Podcast (Video Recorded Lecture Series):
Aerobic Exercise for the USMLE Step One Exam

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Tutorial Services
(check website for details)

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Sloth Exercising

Why don't we die during exercise?

SNS activated→ α-1 adrenergics stimulated

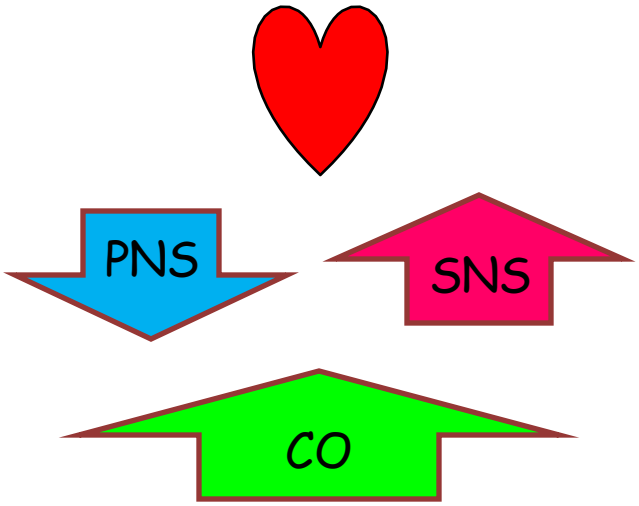
BP should rise; TPR_{Ω} should rise

Cardiac work should increase; vessel walls should thicken

We should die a miserable, cold, lonely death.

Why don't we?

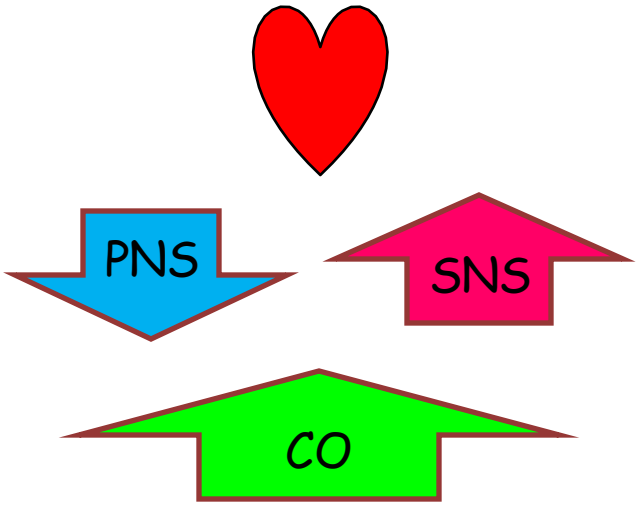
Exercise
(Central and Local Responses)



	HR	Contractility	VR
SNS	β1	β1	Venoconstriction
PNS	↓ Ach → M_2		

Exercise
(Central and Local Responses)

Coronary Vessel Vasodilation →
↑ Coronary blood flow

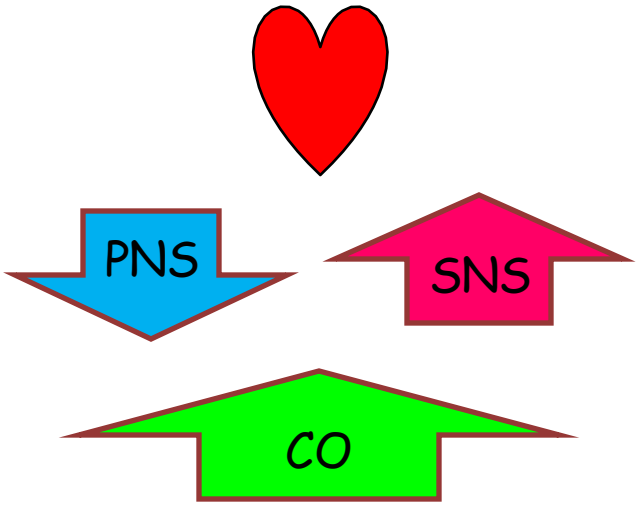


Vasodilate

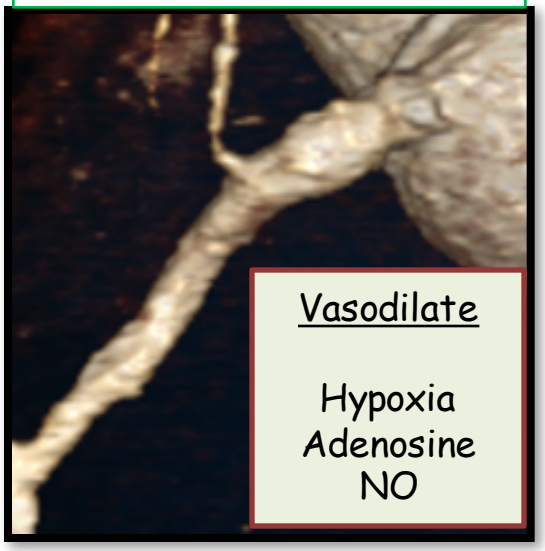
Hypoxia
Adenosine
NO

	HR	Contractility	VR
SNS	β1	β1	Venoconstriction
PNS	↓ Ach → M_2		

Exercise
(Central and Local Responses)



↑ Coronary Blood Flow



Vasodilate
Lactate, K+, Adenosine

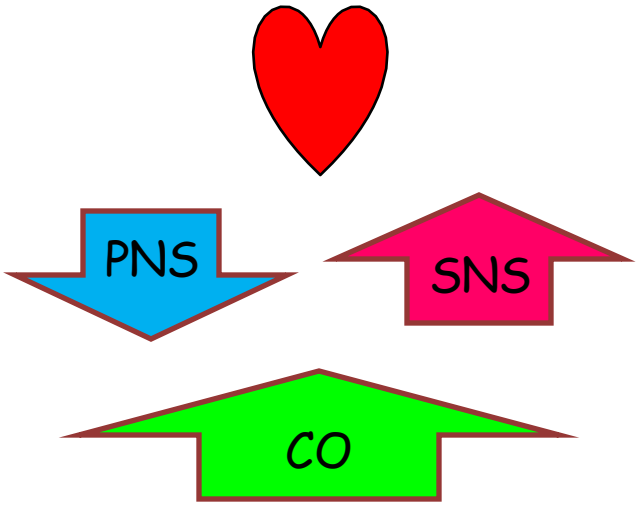
Result: ↓ Afterload

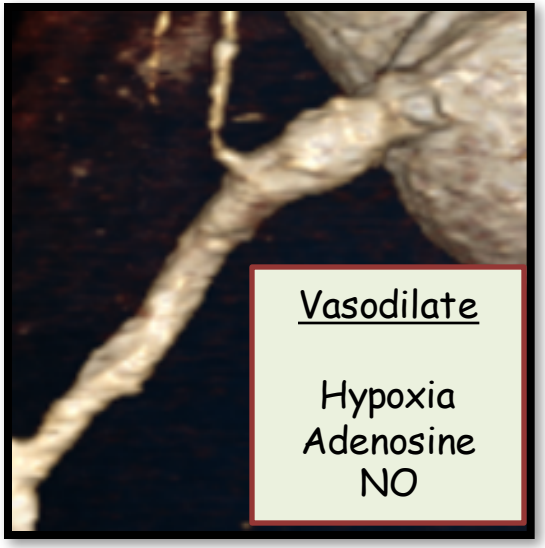
↑ Stroke Volume

	HR	Contractility	VR
SNS	β1	β1	Venoconstriction
PNS	↓ Ach → M_2		

Epi → β-2 stimulation

Exercise
(Central and Local Responses)





Vasodilate
Lactate, K+, Adenosine

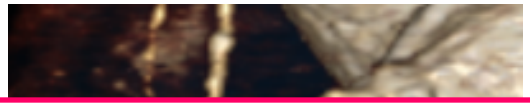
↓ Afterload

	HR	Contractility	VR
SNS	β1	β1	α Venoconstriction
PNS	↓ Ach → M_2		

Vasoconstrict
SNS → α-1

Exercise
(Central and Local Responses)

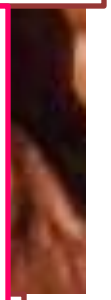
Vasodilate
Lactate, K+, Adenosine

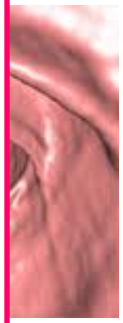


PN

SN

PN





Notes:

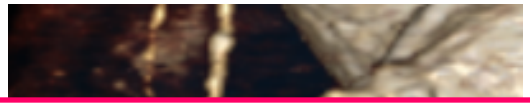
PaO2, PCO2 and pH
play only minor role in the exercise response.

The redistribution of Cardiac Output during exercise is dependent on organ specific changes in arteriolar resistance.

Local mediators (active hyperemia)

Exercise
(Central and Local Responses)

Vasodilate
Lactate, K+, Adenosine



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SN

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Notes:

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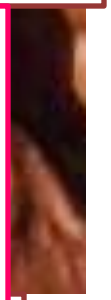
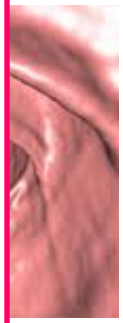


Pulmonary Response:

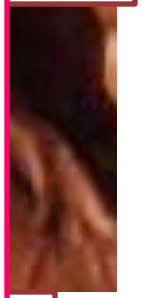
- Lung Volumes: ↑TV; recruit apical dead space
- Airways: Bronchodilation (CW, β-2 agonist/epinephrine)
- Vasculature: Right side must equal Left; capillary recruitment

Minor change in respiratory rate w/ sustained activity



Exercise
(Central and Local Responses)

sine



Vasoconstrict
SNS → α1

Fun Fact:

If TPR decreases due to skeletal mm vessels vasodilating…

…why does the MAP increase?

OR

Given the degree of SNS input, why doesn't the MAP increase more?

They want you to appreciate this balance between stroke volume, systemic RΩ and BP.

SV
α-1
VR

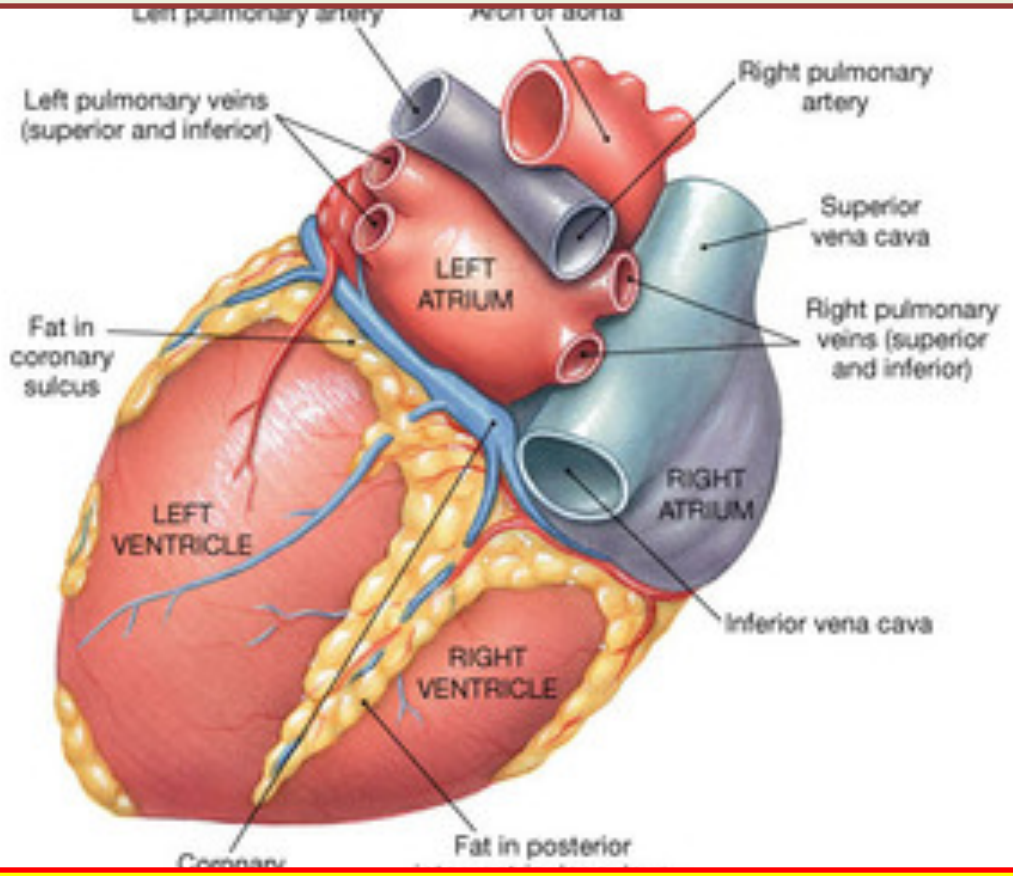
MAP =
HR x SV x TPR_{Ω}

Local mediators
β-2 Stimulation

BP rises but systemic vascular resistance does not

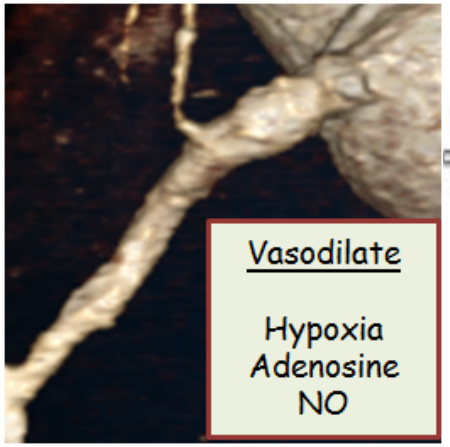
Myocardial Oxygen Consumption

'Max'ed out?'



The only way to increase cardiac uptake of O_2 is by increasing coronary blood flow.

Exercise does INCREASE myocardial oxygen consumption but not significantly (80% → 90% extraction)



Vasodilate

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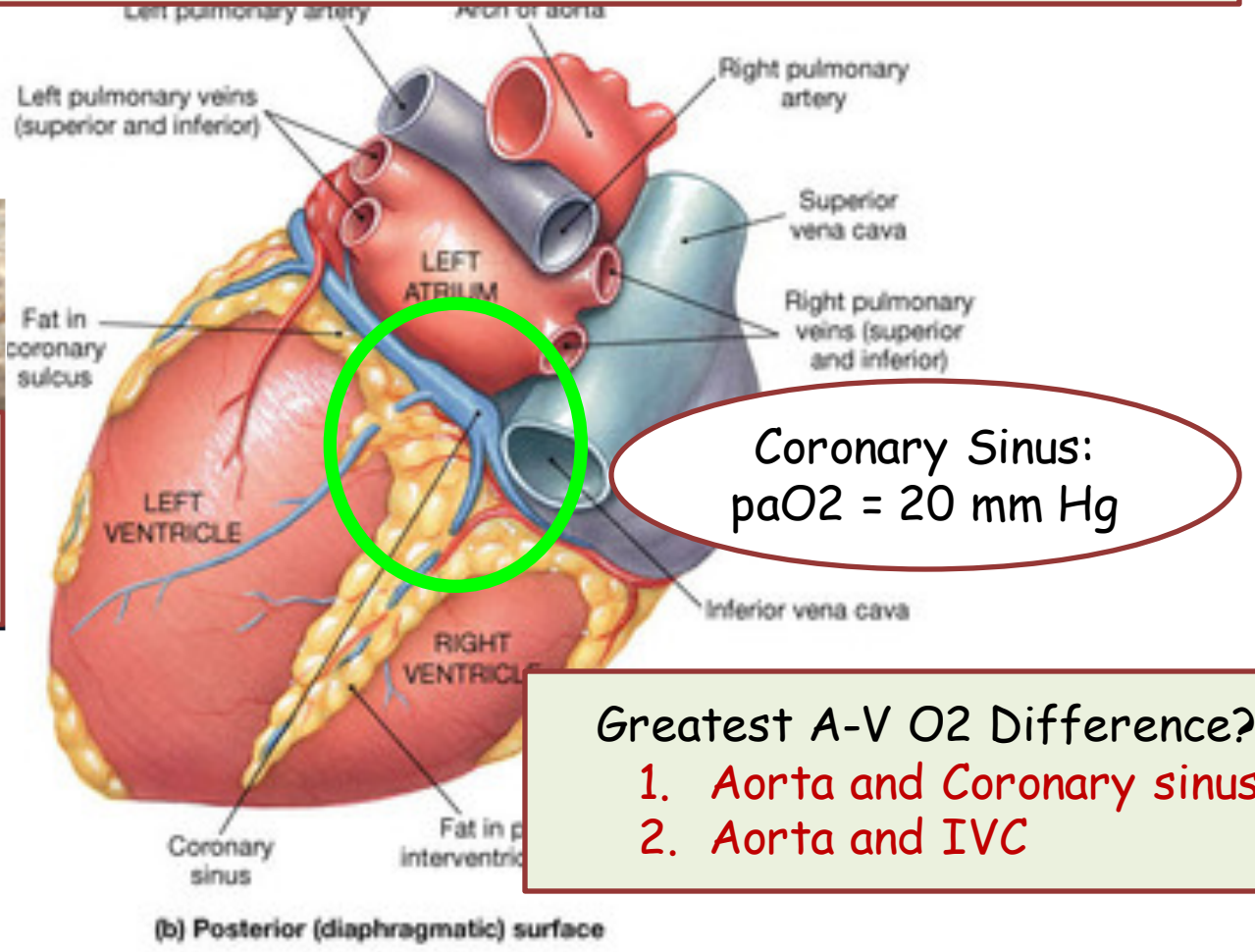


Coronary Sinus:
paO2 = 20 mm Hg

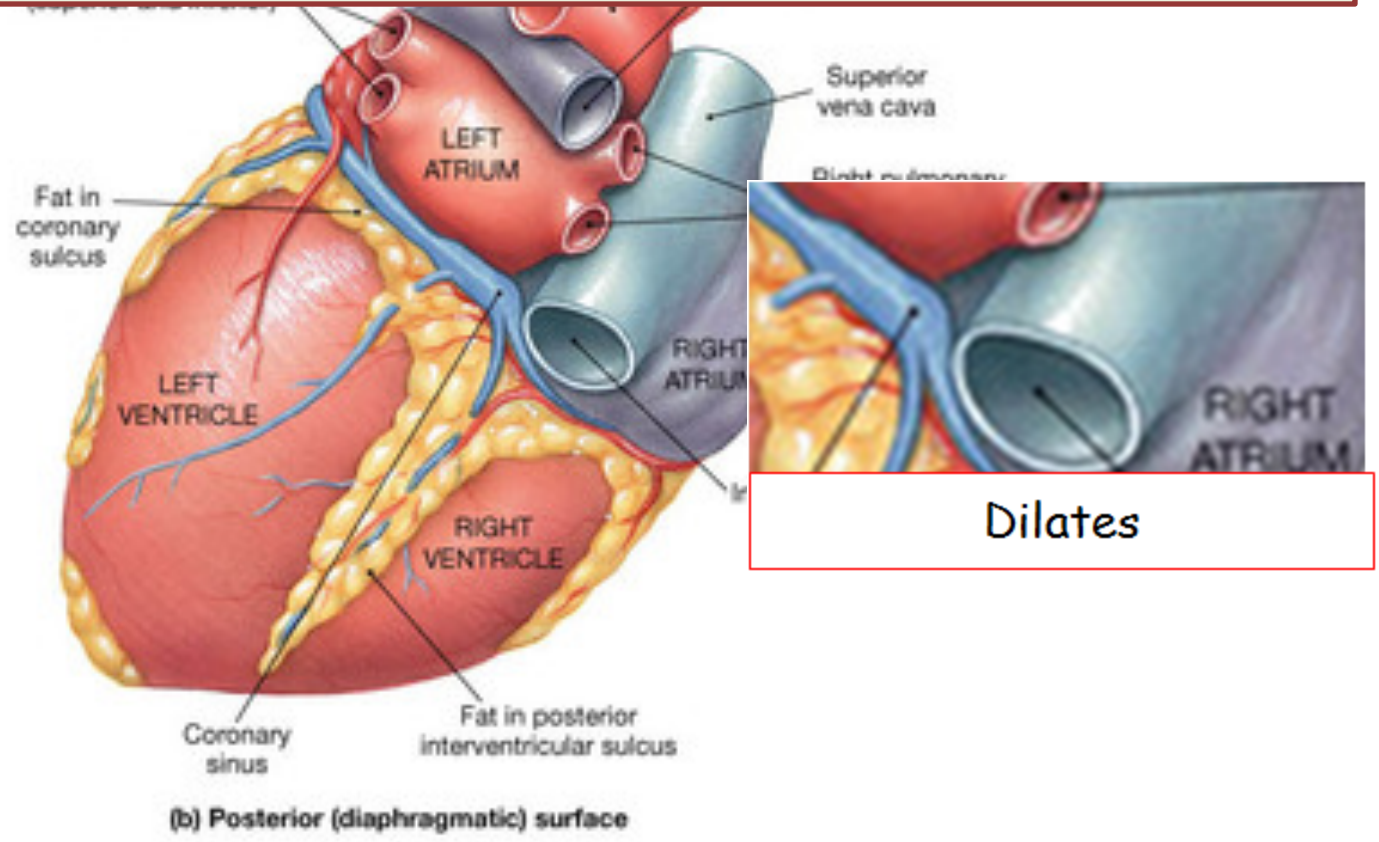
Greatest A-V O2 Difference?

1. Aorta and Coronary sinus
2. Aorta and IVC

Not to beat the coronary sinus to death, but where does it drain?

Answer: Right atrium

Q. What happens with pulmonary HTN/RA enlargement?



(b) Posterior (diaphragmatic) surface

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