

Chapter  
**13**

**The Physiology of Training:  
Effect on  $VO_{2max}$ , Performance,  
Homeostasis & Strength**

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**Objectives**

1. Explain the basic principles of training: overload & specificity.
2. Contrast cross-sectional w/ longitudinal research studies.
3. Indicate the typical  $\Delta$  in  $VO_{2max}$  w/ endurance training programs, & the effect of the initial (pretraining) value on the magnitude of the  $\uparrow$ .
4. State the typical  $VO_{2max}$  values for various sedentary, active, & athletic populations.
5. State the formula for  $VO_{2max}$  using heart rate, stroke volume, & the a- $vO_2$  difference; indicate which of the variables is most important in explaining the wide range of  $VO_{2max}$  values in the population.

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**Objectives**

6. Discuss, using the variables identified in objective 5, how the  $\uparrow$  in  $VO_{2max}$  comes about for the sedentary subject who participates in an endurance training program.
7. Define *preload*, *afterload*, & *contractility*, & discuss the role of each in the  $\uparrow$  in the maximal stroke volume that occurs w/ endurance training.
8. Describe the  $\Delta$ s in muscle structure that are responsible for the  $\uparrow$  in the maximal a- $vO_2$  difference w/ endurance training.
9. Describe the underlying causes for the  $\downarrow$  in  $VO_{2max}$  that occurs w/ cessation of endurance training.

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## Objectives

10. Describe how the capillary & mitochondrial  $\Delta$ s that occur in muscle as a result of an endurance training program are related to the following adaptations to submaximal exercise:
  - a. a lower  $O_2$  deficit
  - b. an  $\uparrow$ d utilization of FFA & a sparing of bld glucose & muscle glycogen
  - c. a reduction in lactate &  $H^+$  formation
  - d. an  $\uparrow$  in lactate removal
11. Discuss how  $\Delta$ s in "central command" & "peripheral feedback" following an endurance training program can lower the heart rate, ventilation, & catecholamine responses to a submaximal exercise bout.
12. Contrast the role of neural adaptations w/ hypertrophy in the  $\uparrow$  in strength that occurs w/ resistance training.

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## Exercise: A Challenge to Homeostasis

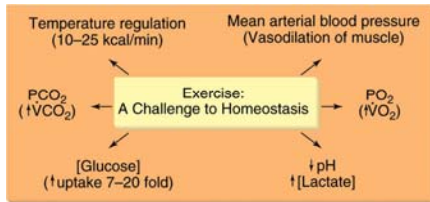


Figure 13.1

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## Principles of Training

- Overload
- Specificity
- Reversibility

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Research Designs to Study Training

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Research Designs to Study Training

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Endurance Training &  $VO_{2max}$

- Training to  $\uparrow VO_{2max}$

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## Range of $VO_{2max}$ Values in the Population

**TABLE 13.1**  $VO_2$  Max Values Measured In Healthy and Diseased Populations

Population	Males	Females
Cross-country skiers	84	72
Distance runners	83	62
Sedentary; young	45	38
Sedentary; middle-aged adults	35	30
Post myocardial infarction patients	22	18
Severe pulmonary disease patients	13	13

Values are expressed in  $ml \cdot kg^{-1} \cdot min^{-1}$ .  
 Taken from Saltin and Åstrand (102), Åstrand and Rodahl (5), and Howey and Franks (58).

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## A Closer Look 13.1 The HERITAGE Family Study

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## In Summary

- Endurance training programs that  $\uparrow VO_{2max}$  involve a large muscle mass in dynamic activity for 20- to 60-minutes/session, 3- to 5- times/week, at an intensity of 50% to 85%  $VO_{2max}$ .
- Although  $VO_{2max}$   $\uparrow$ s an average of about 15% as a result of an endurance training program, the largest  $\uparrow$ s are associated w/ deconditioned or patient populations having very low pretraining  $VO_{2max}$  values.
- Genetic predisposition accounts for 40% to 60% of one's  $VO_{2max}$  value. Very strenuous &/or prolonged training can  $\uparrow VO_{2max}$  in normal sedentary individuals by more than 40%.

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## Calculation of $\dot{V}O_{2\max}$

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## Differences in $\dot{V}O_{2\max}$ Values Among Populations

**TABLE 13.2** Physiological Basis for Differences in  $\dot{V}O_{2\max}$  in Different Populations

Population	$\dot{V}O_{2\max}$ ( $\text{ml} \cdot \text{min}^{-1}$ )	=	Heart Rate ( $\text{beats} \cdot \text{min}^{-1}$ )	×	Stroke Volume ( $\text{l} \cdot \text{beat}^{-1}$ )	×	a-v $\text{O}_2$ Difference ( $\text{ml} \cdot \text{O}_2 \cdot \text{l}^{-1}$ )
Athletes	6,250	=	190	×	205	×	160
Normally active	3,500	=	195	×	112	×	160
Mitral stenosis	1,400	=	190	×	043	×	170

From L. B. Rowell, Human Circulation: Regulation During Physical Stress. Copyright © 1986 Oxford University Press, New York, N.Y. Reprinted by permission.

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## $\Delta$ s in $\dot{V}O_{2\max}$ w/ Training

**TABLE 13.3** Longitudinal Data on Changes in Maximal Oxygen Uptake

	$\dot{V}O_{2\max}$ ( $\text{l} \cdot \text{min}^{-1}$ )	HR max ( $\text{b} \cdot \text{min}^{-1}$ )	Stroke Volume ( $\text{ml} \cdot \text{beat}^{-1}$ )	Cardiac Output ( $\text{l} \cdot \text{min}^{-1}$ )	a-v $\text{O}_2$ Difference ( $\text{ml} \cdot \text{l}^{-1}$ )
Subject LM					
Before training	3.58	206	124	25.5	140
Four months	4.38	210	143	28.1	142
Eighteen months	4.53	205	149	30.5	149
Subject IS					
Before training	3.07	205	122	23.9	126
Four months	3.67	205	134	26.2	131
Thirty-two months	4.36	185	151	27.6	158
Fifty-one months	4.41	186	146	26.6	166

From B. Ekblom, "Effect of Physical Training on Oxygen Transport System in Man," *Acta Physiologica Scandinavica*, Supplement 328, Copyright © 1969 Blackwell Scientific Publications, Ltd., Oxford, England. Reprinted by permission.

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## Stroke Volume

- ↑d maximal stroke volume

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## Factors ↑ng Stroke Volume

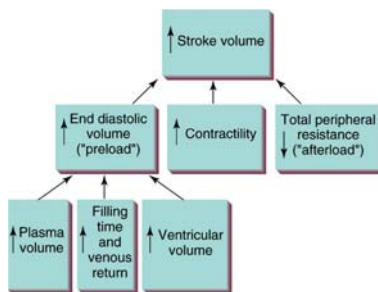


Figure 13.2

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## **A Closer Look 13.2** Why Do Some Individuals Have High $VO_{2max}$ Values w/out Training?

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## Arteriovenous O<sub>2</sub> Difference

- a-vO<sub>2</sub> max

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## Factors Causing ↑d VO<sub>2</sub>max

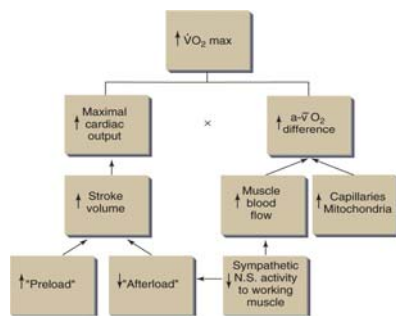


Figure 13.3

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## In Summary

- The training-induced ↑ in maximal stroke volume is due to both an ↑ in preload & a ↓ in afterload.
  - a. The ↑d preload is primarily due to an ↑ in end diastolic ventricular volume & the associated ↑ in plasma volume.
  - b. The ↓d afterload is due to a ↓ in the arteriolar constriction in the trained muscles, ↑ng maximal muscle bld flow w/ no Δ in the mean arterial BP.

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## In Summary

- In young sedentary subjects, 50% of the  $\uparrow$  in  $\text{VO}_{2\text{max}}$  is due to an  $\uparrow$  in the systemic  $\text{a-vO}_2$  difference. The  $\uparrow$  in  $\text{a-vO}_2$  difference is due to an  $\uparrow$  in the capillary density of the trained muscles that is needed to accept the  $\uparrow$  in maximal muscle bld flow. The greater capillary density allows for a sufficiently slow red bld cell transit time through the muscle, providing enough time for oxygen diffusion, which is facilitated by the  $\uparrow$  in the # of mitochondria.

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## Detraining & $\text{VO}_{2\text{max}}$

- $\downarrow$  in  $\text{VO}_{2\text{max}}$  w/ cessation of training

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## Detraining & $\Delta$ s in $\text{VO}_{2\text{max}}$ & Cardiovascular Variables

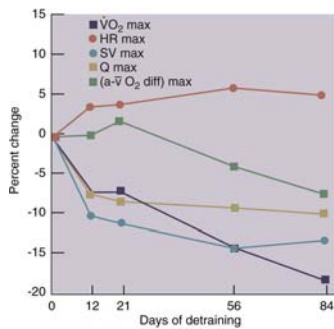


Figure 13.4

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### In Summary

- The ↓ in  $\text{VO}_{2\text{max}}$  w/ cessation of training is due to both a ↓ in maximal stroke volume & a ↓ in oxygen extraction, the reverse of what happens w/ training.

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### Effects of Endurance Training on Performance

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### Structural & Biochemical Adaptations to Endurance Training

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## Δs in Oxidative Enzymes w/ Training

**TABLE 13.4** Succinate Dehydrogenase Activity in Thigh Muscle Fiber Types in Response to Conditioning and Deconditioning

Fitness Level	Range of $\dot{V}O_2$ max ( $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ )	Muscle Fiber Type		
		Type I	Type IIa ( $\mu\text{mol} \cdot \text{g}^{-1} \cdot \text{min}^{-1}$ )	Type IIc
Deconditioned	30-40	5.0	4.0	3.5
Sedentary	40-50	9.2	5.8	4.9
Conditioning (months)	45-55	12.1	10.2	5.5
Endurance athletes	>70	23.2	22.1	22.0

Adapted from Sahn and Golbick (105)

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## Time Course of Training/Detraining Mitochondrial Δs

- Training
- Detraining

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## Time Course of Training/Detraining Mitochondrial Δs

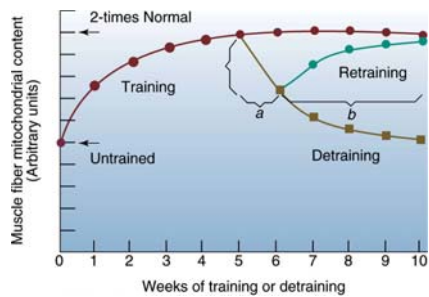


Figure 13.5

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**A Closer Look 13.3**  
**Role of Exercise Intensity & Duration on Mitochondrial Adaptations**

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**$\Delta$ s in Citrate Synthase Activity w/ Exercise**

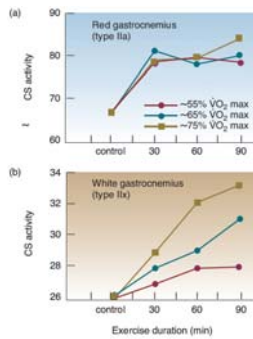


Figure 13.6

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**Biochemical Adaptations & the Oxygen Deficit**

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## Mitochondrial # & ADP Concentration Needed to ↑ $\text{VO}_2$

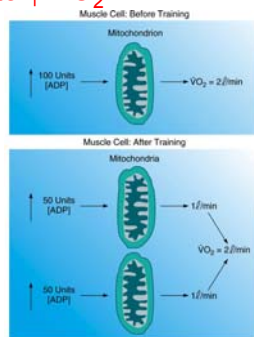


Figure 13.7

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## Endurance Training Reduces the $\text{O}_2$ Deficit

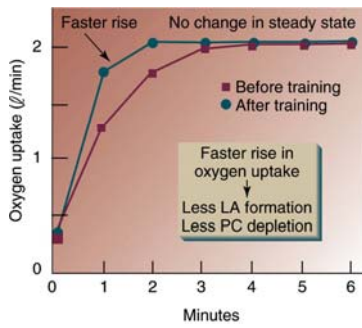


Figure 13.8

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## Biochemical Adaptations & the Plasma Glucose Concentration

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## Effect of Mitochondria & Capillaries on Free-Fatty Acid & Glucose Utilization

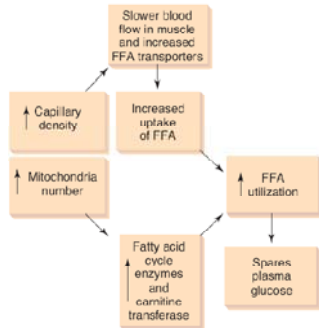


Figure 13.9

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## In Summary

- The combination of the ↑ in the density of capillaries & the # of mitochondria per muscle fiber ↑s the capacity to transport FFA from the plasma → cytoplasm → mitochondria.
- The ↑ in the enzymes of the fatty acid cycle ↑s the rate of formation of acetyl-CoA from FFA for oxidation in the Krebs cycle. This ↑ in fat oxidation in endurance-trained muscle spares both muscle glycogen & plasma glucose, the latter being a focal point of homeostatic regulatory mechanisms. These points are summarized in Figure 13.9.

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## Biochemical Adaptations & bld pH

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## Mitochondrial & Biochemical Adaptations & bld pH

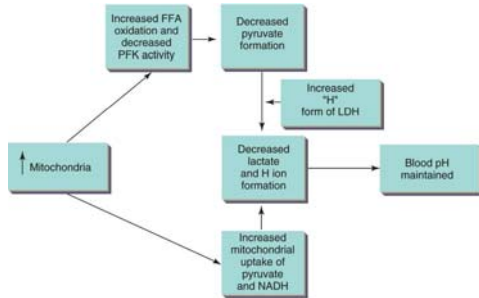


Figure 13.10

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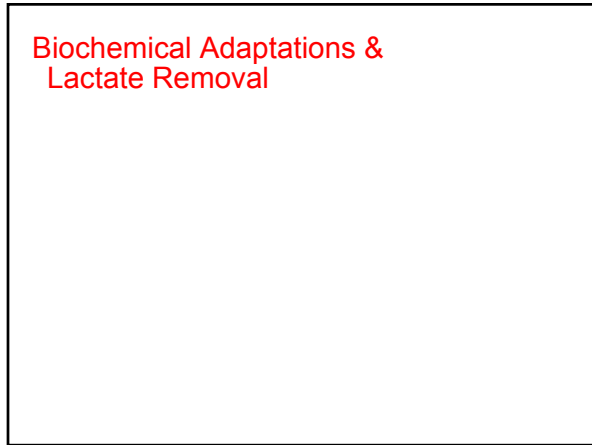
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## Biochemical Adaptations & Lactate Removal




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## Redistribution of bld Flow & Lactate Removal

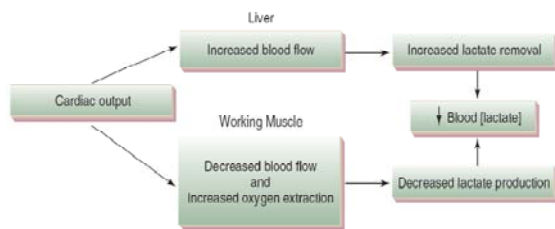


Figure 13.13

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### In Summary

- Mitochondrial adaptations to endurance training include an  $\uparrow$  in the enzymes involved in oxidative metabolism: Krebs cycle, fatty-acid ( $\beta$ -oxidation) cycle, & the electron transport chain.
- Those mitochondrial adaptations result in the following:
  - a. a smaller  $O_2$  deficit due to a more rapid  $\uparrow$  in  $VO_2$  at the onset of work
  - b. an  $\uparrow$  in fat metabolism that spares muscle glycogen & bld glucose
  - c. a reduction in lactate &  $H^+$  formation that helps to maintain the pH of the bld
  - d. an  $\uparrow$  in lactate removal

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### Links Between Muscle & Systemic Physiology

- **Biochemical adaptations to training influence the physiological response to exercise**

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### Peripheral & Central Control of Cardiorespiratory Responses

- **Peripheral feedback from working muscles**

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### Peripheral Control of Heart Rate, Ventilation & Bld Flow

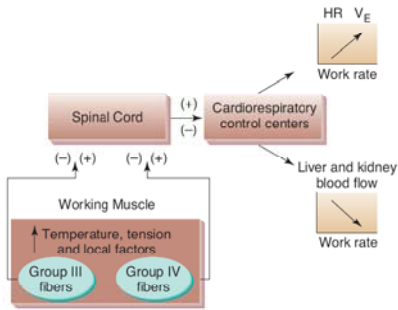


Figure 13.15

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### Central Control of Cardiorespiratory Responses

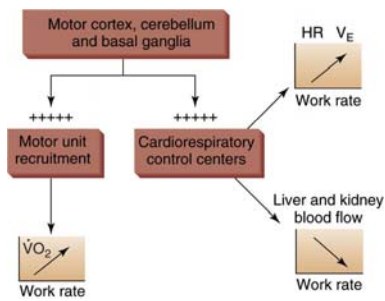


Figure 13.16

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### In Summary

- The biochemical  $\Delta$ s in muscle due to endurance training influence the physiological responses to exercise. The reduction in "feedback" from chemoreceptors in the trained muscle & a reduction in the need to recruit motor units to accomplish a work task results in reduced sympathetic nervous system, heart rate & ventilation responses in submaximal exercise.

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## Physiological Effects of Strength Training

- Muscular strength
- Muscular endurance
- Strength training

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## *A Closer Look 13.5* Aging, Strength, & Training

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## Physiological Mechanisms Causing ↑d Strength

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## Neural & Muscular Adaptations to Resistance Training

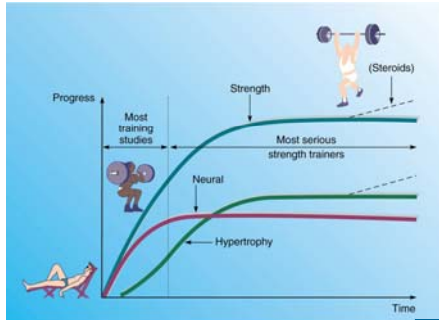


Figure 13.17

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## Neural Factors

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## Muscular Enlargement

- Hypertrophy
  
- Hyperplasia

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**The Winning Edge 13.1**  
**Periodization of Strength Training**

- Traditional training programs

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**Concurrent Strength & Endurance Training**

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**In Summary**

- ↑s in strength due to short-term (eight to twenty weeks) training are the results of neural adaptations, while gains in strength in long-term training programs are due to an ↑ in the size of the muscle.
- There is evidence both for & against the proposition that the physiological effects of strength training interfere w/ the physiological effects of endurance training.

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### Study Questions

1. Define the following principles of training: *overload* & *specificity*.
2. Give one example each of a cross-sectional study & a longitudinal study.
3. What are the typical  $\text{VO}_2$  max values for young men & women? Cardiac patients?
4. Given the formula for  $\text{VO}_2$  max using heart rate, stroke volume, & the a- $\text{vO}_2$  difference, which variable is most important in explaining the differences in  $\text{VO}_2$  max in different populations? Give a quantitative example.
5. Describe how the  $\uparrow$  in  $\text{VO}_2$  max comes about for the sedentary subject who undertakes an endurance training program.
6. Explain the importance of preload, afterload, & contractility in the  $\uparrow$  of the maximal stroke volume that occurs w/ endurance training.

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### Study Questions

7. What are the most important  $\Delta$ s in muscle structure that are responsible for the  $\uparrow$  in the maximal a- $\text{vO}_2$  difference that occurs w/ endurance training?
8. What causes the  $\text{VO}_{2\text{max}}$  to  $\downarrow$  following termination of an endurance training program?
9. Describe how the capillary & mitochondrial  $\Delta$ s that occur in muscle as a result of an endurance training program are related to the following adaptations to submaximal exercise:
  - a. a lower  $\text{O}_2$  deficit
  - b. an  $\uparrow$  utilization of FFA & sparing of bld glucose & muscle glycogen
  - c. a reduction in lactate &  $\text{H}^+$  formation that helps to maintain the pH of the bld
  - d. an  $\uparrow$  in lactate removal

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### Study Questions

10. Define *central command* and *peripheral feedback* & explain how  $\Delta$ s in muscle as a result of endurance training can be responsible for the lower heart rate, ventilation, & catecholamine response to a submaximal exercise bout.
11. In short-term training programs, what neural factors may be responsible for the  $\uparrow$  in strength?
12. Contrast hyperplasia w/ hypertrophy, & explain the role of each in the  $\uparrow$  in muscle size that occurs w/ long-term strength training.
13. Does strength training interfere w/ the physiological effects of endurance training?

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