate maximally and blood flow is very high.**



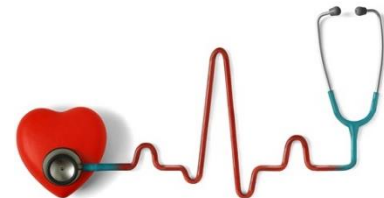


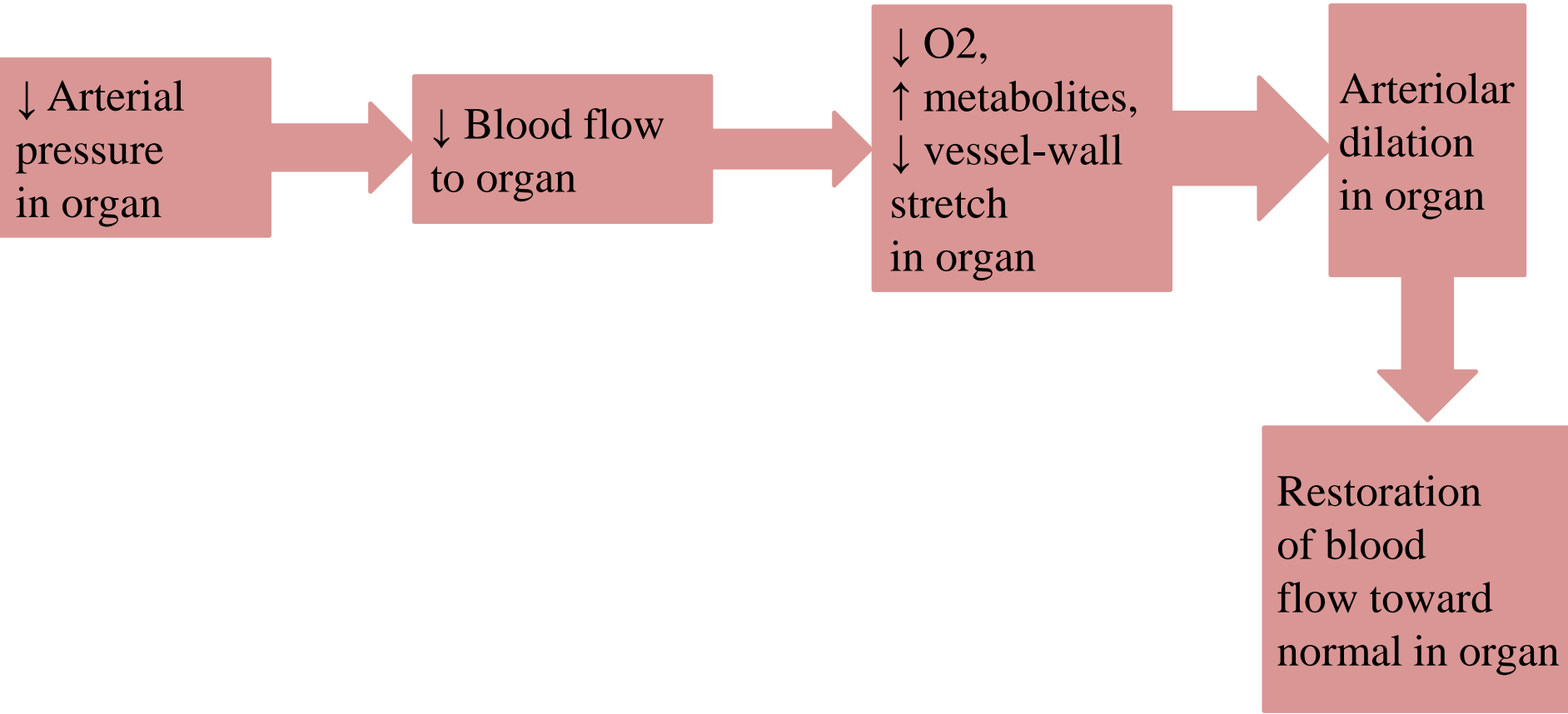
# Autoregulation

-  Autoregulation is the capacity of tissues to regulate their own blood flow.
-  Vascular beds have an intrinsic capacity to compensate for moderate changes in perfusion pressure.
-  The contraction of VSMCs that underlies autoregulation is autonomous.
-  That is entirely local control.

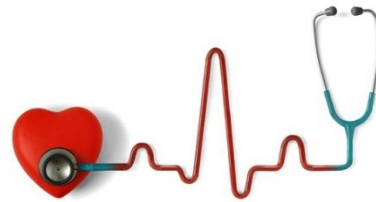


- ❖ Independent of neural or endocrine mechanisms.
- ❖ Both myogenic & metabolic mechanisms play an important role.
- ❖ Autoregulation is very important for brain, heart & kidneys. It has also been observed in the mesentery, skeletal muscles and liver.






🚒 ↑ in BP would produce opposite changes

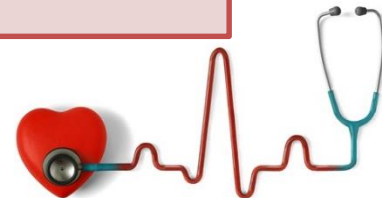


# Veins

 **Veins serve as low-resistance conduits for venous return and they are high capacitance vessels.**

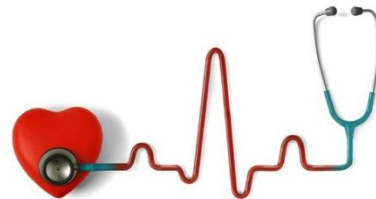
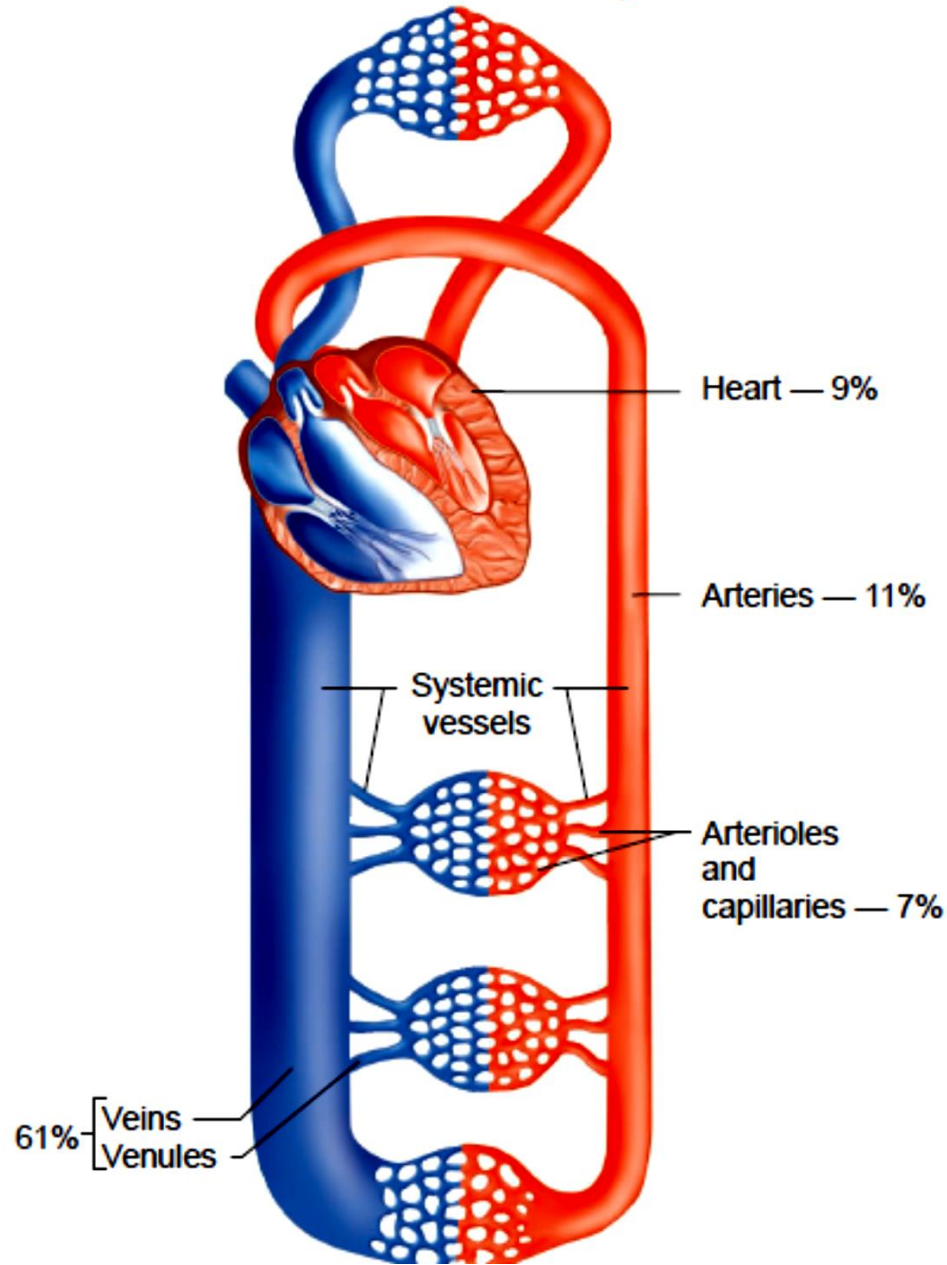
 **The pressure in veins is much affected by volume of blood they contain.**

 **The veins contain most of blood in the vascular system.**



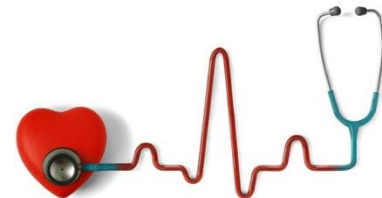


Pulmonary circulation — 12%

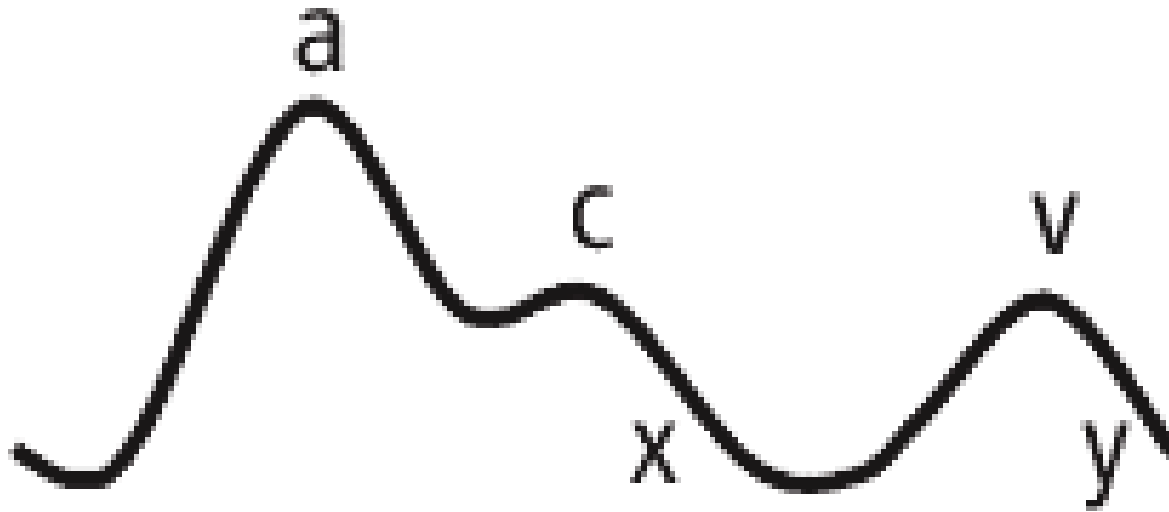


**Central  
venous  
pressure**

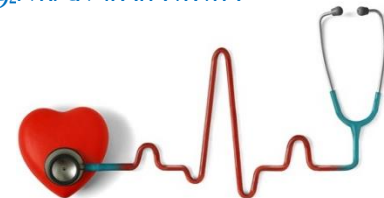
 **The P. in the great  
veins supplying  
the heart.**



# Central venous pressure



<https://www.semanticscholar.org/paper/Central-venous-pressure-monitoring.-Chow-Dilley/a5cfc1f12a1b50a0b7f781c942bc02563d3767d3>



## Venous return

 **The rate of blood  
flow back to the  
heart.**



*Thank you*

