Effect of exercise on Cardiovascular system

BY

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Introduction

An adequate O2 supply is essential for performance of muscular exercise.

This is achieved through alterations in both the **circulatory** and **respiratory** systems.

The resting O2 consumption (**250 ml/min**) is markedly increased during exercise .

The circulatory adjustments during exercise **aim to increase the muscular blood flow**.

This may increase from the resting level (**2-4 ml/100gm /min**) up to **30-fold** and it occurs mainly in the periods between muscle contractions (during which the blood vessels are not compressed).

The muscular blood flow increases as a result of both **systemic circulatory changes** as well as **local changes in the active muscles**

A- Systemic circulatory changes

(1) Increase of the cardiac output

The CO markedly increases (**up to 35 liters/minute**) due to an increase in both the **heart rate** and **stroke volume**, and the magnitude of its increase is **proportional** to the increase in **O2 consumption**.

(a)The heart rate

This increases up to **180-200 beats/minute** as a result of :

-**Psychic stimuli** initiated in the cerebral cortex. These increase the heart rate before starting the exercise by **decreasing the vagal tone** and **stimulating the V.C.C**. (which **increases the sympathetic discharge** to the heart and the **secretion of catecholamines** from the adrenal medulla).

-**Bainbridge reflex (An increase in right atrial pressure, leads to heart acceleration)**initiated by the increased venous return.

-**ALAM - SMIRK reflex** (Voluntary muscle contraction, **leads to increase in heart rate)**

-**The increase in PCO2 & H+** which activate the **V.C.C** through stimulating the **Central & peripheral chemoreceptors**

-**Rise of the blood temperature**.

(b) The stroke volume

This increases due to **forceful ventricular contraction** that is produced by:

-The increased **sympathetic activity** & **catecholamines secretion**

-In normal hearts, **the EDV** is not increased during exercise .Accordingly, the Starling's mechanism plays **almost no role** in increasing the stroke volume in normal hearts during exercise. However, it becomes the main mechanism that increases the stroke volume in transplanted hearts because they are denervated, so they do not respond to the increased sympathetic activity (but they still respond to the circulating catecholamines)

The transplanted hearts beat by the **S-A rhythm** (100-110 beats/minute) and can pump about 70 % of the maximal CO of normal hearts.

(2) Increase of the venous return

This is increased **4-5 times** as a result of:

a- An increase in both the muscle and respiratory pumps .

b-Mobilization of blood from the viscera and other reservoirs .

c- Arteriolar dilatation in the active muscles

d- Venoconstriction produced by the increased sympathetic activity.

(3) Increase of the arterial blood pressure

The increased CO leads to rise of the mean arterial B.P. The systolic pressure increases markedly due to the increase in the stroke volume ,While the diastolic pressure is often unchanged or even falls because the total peripheral resistance falls as a result of the **marked V.D.** in the active muscles.

(4) Generalized V.C. (redistribution of blood)

Generalized **V.C** occurs in areas **other than the active muscles**, specially the **splanchnic area** and **skin,** so blood is shifted from the viscera and skin to the active muscles and the heart (where V.D. occurs).

This V.C is produced by the increased symp. activity & catecholamines secretion, which occur as a result of stimulation of the **VCC** by

(a) Psychic stimuli (b) Stimulating the chemoreceptors by the raised PCO2 & H+

* During exercise, the cerebral blood flow remains almost constant , While the coronary blood flow increases **about 5-fold**

*If the exercise is prolonged and the body temperature increases, the cutaneous vessels dilate to increase the rate of heat loss.

Also, the prolonged V.C of the renal vessels may result in **transient albuminuria** ,and together with the excessive sweating, it also leads to **oliguria**

B- Local changes in the active muscles

(1) V.D. of the muscle arterioles

This together with the above systemic effects, leads to a marked increase of the muscle blood flow (up to 30 fold), It occurs even before starting the exercise through activation of the **sympathetic V.D. system** and is then maintained and augmented by the effects of:

a-Certain metabolites e.g. K+ and adenosine.

b-The local O_2lack and increased PCO_2 and H+ .

c-The excess heat liberated in the active muscles.

d- The increased arterial B.P(which mechanically dilates the arterioles)

the arteriolar V.D. also leads to reduction of the peripheral resistance and slowing of the blood flow.

(2) Capillary changes

The precapillary sphincters are relaxed, so the open capillaries increase 10-100 times, and they become widely dilated.

These effects increase both the **capillary pressure** & **capillary permeability**, which lead to filtration of excessive amounts of interstitial fluid and local edema. Also some plasma proteins often escape in the filtrate, and by their osmotic pressure, they help filtration of more fluid (which may then occur along the whole length of the capillaries).

(3) Increase of the lymph flow

Large amounts of lymph are formed in active muscles due to

the excessive filtration of the interstitial fluid , and the lymph flow from these muscles **markedly increases** as a result of their increased pumping activity (which also increases the venous return from them).

This decreases the **local edema**, but it often does not abolish it.

(4) Increase of the O2 uptake

In active muscles, the **O2 consumption** may increase 60-100-fold as a result of the following effects:

a-The marked increase in the muscle blood flow .

b-More unloading of O2 from oxy-Hb: This is helped by the local increase of CO2, H+ and temperature, as well as by the increase in 2,3 DPG(=2.3diphosphoglycerate). All these factors decrease the Hb affinity to O2& shift the O2 dissociation curve to the right .

c-The greater O2 extraction from the blood: This is facilitated by the slow blood flow, and it results in rise of the arterio-venous O2 difference 3 times (from 5 to 15 ml%).

CO2 transport out of active muscles is also facilitated

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.

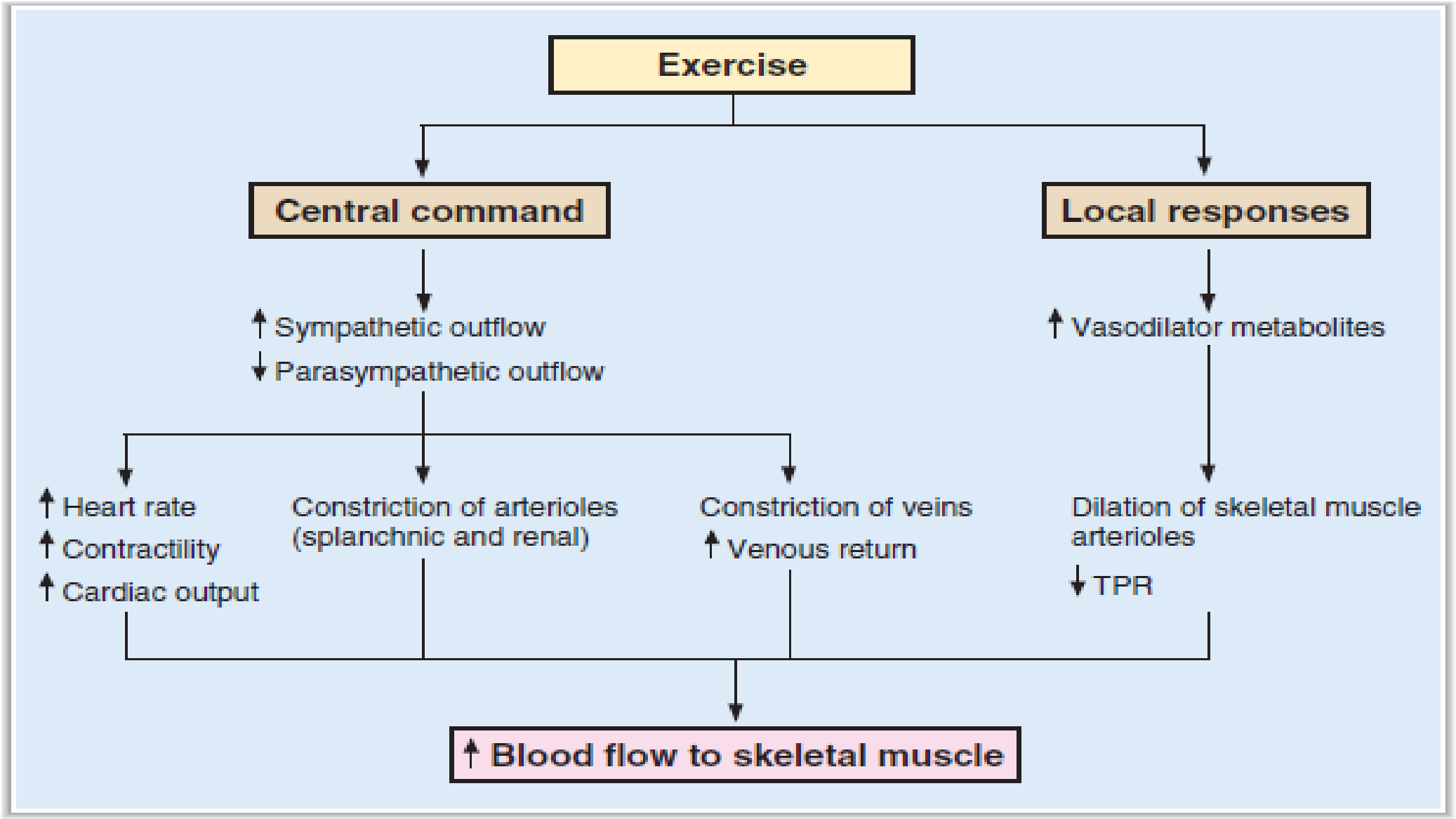

FIGURE 3.20 Cardiovascular responses to exercise. TPR = total peripheral resistance.

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