Exercise impacts on Cardiovascular system

By

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1. The central command (anticipation of exercise)

→ ■■ **Impulses originate in the *motor cortex* or from *reflexes initiated in muscle proprioceptors* when exercise is anticipated.**

→ ■■ **initiates the following changes**

1. Sympathetic innervation enhancement

- is the primary regulator of blood flow to the skeletal muscle at **rest**
- The arterioles of skeletal muscle are densely innervated by sympathetic fibers. The veins also are innervated, but less densely.
- There are both **α1** and **β2** receptors on the blood vessels of skeletal muscle.
- Stimulation of **α1 receptors** causes **vasoconstriction.**
- Stimulation of **β2 receptors** causes **vasodilation.**

Stimulation of **Sympathetic post ganglionic N.F** causes **vasodilation** during **exercise.**

Sympathetic outflow to the heart and blood vessels is increased. At the same time, parasympathetic outflow to the heart is decreased. As a result, heart rate and contractility (stroke volume) are increased

Cardiac output is increased, primarily as a result of the increased heart rate and, to a lesser extent, the increased stroke volume.

Venous return is increased as a result of muscular activity and venoconstriction.

→ Increased venous return provides more blood for each stroke volume

Arteriolar resistance in the skin, splanchnic regions, kidneys, and inactive muscles is increased. Accordingly, blood flow to these organs is decreased.

In summary, there is vasoconstriction in some vascular beds so that blood flow can be redistributed to the exercising skeletal muscle and the heart, with blood flow being maintained in essential organs such as the brain.

2. Increased metabolic activity of skeletal muscle

Blood flow in skeletal muscle exhibits autoregulation and active and reactive hyperemia.

-Demand for **O2** in skeletal muscle varies with metabolic activity level, and blood flow is regulated to meet demand.

-During **exercise,** when demand is high, these local metabolic mechanisms are dominant.

Vasodilator metabolites (lactate, K+, and **adenosine)** accumulate because of increased metabolism of the exercising muscle.

These metabolites cause arteriolar dilation in the active skeletal muscle, thus increasing skeletal muscle blood flow. As a result of the increased blood flow, **O2** delivery to the muscle is increased.

This vasodilation accounts for the **decrease** **in** **TPR** that occurs with **exercise.**

Mechanical effects during exercise **temporarily** compress the arteries and decrease blood flow. During the post-occlusion period, **reactive hyperemia** increases blood flow.

Intrinsic regulation

→ **Intrinsic regulation (Auto-regulation)**

It is the ability of the cardiac muscle to increase its force of contraction independent on neither nervous nor chemical factors **but dependent on the loads.**

This mechanism **affects only the SV** by affecting contractility.

Auto-regulation include: ***hetero-metric*** & ***homeo-metric mechanisms***.

	Hetero-metric autoregulation
Definition	It is the ability of the cardiac muscle to ⇧ its force of contraction **as a result of ⇧ EDV.** (2ry to ⇧ **length**).
Stimulus	⇧ EDV (⇧VR).
Load	⇧ **Pre load.**
Mechanism	Starling law.
Timing	Starts 30 **seconds** after ⇧ VR.
Explanation	At the **beginning** of exercise. - There is increase in VR but the COP is not increased. - So, the heart is dilated to accommodate excess VR. - ⇧ VR ⇨ ⇧ EDV ⇨ ⇧ force of contraction ⇨ ↑ SV ⇨ ⇧ COP.
Limits	Over stretch.
⇧ SV	At expense of ⇧ EDV.
In H. failure	Present.

	Homeo-metric autoregulation
Definition	It is the ability of the cardiac muscle to ⇧ its force of contraction **without ⇧ EDV.** (without ⇧ **length**).
Stimulus	⇧ Aortic pressure (⇧ ABP).
Load	⇧ **After load.**
Mechanism	⇧ Intraventricular pressure and aortic pressure produced by increase in EDV and increased contractility during heterometric autorgulation.
Timing	Starts 2-5 **minutes** after ⇧ VR. Present as long as the venous return is elevated.
Explanation	In **prolonged** exercise. - After heterometric autoregulation, the EDV starts to return gradually to normal level due to rapid pumping of the blood. - However, the force of contraction is still high and so the SV is still elevated.
Limits	⇩ Energy store in heart. Heart failure.
⇧ SV	At expense of ⇩ ESV.
In H. failure	Lost.

table 3.5 Summary of Effects of Exercise

Parameter	Effect
Heart rate	↑↑
Stroke volume	↑
Cardiac output	↑↑
Arterial pressure	↑ (slight)
Pulse pressure	↑ (due to increased stroke volume)
TPR	↓↓ (due to vasodilation of skeletal muscle beds)
AV O_2 difference	↑↑ (due to increased O_2 consumption)

AV = arteriovenous; TPR = total peripheral resistance.

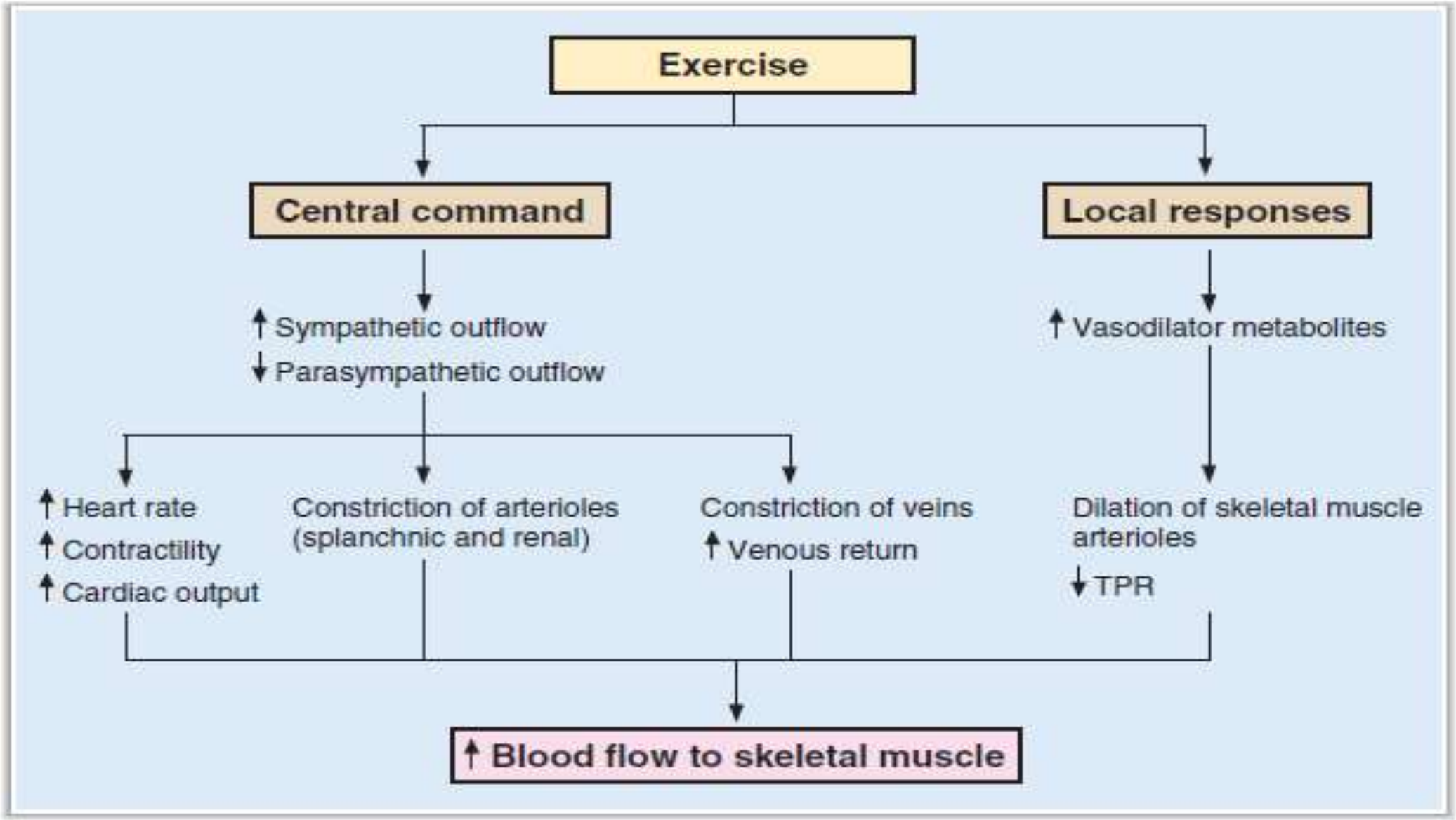

FIGURE 3.20 Cardiovascular responses to exercise. TPR = total peripheral resistance.

Active hyperemia illustrates the concept that blood flow to an organ is proportional to its metabolic activity. If metabolic activity in skeletal muscle increases as a result of exercise, then blood flow to the muscle will increase proportionately to meet the increased metabolic demand.

Reactive hyperemia is an increase in blood flow *in response* to or *reacting* to a prior period of decreased blood flow. For example, reactive hyperemia is the increase in blood flow to an organ that occurs following a period of arterial occlusion. During the occlusion, an O2 debt is accumulated. The longer the period of occlusion, the greater the O2 debt and the greater the subsequent increase in blood flow


Thank you