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# The Physiology of Training: Effect on $VO_2$ Max, Performance, Homeostasis, and Strength

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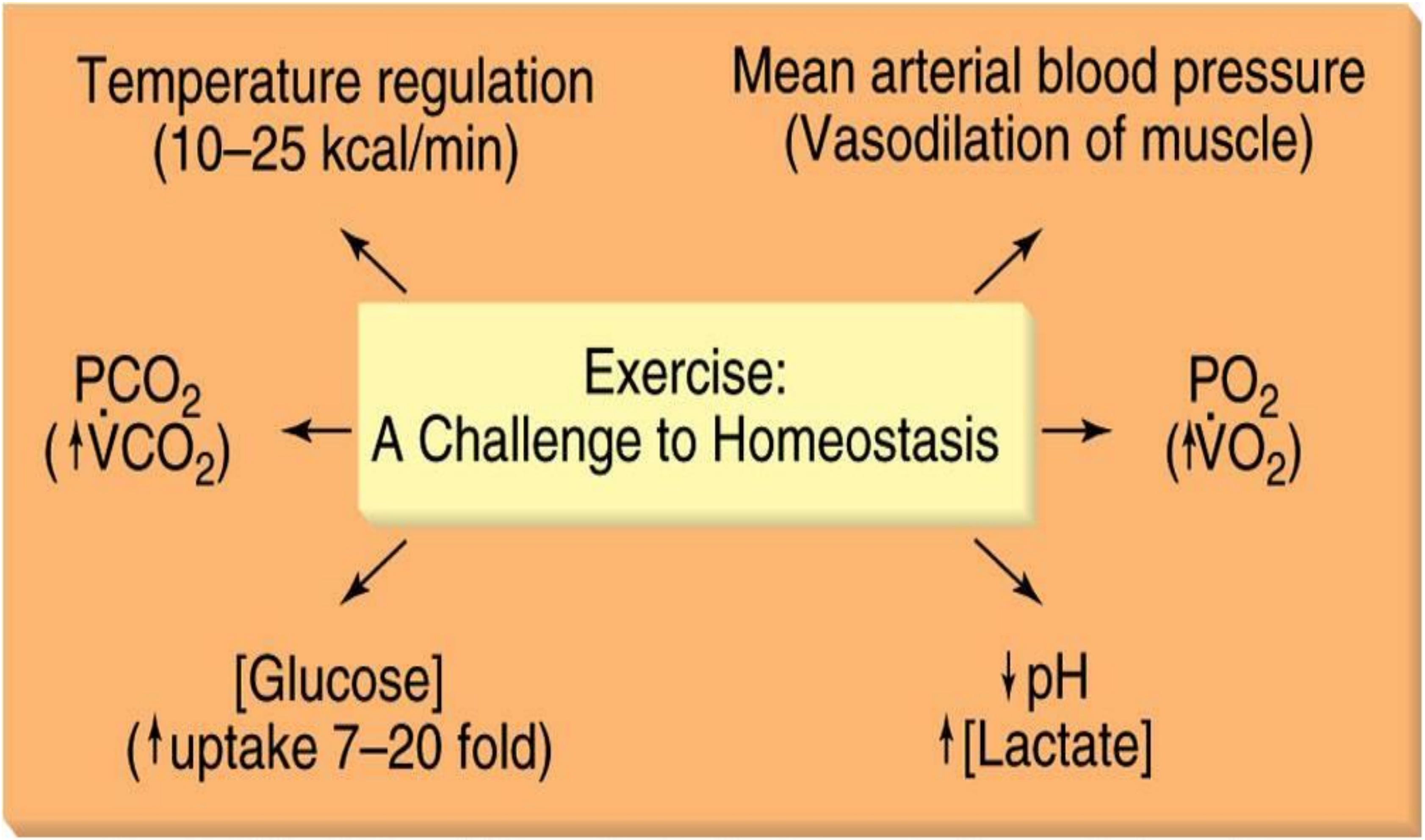
# Exercise: A Challenge to Homeostasis



# Principles of Training

- Overload
  - Training effect occurs when a system is exercised at a level beyond which it is normally accustomed
- Specificity
  - Training effect is specific to:
    - Muscle fibers involved
    - Energy system involved (aerobic vs. anaerobic)
    - Velocity of contraction
    - Type of contraction (eccentric, concentric, isometric)

- Reversibility
  - Gains are lost when overload is removed

# In Summary

- The principle of overload states that for a training effect to occur, a system or tissue must be challenged with an intensity, duration, or frequency of exercise to which it is unaccustomed. Over time the tissue or system adapts to this load. The reversibility principle is a corollary to the overload principle.
- The principle of specificity indicates that the training effect is limited to the muscle fibers involved in the activity. In addition, the muscle fiber adapts specifically to the type of activity: mitochondrial and capillary adaptations to endurance training.

# Research Designs to Study Training

- Cross-sectional studies
  - Examine groups of differing physical activity at one time
  - Record differences between groups
- Longitudinal studies
  - Examine groups before and after training
  - Record changes over time in the groups

# In Summary

- Cross-sectional training studies contrast the physiological responses of groups differing in habitual physical activity (e.g., sedentary individuals versus runners).
- Longitudinal training studies examine the changes taking place over the course of a training program.

# Endurance Training and $VO_2$ Max

- Training to increase $VO_2$ max
  - Large muscle groups, dynamic activity
  - 20–60 min, 3–5 times/week, 50–85% $VO_2$ max
- Expected increases in $VO_2$ max
  - Average = 15%
  - **2–3%** in those with high initial $VO_2$ max
    - Requires intensity of 95–100% $VO_2$ max
  - **30–50%** in those with low initial $VO_2$ max
    - Training intensity of 40–70% $VO_2$ max

# Range of $VO_2$ Max Values in the Population

**TABLE 13.1** $\dot{V}O_2$ **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), Åtrand and Rodahl (5), and Howley and Franks (58).

# In Summary

- Endurance training programs that increase $VO_2$ max involve a large muscle mass in dynamic activity for twenty to sixty minutes per session, three to five times per week, at an intensity of 50% to 85% $VO_2$ max.
- Although $VO_2$ max increases an average of about 15% as a result of an endurance training program, the largest increases are associated with deconditioned or patient populations having very low pretraining $VO_2$ max values.
- Genetic predisposition accounts for 40% to 60% of one’s $VO_2$ max value.



# Calculation of $VO_2$ Max

- Product of maximal cardiac output and arteriovenous difference

**$VO_2$ max = HR max x SV max x (a-v$O_2$) max**

- Differences in $VO_2$ max in different populations
  - Primarily due to differences in SV max
- Improvements in $VO_2$ max
  - 50% due to ↑SV
  - 50% due to ↑a-v$O_2$



# Changes in $VO_2$ Max with Training

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

| | $\dot{V}O_2$ max ($\ell \cdot min^{-1}$) | HR max ($b \cdot min^{-1}$) | Stroke Volume ($ml \cdot beat^{-1}$) | Cardiac Output ($\ell \cdot min^{-1}$) | a-$\bar{v}$ $O_2$ Difference ($ml \cdot \ell^{-1}$) |
|---|---|---|---|---|---|
| *Subject* | | | | | |
| 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* | | | | | |
| Before training | 3.07 | 205 | 122 | 23.9 | 126 |
| Four months | 3.87 | 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 |



# In Summary

- In young sedentary subjects, approximately 50% of the increase in $VO_2$ max due to training is related to an increase in maximal stroke volume (maximal heart rate remains the same), and 50% is due to an increase in the a-$vO_2$ difference.
- The large differences in $VO_2$ max in the normal population (2 versus 6 liters/min) are due to differences in maximal stroke volume.



# Stroke Volume

- Increased maximal stroke volume
  - ↑ Preload (EDV)
    - ↑ Plasma volume
    - ↑ Venous return
    - ↑ Ventricular volume
  - ↓ Afterload (TPR)
    - ↓ Arterial constriction
    - ↑ Maximal muscle blood flow with no change in mean arterial pressure
  - ↑ Contractility
- Changes occur rapidly
  - 11% increase in plasma volume with six days of training

# Factors Increasing Stroke Volume

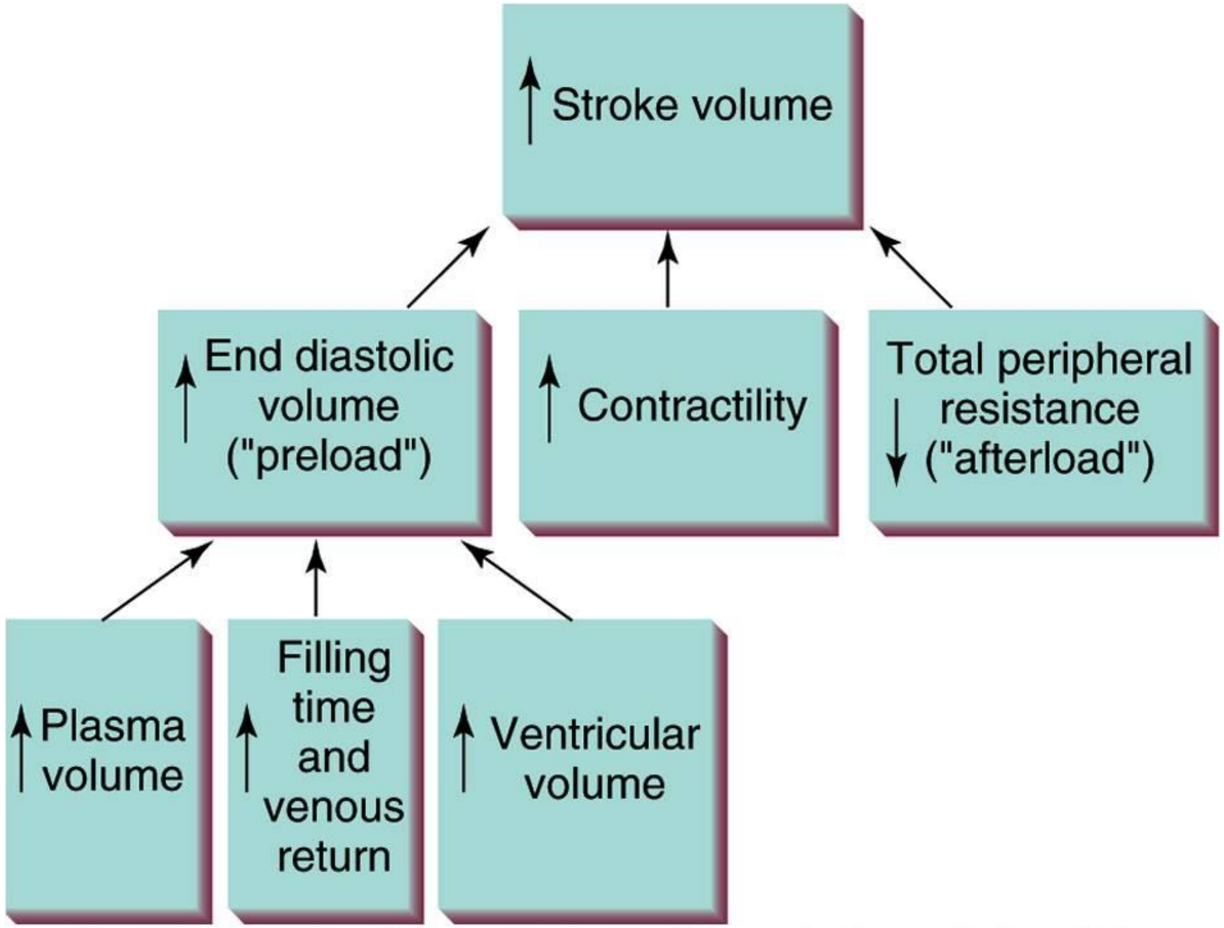



Figure 13.2



# Why Do Some Individuals Have High $VO_2$ Max Values Without Training?

- Some individuals have very high $VO_2$ max values with no history of training
  - $VO_2$ max = 65.3 ml•kg$^{-1}$•min$^{-1}$
  - Compared to 46.3 ml•kg$^{-1}$•min$^{-1}$ in sedentary with low $VO_2$ max
- Higher $VO_2$ max due to:
  - Higher maximal cardiac output, stroke volume, and lower total peripheral resistance
  - a-$vO_2$ difference
- Higher stroke volume linked to:
  - Higher blood volume and red cell volume



# Arteriovenous $O_2$ Difference

- a-v$O_2$ max
  - ↑ Muscle blood flow
    - ↓ SNS vasoconstriction
  - Improved ability of the muscle to extract oxygen from the blood
    - ↑ Capillary density
    - ↑ Mitochondial number

# Factors Causing Increased $VO_2$ Max

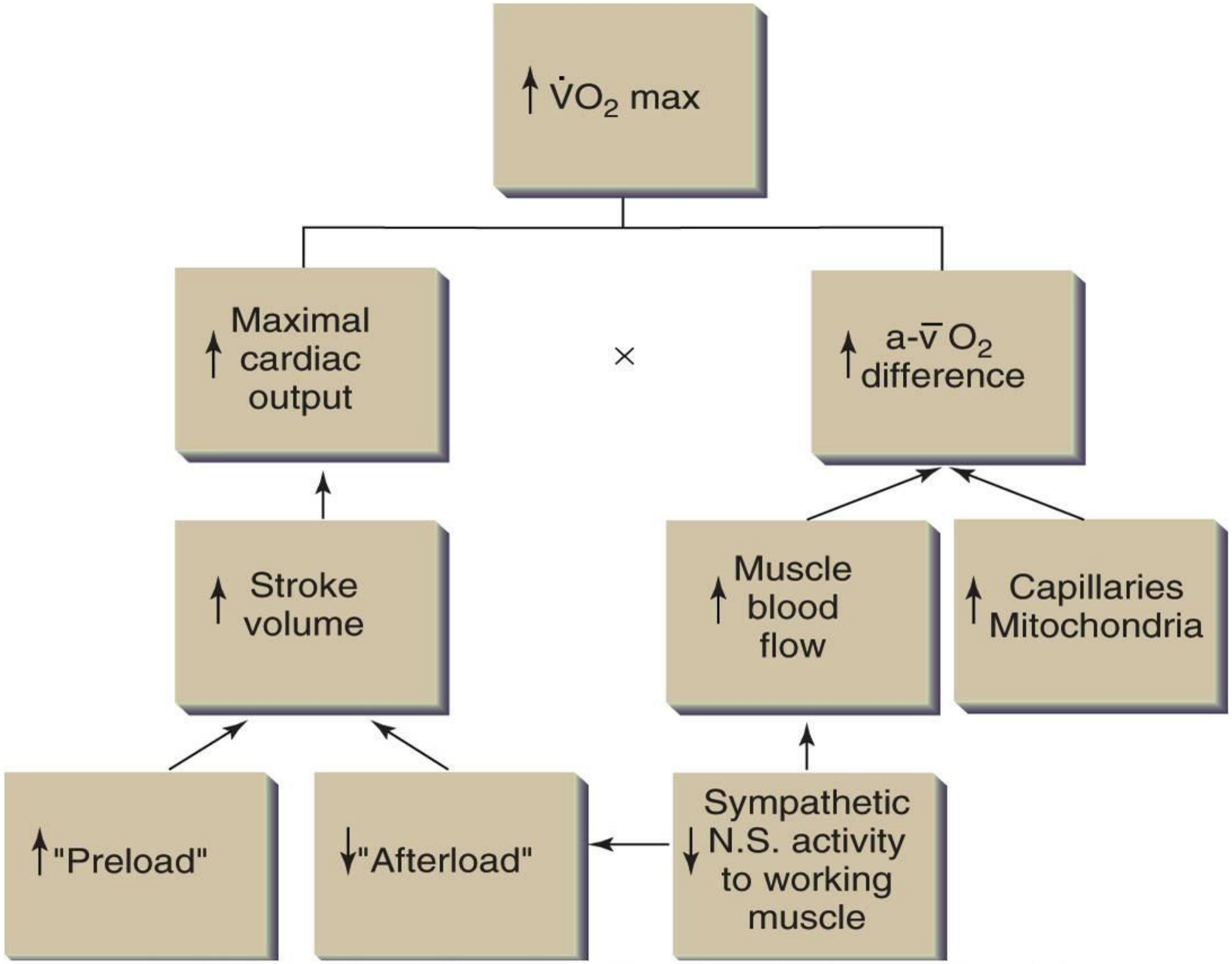



Figure 13.3



## In Summary

- The training-induced increase in maximal stroke volume is due to both an increase in preload and a decrease in afterload.
  a. The increased preload is primarily due to an increase in end diastolic ventricular volume and the associated increase in plasma volume.
  b. The decreased afterload is due to a decrease in the arteriolar constriction in the trained muscles, increasing maximal muscle blood flow with no change in the mean arterial blood pressure.

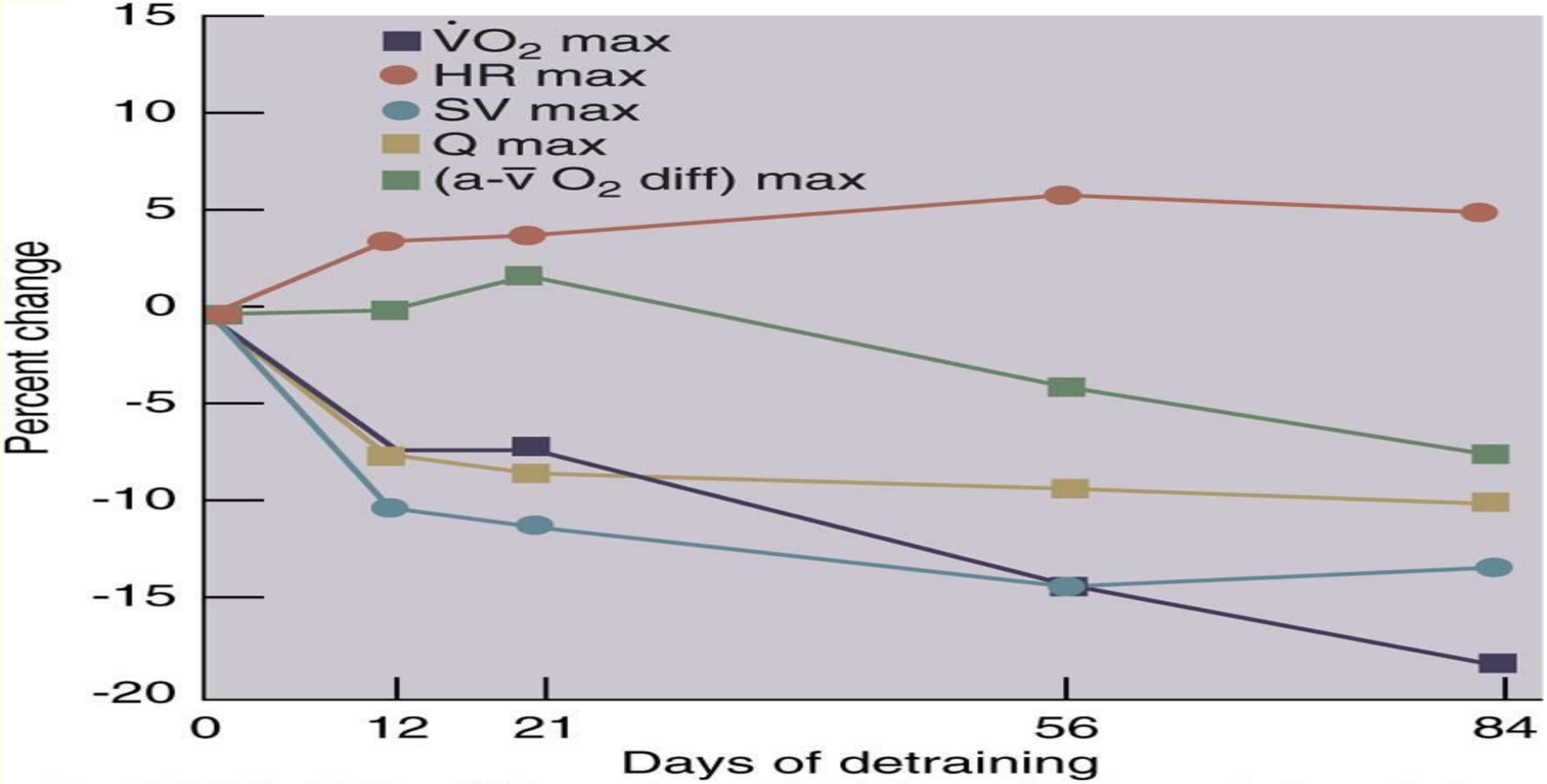
# In Summary

- In young sedentary subjects, 50% of the increase in $VO_2$ max is due to an increase in the systemic a-$vO_2$ difference. The increased a-$vO_2$ difference is due to an increase in the capillary density of the trained muscles that is needed to accept the increase in maximal muscle blood flow. The greater capillary density allows for a sufficiently slow red blood cell transit time through the muscle, providing enough time for oxygen diffusion, which is facilitated by the increase in the number of mitochondria.

# Detraining and $VO_2$ Max

- Decrease in $VO_2$ max with cessation of training
  - ↓ SV max
    - Rapid loss of plasma volume
  - ↓ Maximal a-v$O_2$ difference
    - ↓ Mitochondria
    - ↓ Oxidative capacity of muscle
      - ↓ Type IIa fibers and ↑ type IIx fibers
  - Initial decrease (12 days) due to ↓ SV max
  - Later decrease due to ↓ a-v$O_2$ max

# Detraining and Changes in $VO_2$ Max and Cardiovascular Variables



# In Summary

- The decrease in $VO_2$ max with cessation of training is due to both a decrease in maximal stroke volume and a decrease in oxygen extraction, the reverse of what happens with training.



# Effects of Endurance Training on Performance

- Maintenance of homeostasis
  - More rapid transition from rest to steady-state
  - Reduced reliance on glycogen stores
  - Cardiovascular and thermoregulatory adaptations
- Neural and hormonal adaptations
  - Initial changes in performance
- Structural and biochemical changes in muscle
  - ↑ Mitochondrial number
  - ↑ Capillary density



# Time Course of Training/Detraining

## Mitochondrial Changes

- Training
  - Mitochondria double with five weeks of training
- Detraining
  - About 50% of the increase in mitochondrial content was lost after one week of detraining
  - All of the adaptations were lost after five weeks of detraining
  - It took four weeks of retraining to regain the adaptations lost in the first week of detraining

# Time Course of Training/Detraining

## Mitochondrial Changes

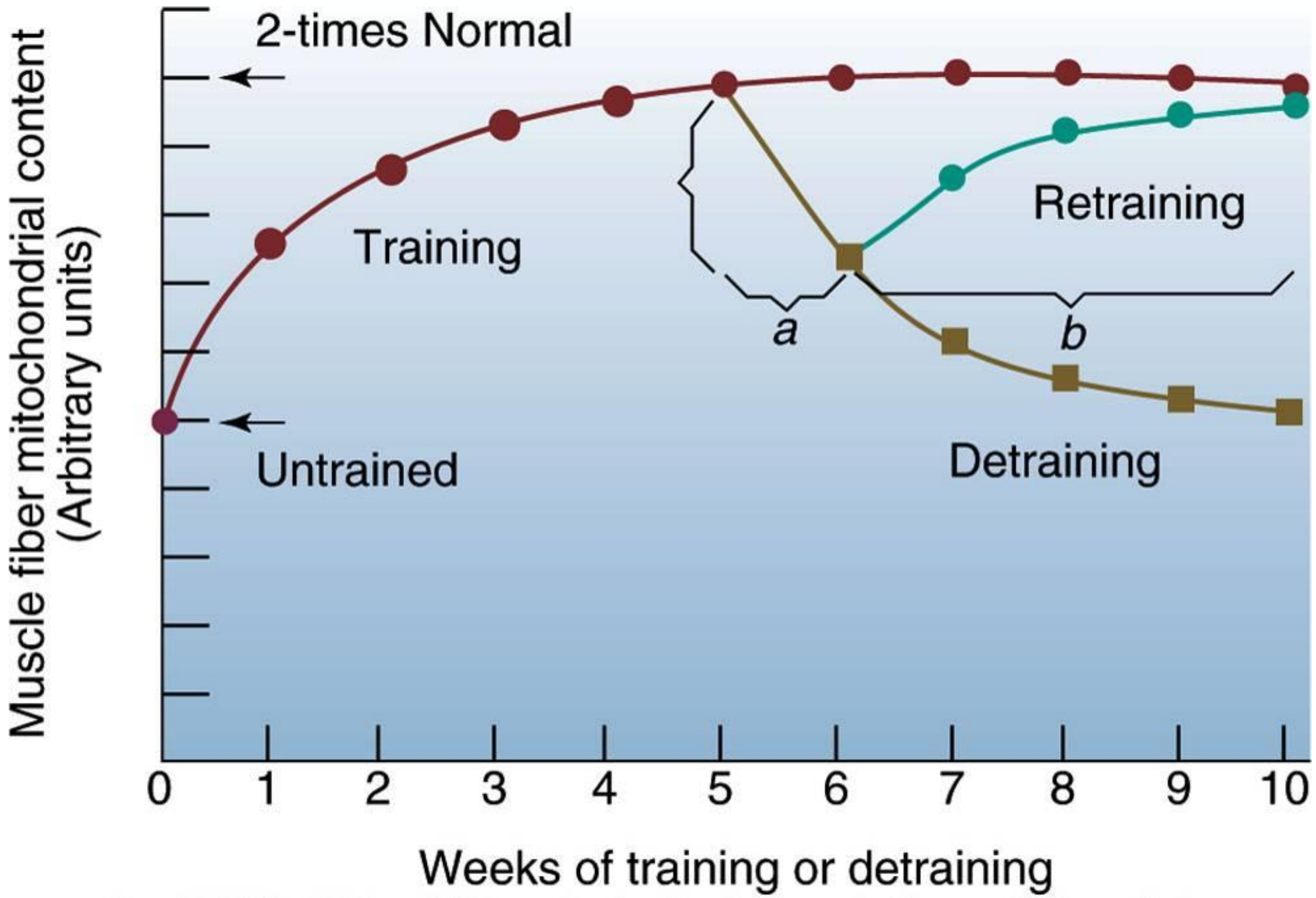



**Figure 13.5**

# Role of Exercise Intensity and Duration on Mitochondrial Adaptations

- Citrate synthase (CS)
  - Marker of mitochondrial oxidative capacity
- Effect of exercise intensity
  - 55%, 65%, or 75% $VO_2$ max
  - Increased CS in oxidative (IIa) fibers with all training intensities
- Effect of exercise duration
  - 30, 60, or 90 minutes
  - No difference between durations on CS activity in IIa fibers
  - Increase in CS activity in IIx fibers with higher-intensity, longer-duration training

# Physiological Effects of Strength Training

- Muscular strength
  - Maximal force a muscle or muscle group can generate
    - 1 repetition maximum (1-RM)
- Muscular endurance
  - Ability to make repeated contractions against a submaximal load
- Strength training
  - Percent gain inversely proportional to initial strength
    - Genetic limitation to gains in strength
  - High-resistance (2–10 RM) training
    - Gains in strength
  - Low-resistance training
    - Gains in endurance



# Aging, Strength, and Training

- Decline in strength after age 50
  - Loss of muscle mass (sarcopenia)
    - Loss of both type I and II fibers
    - Atrophy of type II fibers
    - Loss of intramuscular fat and connective tissue
  - Loss of motor units
  - Reorganization of motor units
- Progressive resistance training
  - Causes muscle hypertrophy and strength gains
  - Important for activities of daily living, balance, and reduced risk of falls



# Physiological Mechanisms Causing Increased Strength

- Strength training results in increased muscle size and strength
- Initial 8–20 weeks
  - Neural adaptations
- Long-term training (20+ weeks)
  - Muscle hypertrophy
  - High-intensity training can result in hypertrophy with 10 sessions

# Neural and Muscular Adaptations to Resistance Training

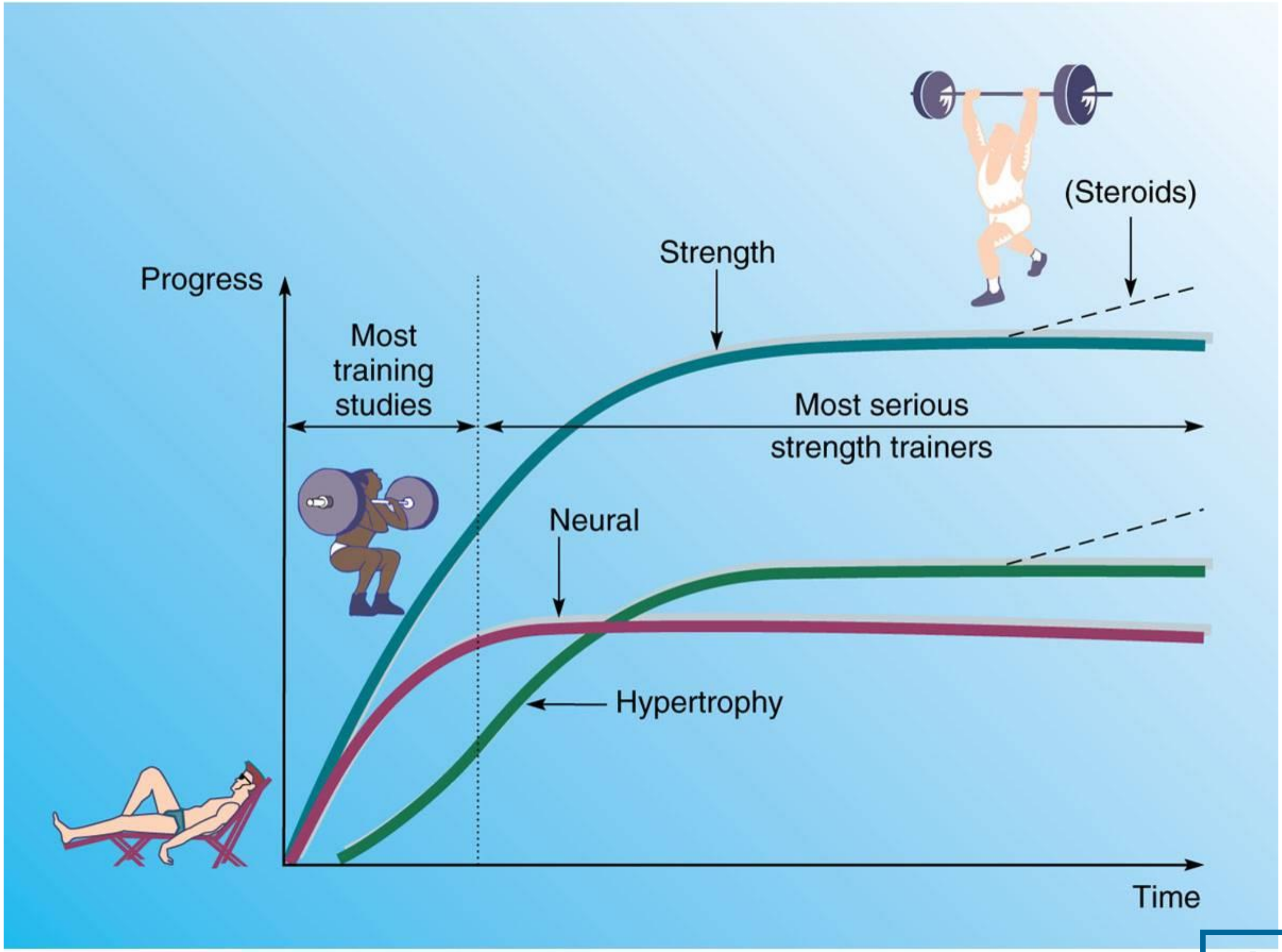



Figure 13.17



# Neural Factors

- Early gains in strength
  - Initial 8–20 weeks
- Adaptations
  - Improved ability to recruit motor units
  - Learning
  - Coordination

# Muscular Enlargement

- Hypertrophy
  - Enlargement of both type I and II fibers
    - Low-intensity (high RM), high-volume training results in smaller type II fibers
    - Heavy resistance (low RM) results in larger type II fibers
  - No increase in capillary density
- Hyperplasia
  - Increase in muscle fiber number
  - Mainly seen in long-term strength training
    - Not as much evidence as muscle hypertrophy



# Periodization of Strength Training

- Traditional training programs
  - Variations in intensity (RM), sets, and repetitions
- Periodization
  - Also includes variation of:
    - Rest periods, type of exercise, number of training sessions, and training volume
  - Develop workouts to achieve optimal gains in:
    - Strength, power, motor performance, and/or hypertrophy

- Linear and undulating programs
  - Variations in volume/intensity over time
- More effective than non-periodized training for improving strength and endurance



# Concurrent Strength and Endurance Training

- Potential for interference of adaptations
  - Endurance training increases mitochondial protein
  - Strength training increases contractile protein
  - Depends on intensity, volume, and  frequency of training
- Studies show conflicting results
  - Depends on intensity, volume, and  frequency of training

# In Summary

- Increases 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 increase in the size of the muscle.
- There is evidence both for and against the proposition that the physiological effects of strength training interfere with the physiological effects of endurance training.

# Thank You !

100%