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**SPORTS MEDICINE AND MEDICAL  
PHYSICAL TRAINING:  
BASIC CONCEPTS OF EXERCISE PHYSIOLOGY  
AND EXERCISE PRESCRIPTION**

Study book

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## **BASIC CONCEPTS OF EXERCISE PHYSIOLOGY**

Exercise represents one of the highest levels of extreme stresses to which the body can be exposed. For example, in a person who has an extremely high fever approaching the level of lethality, the body metabolism increases to approximately 100% above normal; by comparison, the metabolism of the body during a marathon race increases to 2000% above normal. This article describes the basic physiology of exercise. The focus of this article is mainly at a subspecialty level; however, more detailed descriptions of various basic mechanisms are also provided for the casual reader

### **GENDER DIFFERENCES**

In general, the exercise-related measurements established for women follow the same general principles as those established for men, except for the quantitative differences caused by differences in body size, body composition, and levels of testosterone.

In women, the values of muscle strength, pulmonary ventilation, and cardiac output (all variables related with muscle mass) generally are 60-75% of the values recorded in men. When measured in terms of strength per square centimeter, the female muscle can achieve the same force of contraction as that of a male.

## **MUSCULOSKELETAL SYSTEM AND EXERCISE**

### **Functions of muscle tissue**

Muscle tissue has 4 characteristics that assume roles in homeostasis, as follows:

- Excitability - Property of receiving and responding to stimuli such as the following:

- Neurotransmitters: Acetylcholine (ACh) stimulates skeletal muscle to contract.
- Electrical stimuli: Applying electrical stimuli between cardiac and smooth muscle cells causes the muscles to contract. Applying a shock to skeletal muscle causes contraction.
- Hormonal stimuli: Oxytocin stimulates smooth muscle in the uterus to contract during labor.
- Contractility - Ability to shorten
- Extensibility - Ability to stretch without damage
- Elasticity - Ability to return to original shape after extension

Through contraction, muscle provides motion of the body (skeletal muscle), motion of blood (cardiac muscle), and motion of hollow organs such as the uterus, esophagus, stomach, intestines, and bladder (smooth muscle).

Muscle tissue also helps maintain posture and produce heat. A large amount of body heat is produced by metabolism and by muscle contraction. Muscle contraction during shivering warms the body.

### **Histology of skeletal muscle tissue**

Skeletal muscle consists of fibers (cells). These cells are up to 100  $\mu\text{m}$  in diameter and often are as long as the muscle. Each contains sarcoplasm (cytoplasm) and multiple peripheral nuclei per fiber. Skeletal muscle is actually formed by the fusion of hundreds of embryonic cells. Other cell structures include the following:

- Each fiber is covered by a sarcolemma (plasma membrane).
- The sarcoplasmic reticulum (smooth endoplasmic reticulum) stores calcium, which is released into the sarcoplasm during muscle contraction.
- Transverse tubules (T tubules), which are extensions of the sarcolemma that penetrate cells, transmit electrical impulses from the sarcolemma inward, so electrical impulses penetrate deeply into the cell. Besides conducting electricity along their walls, T tubules contain extracellular fluid rich in glucose and oxygen.

- The sarcoplasm of fiber is rich in glycogen (glucose polymer) granules and myoglobin (oxygen-storing protein). It also is rich in mitochondria.

Each fiber contains hundreds to thousands of rodlike myofibrils, which are bundles of thin and thick protein chains termed myofilaments. From a cross-sectional view of a myofibril, each thick filament is surrounded by a hexagonal array of 6 thin filaments. Each thin filament is surrounded by a triangular array of thick filaments.

- Thin myofilaments are composed of 3 proteins: actin, tropomyosin, and troponin.

- Thick myofilaments consist of bundles of approximately 200 myosin molecules. Myosin molecules look like double-headed golf clubs (both heads at the same end). The heads of the golf clubs are called myosin heads; they also are called cross-bridges because they link thick and thin filaments during contraction. They contain actin and ATP binding sites. Myosin heads project out from the thick filaments, allowing them to bind to the thin filaments during contraction.

- Actin is a long chain of multiple globular proteins, similar in shape to kidney beans. Each globular subunit contains a myosin-binding site.

- Tropomyosin is a long strand of protein that covers the myosin-binding sites on actin when the muscle is relaxed.

- Troponin is a polypeptide complex that binds to tropomyosin, helping to position it over the myosin-binding sites on actin. During muscle contraction, calcium binds troponin, which causes tropomyosin to roll off of the myosin binding sites on actin.

### **Muscle contraction (overview of the sliding filament mechanism)**

A muscle action potential travels over sarcolemma and enters the T tubules, causing the sarcoplasmic reticulum to release calcium into the sarcoplasm. This triggers the contractile process.

Myosin cross-bridges pull on the actin myofilaments, causing the thin myofilaments of a sarcomere to slide toward the centers of the H zones.

## **Other components of skeletal muscle**

### **Connective-tissue components**

Deep fascia is a broad band of dense irregular connective tissue beneath and around muscle and organs. Deep fascia is different from superficial fascia, which is loose areolar connective tissue.

Other connective-tissue components (all are extensions of deep fascia) include epimysium, which covers the entire muscle; perimysium, which penetrates into muscle and surrounds bundles of fibers called fascicles; and endomysium, which is delicate, barely visible, loose areolar tissue covering individual fibers (ie, individual cells).

Tendons and aponeuroses are tough extensions of epimysium, perimysium, and endomysium. Tendons and aponeuroses are made of dense regular connective tissue and attach the muscle to bone or other muscle. Aponeuroses are broad, flat tendons. Tendon sheaths contain synovial fluid and enclose certain tendons. Tendon sheaths allow tendons to slide back and forth next to each other with lower friction. Tenosynovitis is inflammation of the tendon sheaths and tendons, especially those of the wrists, shoulders, and elbows. Tendons are not contractile and not very stretchy; furthermore, they are not very vascular and they heal poorly.

### **Nerve and blood supply**

Nerves convey impulses for muscular contraction. Nerves are bundles of nerve cell processes. Each nerve cell process (ie, axon) divides at its tip into a few to 10,000 branches called telodendria. At the end of each of these branches is an axon terminal that is rich in neurotransmitters.

Blood provides nutrients and oxygen for contraction. An artery and a vein usually accompany a nerve that penetrates skeletal muscle. Arteries in muscles dilate during active muscular activity, thus increasing the supply of oxygen and glucose.

## **Motor units**

A motor nerve is a bundle of axons that conducts nerve impulses away from the brain or spinal cord toward muscles. Each axon transmits an action potential (ie, nerve impulse), which is a burst of electricity. The nerve impulse travels along the axons at a steady rate, like fire travels along a fuse; however, nerve impulses travel extremely fast. Each axon has 4-2000 or more branches (ie, telodendria), with an average of 150 telodendria. Each separate branch supplies a separate muscle cell. Thus, if an axon has 10 branches, it supplies 10 muscle fibers. Small motor units are for fine control of muscles; large motor units are for muscles that do not require such fine control.

## **Neuromuscular junction**

The neuromuscular junction is made of an axon terminal and the portion of the muscle fiber sarcolemma it nearly touches (called the motor endplate). The neurotransmitter released at the neuromuscular junction in skeletal muscle is ACh. The motor endplate is rich in thousands of ACh receptors; the receptors are integral proteins containing binding sites for ACh and sodium channels.

## **Physiology of contraction**

1. Nerve impulse (action potential) reaches the axon terminal, which triggers calcium influx into the axon terminal.
2. Calcium influx causes synaptic vesicles to release ACh via exocytosis.
3. ACh diffuses across synaptic cleft.
4. ACh binds to the ACh receptor on the sarcolemma. Succinylcholine, a drug used to induce paralysis during surgery, binds to ACh receptors more tightly than ACh. Succinylcholine initially causes some depolarization, but then it binds to the receptor, preventing ACh from binding. Therefore, it blocks the muscle's stimulation by ACh, causing paralysis. Another drug that acts in a similar fashion is curare. These drugs do not cause pain relief or unconsciousness; thus, they are combined with other drugs during surgery.

5. When ACh binds the receptor, it opens chemically regulated ion channels, which are sodium channels through the receptor molecule. Sodium, which is in high concentration outside cells and in low concentration inside cells, rushes into the cell through the channels.

6. The cell, whose resting membrane potential along the inside of the membrane is negative when compared with the outside of the membrane, becomes positively charged along the inside of the membrane when sodium (a positive ion) rushes in. This change from a negative charge to a positive charge along the inner membrane is termed depolarization.

7. The depolarization of one region of the sarcolemma (the motor endplate) initiates an action potential, which is a propagating wave of depolarization that travels (propagates) along the sarcolemma. Regions of membrane that become depolarized rapidly restore their proper ionic concentrations along their inner and outer surfaces in a process termed repolarization. (This process of depolarization, propagation, and repolarization is similar to dominoes that topple each other but also spring back into the upright position shortly afterward.)

8. The action potential also propagates along the membrane lining the T tubules entering the cell.

9. This action potential traveling along the T tubules causes the sarcoplasmic reticulum to release calcium into sarcoplasm.

10. Calcium binds with troponin, causing it to pull on tropomyosin to change its orientation, exposing myosin-binding sites on actin.

11. An ATPase, which also functions as a myosin cross-bridging protein, splits ATP into adenosine diphosphate (ADP) + phosphate (P) in the previous contraction cycle. This energizes the myosin head. The energized myosin head, or cross-bridge, combines with myosin-binding sites on actin.

12. Power stroke occurs. The attachment of the energized cross-bridge triggers a pivoting motion (ie, power stroke) of the myosin head. During the power stroke, ADP and P are released from the myosin cross-bridge. The power stroke



causes thin actin myofilaments to slide past thick myosin myofilaments toward the center of the A bands.

13. ATP attaches to the myosin head again, allowing it to detach from actin. (In rigor mortis, an ATP deficiency occurs. Cross-bridges remain, and the muscles are rigid.)

14. ATP is broken down to ADP and P, which cocks the myosin head again, preparing it to perform another power stroke if needed.

15. Repeated detachment and reattachment of the cross-bridges results in shortening without much increase in tension during the shortening phase (isotonic contraction) or results in increased tension without shortening (isometric contraction).

16. Release of the enzyme acetylcholinesterase in the neuromuscular junction destroys ACh and stops the generation of a muscle action potential. Calcium is taken back up (resequestered) in the sarcoplasmic reticulum, and myosin cross-bridges separate. ATP is required to separate myosin-actin cross-bridges. The muscle fiber resumes its resting state.

### **Muscle metabolic systems during exercise**

The chemical energy that fuels muscular activities is ATP. For the first 5 or 6 seconds of muscle power, muscular activity can depend on the ATP that is already present in the muscle cells. Beyond this time, new amounts of ATP must be formed to enable the activation of muscular contractions needed to support longer and more vigorous physical activities. For activities that require a quick burst of energy that cannot be supplied by the ATP present in the muscle cells, the next 10-15 seconds of muscle power can be provided through the body's use of the phosphagen system, which uses a substance called creatine phosphate to recycle ADP into ATP. For longer and more intense periods of physical activity, the body must rely on systems that break down the sugars (glucose) to produce ATP.

The complete breakdown of glucose occurs in 2 ways: through anaerobic respiration (does not use oxygen) and through aerobic respiration (occurs in the presence of oxygen). The anaerobic use of glucose to form ATP occurs as the body

increases its muscle use beyond the capability of the phosphagen system to supply energy. In particular, the glycogen lactic acid system, through its anaerobic breakdown of glucose, provides approximately 30-40 seconds more of maximal muscle activity. For this system, each glucose molecule is split into 2 pyruvic acid molecules, and energy is released to form several ATP molecules, providing the extra energy. Then, the pyruvic acid partially breaks down further to produce lactic acid. If the lactic acid is allowed to accumulate in the muscle, one experiences muscle fatigue. At this point, the aerobic system must activate.

The aerobic system in the body is used for sports that require an extensive and enduring expenditure of energy, such as a marathon race. Endurance sports absolutely require aerobic energy. A large amount of ATP must be provided to muscles to sustain the muscle power needed to perform such events without an excessive production of lactic acid. This can only be accomplished when oxygen in the body is used to break down the pyruvic acid (that was produced anaerobically) into carbon dioxide, water, and energy by way of a very complex series of reactions known as the citric acid cycle. This cycle supports muscle usage for as long as the nutrients in the body last. The breakdown of pyruvic acid requires oxygen and slows or eliminates the accumulation of lactic acid. In summary, the 3 different muscle metabolic systems that supply the energy required for various activities are as follows:

- Phosphagen system (for 10- to 15-second bursts of energy)
- Glycogen lactic acid system (for another 30-40 seconds of energy)
- Aerobic system (provides a great deal of energy that is only limited by the body's ability to supply oxygen and other important nutrients)

Many sports require the use of a combination of these metabolic systems. By considering the vigor of a sports activity and its duration, one can estimate very closely which of the energy systems are used for each activity.

## **Postexercise recovery**

### **Oxygen debt**

During muscular exercise, blood vessels in muscles dilate and blood flow is increased in order to increase the available oxygen supply. Up to a point, the available oxygen is sufficient to meet the energy needs of the body. However, when muscular exertion is very great, oxygen cannot be supplied to muscle fibers fast enough, and the aerobic breakdown of pyruvic acid cannot produce all the ATP required for further muscle contraction.

During such periods, additional ATP is generated by anaerobic glycolysis. In the process, most of the pyruvic acid produced is converted to lactic acid. Although approximately 80% of the lactic acid diffuses from the skeletal muscles and is transported to the liver for conversion back to glucose or glycogen, some lactic acid accumulates in muscle tissue, making muscle contraction painful and causing fatigue. Ultimately, once adequate oxygen is available, lactic acid must be catabolized completely into carbon dioxide and water.

After exercise has stopped, extra oxygen is required to metabolize lactic acid; to replenish ATP, phosphocreatine, and glycogen; and to replace (“pay back”) any oxygen that has been borrowed from hemoglobin, myoglobin (an iron-containing substance similar to hemoglobin that is found in muscle fibers), air in the lungs, and body fluids. The additional oxygen that must be taken into the body after vigorous exercise to restore all systems to their normal states is called oxygen debt. The debt is paid back by labored breathing that continues after exercise has stopped. Thus, the accumulation of lactic acid causes hard breathing and sufficient discomfort to stop muscle activity until homeostasis is restored.

### **Recovery of muscle glycogen postexercise**

Eventually, muscle glycogen also must be restored. Restoration of muscle glycogen is accomplished through diet and may take several days, depending on the intensity of exercise. The maximum rate of oxygen consumption during the aerobic catabolism of pyruvic acid is called maximal oxygen uptake. Maximal oxygen uptake

is determined by sex (higher in males), age (highest at approximately age 20 y), and size (increases with body size). Highly trained athletes can have maximal oxygen uptakes that are twice that of average people, probably owing to a combination of genetics and training. As a result, highly trained athletes are capable of greater muscular activity without increasing their lactic acid production and have lower oxygen debts, which is why they do not become short of breath as readily as untrained individuals.

## **FUEL USAGE**

### **Fuel usage (light exercise)**

The best examples of light exercise are walking and light jogging. The muscles that are recruited during this type of exercise are those that contain a large amount of type I muscle cells, and, because these cells have a good blood supply, it is easy for fuels and oxygen to travel to the muscle. ATP consumption makes ADP available for new ATP synthesis.

The presence of ADP (and the resulting synthesis of ATP) stimulates the movement of hydrogen ( $H^+$ ) into the mitochondria; this, in turn, reduces the proton gradient and thus stimulates electron transport. The hydrogen on the reduced form of nicotinamide adenine dinucleotide (NADH) is used up, nicotinamide adenine dinucleotide (NAD) becomes available, and fatty acids and glucose are oxidized. Incidentally, the calcium released during contraction stimulates the enzymes in the Krebs cycle and stimulates the movement of the glucose transporter 4 (GLUT-4) from inside of the muscle cell to the cell membrane. Both these exercise-induced responses augment the elevation in fuel oxidation caused by the increase in ATP consumption.

### **Fuel usage (moderate exercise)**

An increase in the pace of running simply results in an increased rate of fuel consumption, an increased fatty acid release, and, therefore, an increase in the rate of

muscle fatty acid oxidation. However, if the intensity of the exercise increases even further, a stage is reached in which the rate of fatty acid oxidation becomes limited.

The reasons why the rate of fatty acid oxidation reaches a maximum are not clear, but it is possible that the enzymes in the beta-oxidation pathway are saturated (ie, they reach a stage in which their maximal velocity [ $V_{\max}$ ] is less than the rate of acetyl-coenzyme A [acetyl-CoA] consumption in the Krebs cycle). Alternatively, it may be that the availability of carnitine (the chemical required to transport the fatty acids into the mitochondria) becomes limited.

Whatever the reason, the consequence is that as the pace rises, the demand for acetyl-CoA cannot be met by fatty acid oxidation alone. The accumulation of acetyl-CoA that was so effective at inhibiting the oxidation of glucose is no longer present, so pyruvate dehydrogenase starts working again and pyruvate is converted into acetyl-CoA. In other words, more of the glucose that enters the muscle cell is oxidized fully to carbon dioxide. Therefore, the energy used during moderate exercise is derived from a mixture of fatty acid and glucose oxidation.

### **Fuel usage (strenuous exercise)**

As the intensity of the exercise increases even further (ie, running at the pace of middle-distance races), the rate at which the muscles can extract glucose from the blood becomes limited. In other words, the rate of glucose transport reaches  $V_{\max}$ , either because the blood cannot supply the glucose fast enough or the number of GLUT-4s becomes limited. ATP generation cannot be serviced completely by exogenous fuels, and ATP levels decrease. Not only does this stimulate phosphofructokinase, it also stimulates glycogen phosphorylase. This means that glycogen stored within the muscle cells is broken down to provide glucose. Therefore, the fuel mix during strenuous exercise is composed of contributions from blood-borne glucose and fatty acids and from endogenously stored glycogen.

## **Fuel usage in individuals who are unfit**

Being fit (biochemically speaking) means that the individual has a well-developed cardiovascular system that can efficiently supply nutrients and oxygen to the muscles. Fit people have muscle cells that are well perfused with capillaries (ie, they have a good muscle blood supply). Their muscle cells also have a large number of mitochondria, and those mitochondria have a high activity of Krebs cycle enzymes, electron transport carriers, and oxidation enzymes.

Individuals who are unfit must endure the consequences of a poorer blood supply, fewer mitochondria, less electron transport units, a lower activity of the Krebs cycle, and poorer activity of beta-oxidation enzymes. To generate ATP in the mitochondria, a steady supply of fuel and oxygen and decent activity of the oxidizing enzymes and carriers are needed. If any of these components are lacking, the rate at which ATP can be produced by mitochondria is compromised. Under these circumstances, the production of ATP by aerobic means is not sufficient to provide the muscles with sufficient ATP to sustain contractions. The result is anaerobic ATP generation using glycolysis. Increasing the flux through glycolysis but not increasing the oxidative consumption of the resulting pyruvate increases the production of lactate.

## **PULMONARY PHYSIOLOGY DURING EXERCISE**

The purpose of respiration is to provide oxygen to the tissues and to remove carbon dioxide from the tissues. To accomplish this, 4 major events must be regulated, as follows:

- Pulmonary ventilation
- Diffusion of oxygen and carbon dioxide between the alveoli and the blood
- Transport of oxygen and carbon dioxide in the blood and body fluids and to and from the cells

- Regulation of ventilation and other aspects of respiration: Exercise causes these factors to change, but the body is designed to maintain homeostasis.

When one goes from a state of rest to a state of maximal intensity of exercise, oxygen consumption, carbon dioxide formation, and total pulmonary and alveolar ventilation increase by approximately 20-fold. A linear relationship exists between oxygen consumption and ventilation. At maximal exercise, pulmonary ventilation is 100-110 L/min, whereas maximal breathing capacity is 150-170 L/min. Thus, the maximal breathing capacity is approximately 50% greater than the actual pulmonary ventilation during maximal exercise. This extra ventilation provides an element of safety that can be called on if the situation demands it (eg, at high altitudes, under hot conditions, abnormality in the respiratory system). Therefore, the respiratory system itself is not usually the most limiting factor in the delivery of oxygen to the muscles during maximal muscle aerobic metabolism.

$\text{VO}_{2 \text{ max}}$  is the rate of oxygen consumption under maximal aerobic metabolism. This rate in short-term studies is found to increase only 10% with the effect of training. However, that of a person who runs in marathons is 45% greater than that of an untrained person. This is believed to be partly genetically determined (eg, stronger respiratory muscles, larger chest size in relation to body size) and partly due to long-term training.

Oxygen diffusing capacity is a measure of the rate at which oxygen can diffuse from the alveoli into the blood. An increase in diffusing capacity is observed in a state of maximal exercise. This results from the fact that blood flow through many of the pulmonary capillaries is sluggish in the resting state. In exercise, increased blood flow through the lungs causes all of the pulmonary capillaries to be perfused at their maximal level, providing a greater surface area through which oxygen can diffuse into the pulmonary capillary blood. Athletes who require greater amounts of oxygen per minute have been found to have higher diffusing capacities, but the exact reason why is not yet known. Although one would expect the oxygen pressure of arterial blood to decrease during strenuous exercise and carbon dioxide pressure of venous

blood to increase far above normal, this is not the case. Both of these values remain close to normal.

Stimulatory impulses from higher centers of the brain and from joint and muscle proprioceptive stimulatory reflexes account for the nervous stimulation of the respiratory and vasomotor center that provides almost exactly the proper increase in pulmonary ventilation to keep the blood respiratory gases almost normal. If nervous signals are too strong or weak, chemical factors bring about the final adjustment in respiration required to maintain homeostasis.

## **CARDIOVASCULAR SYSTEM AND EXERCISE**

Regular exercise makes the cardiovascular system more efficient at pumping blood and delivering oxygen to the exercise muscles. Releases of adrenaline and lactic acid into the blood result in an increase of the heart rate (HR).

Basic definitions of terms are as follows:

- $VO_2$  equals cardiac output times oxygen uptake necessary to supply oxygen to muscles.
- The Fick equation is the basis for determination of  $VO_2$ .

Exercises increase some of the different components of the cardiovascular system, such as stroke volume (SV), cardiac output, systolic blood pressure (BP), and mean arterial pressure. A greater percentage of the cardiac output goes to the exercising muscles. At rest, muscles receive approximately 20% of the total blood flow, but, during exercise, the blood flow to muscles increases to 80-85%.

To meet the metabolic demands of skeletal muscle during exercise, 2 major adjustments to blood flow must occur. First, cardiac output from the heart must increase. Second, blood flow from inactive organs and tissues must be redistributed to active skeletal muscle.

Generally, the longer the duration of exercise, the greater the role the cardiovascular system plays in metabolism and performance during the exercise bout.



An example would be the 100-meter sprint (little or no cardiovascular involvement) versus a marathon (maximal cardiovascular involvement).

### **General functions of the cardiovascular system**

The cardiovascular system helps transport oxygen and nutrients to tissues, transport carbon dioxide and other metabolites to the lungs and kidneys, and distribute hormones throughout the body. The cardiovascular system also assists with thermoregulation.

### **Cardiac cycle**

The pumping of blood by the heart requires the following 2 mechanisms to be efficient:

- Alternate periods of relaxation and contraction of the atria and ventricles
- Coordinated opening and closing of the heart valves for unidirectional

flow of blood

The cardiac cycle is divided into 2 phases: ventricular diastole and ventricular systole.

- Ventricular diastole
  - This phase begins with the opening of the atrioventricular (AV) valves.

The mitral valve (located between the left atrium and left ventricle) opens when the left ventricular pressure falls below the left atrial pressure, and the blood from left atrium enters the left ventricle.

- Later, as the blood continues to flow into the left ventricle, the pressure in both chambers tends to equalize.

- At the end of the diastole, left atrial contractions cause an increase in left atrial pressure, thus again creating a pressure gradient between the left atrium and ventricle and forcing blood into the left ventricle.

- Ventricular systole
  - Ventricular systole begins with the contraction of the left ventricle, which is caused by the spread of an action potential over the left ventricle. The

contraction of the left ventricle causes an increase in the left ventricular pressure. When this pressure is higher than the left atrial pressure, the mitral valve is closed abruptly.

- The left ventricular pressure continues to rise after the mitral valve is closed. When the left ventricular pressure rises above the pressure in the aorta, the aortic valve opens. This period between the closure of the mitral valve and the opening of the aortic valve is called isovolumetric contraction phase.

- The blood ejects out of the left ventricle and into the aorta once the aortic valve is opened. As the left ventricular contraction is continued, 2 processes lead to a fall in the left ventricular pressure. These include a decrease in the strength of the ventricular contraction and a decrease in the volume of blood in the ventricle.

- When the left ventricular pressure falls below the aortic pressure, the aortic valve is closed. After the closure of the aortic valve, the left ventricular pressure falls rapidly as the left ventricle relaxes. When this pressure falls below the left atrial pressure, the mitral valve opens and allows blood to enter left ventricle. The period between the closure of the aortic valve closure and the opening of the mitral valve is called isovolumetric relaxation time.

- Right-sided heart chambers undergo the same phases simultaneously.

### **Pressure changes during the cardiac cycle**

Most of the work of the heart is completed when ventricular pressure exists. The greater the ventricular pressure, the greater the workload of the heart. Increases in BP dramatically increase the workload of the heart, and this is why hypertension is so harmful to the heart.

Arterial BP is the pressure that is exerted against the walls of the vascular system. BP is determined by cardiac output and peripheral resistance. Arterial pressure can be estimated using a sphygmomanometer and a stethoscope. The reference range for males is 120/80 mm Hg. The reference range for females is 110/70 mm Hg.

The difference between systolic and diastolic pressure is called the pulse pressure. The average pressure during a cardiac cycle is called the mean arterial pressure (MAP). MAP determines the rate of blood flow through the systemic circulation.

- During rest,  $MAP = \text{diastolic BP} + (0.33 \times \text{pulse pressure})$ . For example,  $MAP = 80 + (0.33 \times [120-80])$ ,  $MAP = 93 \text{ mm Hg}$ .
- During exercise,  $MAP = \text{diastolic BP} + (0.50 \times \text{pulse pressure})$ . For example,  $MAP = 80 + (0.50 \times [160-80])$ ,  $MAP = 120 \text{ mm Hg}$ .

### **Coordinated control of the heart**

The heart has the ability to generate its own electrical activity, which is known as intrinsic rhythm. In the healthy heart, contraction is initiated in the sinoatrial (SA) node, which often is called the heart's pacemaker. If the SA node cannot set the rate, then other tissues in the heart are able to generate an electrical potential and establish the HR.

### **Control of cardiac output (HR)**

The parasympathetic nervous system and the sympathetic nervous system affect a person's HR.

- Parasympathetic nervous system: The vagus nerve originates in the medulla and innervates the SA and AV nodes. The nerve releases ACh as the neurotransmitter. The response is a decrease in SA node and AV node activity, which causes a decrease in HR.
- Sympathetic nervous system: The nerves arise from the spinal cord. The nerves innervate the SA node and ventricular muscle mass. The nerves release norepinephrine as the neurotransmitter. The response is an increase in HR and a force of contraction of the ventricles.

## **Control of sympathetic and parasympathetic activity**

At rest, sympathetic and parasympathetic nervous stimulation are in balance. During exercise, parasympathetic stimulation decreases and sympathetic stimulation increases. Several factors can alter sympathetic nervous system input.

Baroreceptors are groups of neurons located in the carotid arteries, the arch of aorta, and the right atrium. These neurons sense changes in pressure in the vascular system. An increase in BP results in an increase in parasympathetic activity except during exercise, when the sympathetic activity overrides the parasympathetic activity.

Chemoreceptors are groups of neurons located in the arch of the aorta and the carotid arteries. These neurons sense changes in oxygen concentration. When oxygen concentration in the blood is decreased, parasympathetic activity decreases and sympathetic activity increases.

Temperature receptors are neurons located throughout the body. These neurons are sensitive to changes in body temperature. As temperature increases, sympathetic activity increases to cool the body and to reduce internal core temperature.

## **Control of cardiac output (SV)**

SV is controlled by end-diastolic volume, average aortic BP, and the strength of ventricular contraction.

- End-diastolic volume: This is often referred to as the preload. If the end-diastolic volume increases, the SV increases. With an increased end-diastolic volume, a slight stretching of the cardiac muscle fibers occurs, which increases the force of contraction
- Average aortic BP: This is often referred to as the after load. The BP in the aorta represents a barrier to the blood being ejected from the heart. The SV is inversely proportional to the aortic BP. During exercise, the after load is reduced, which allows for an increase in SV.
- Strength of ventricular contraction: Epinephrine and norepinephrine can increase the contractility of the heart by increasing the calcium concentration within the cardiac muscle fiber. Epinephrine and norepinephrine allow for greater calcium

entry through the calcium channels in cardiac muscle fiber membranes. This allows for greater myosin and actin interaction and an increase in force production.

### **Control of cardiac output (venous return)**

Venoconstriction occurs as a response to sympathetic nervous system stimulation. Sympathetic stimulation constricts the veins that drain skeletal muscle. This causes greater blood to flow back to the heart.

The muscle pump is the rhythmic contraction and relaxation of skeletal muscle that compresses the veins and thus drains the skeletal muscle. This causes greater blood flow back to the heart. The muscle pump is very important during both resting and exercise conditions.

During exercise, the respiratory pump helps increase venous return. The pressure within the chest decreases and abdominal pressure increases with inhalation, thus facilitating blood flow back to the heart. Because of the increased respiratory rate and depth of breathing during exercise, this is an effective way to increase venous return.

### **Hemodynamics**

The circulatory system is a closed-loop system, and flow through the circulatory system is the result of pressure differences between the 2 ends of the system, the left ventricle (90 mm Hg) and the right atrium (approximately 0 mm Hg).

Systemic blood flow affects hemodynamics. The control of blood flow during exercise is extremely important to ensure that blood and oxygen are transported to the tissues that need them most. Blood flow to tissues is dependent of the relationship between BP and the resistance provided by the blood vessels.

Blood flow at rest is equal to the change in pressure divided by the resistance of the vessels (ie,  $BF = P/R$ , where BF is blood flow, P is pressure, and R is resistance). Blood flow during exercise is regulated by changing BP and altering the peripheral resistance of the vessels.

The pressure change at rest in the cardiovascular system is 93 mm Hg, as follows: Mean aortic pressure = 93 mm Hg, mean right atrial pressure = 0 mm Hg, and driving pressure in the system = 93 mm Hg.

During exercise, BP increases so that blood flow through the body increases. Blood flow is also increased during exercise by decreasing the resistance of the vessels in the systemic circulation of active skeletal muscle. Resistance is determined by the following formula: Resistance = (length of tube X viscosity of blood)/radius. Changing the radius of the vessels has the most profound effect on blood flow. Doubling the radius of a blood vessel decreases resistance by a factor of 16. Decreasing the radius of a blood vessel by half increases resistance by a factor of 16. The arterioles have the most control over blood flow in the systemic circulation.

### **Changes in oxygen delivery to muscle during exercise**

BP increases as exercise intensity increases. BP rises from approximately 120 mm Hg to approximately 200 mm Hg. SV increases during exercise until 40% of  $VO_{2max}$  (maximum oxygen uptake level) is reached. SV rises from approximately 80 mL/beat to approximately 120 mL/beat. HR increases with intensity until  $VO_{2max}$  is reached. HR rises from approximately 70 beats per minute to approximately 200 beats per minute. Cardiac output increases with intensity until  $VO_{2max}$  is reached. Cardiac output rises from approximately 5 L/min to approximately 25-30 L/min.

The arterial-venous oxygen difference is the amount of oxygen extracted from the blood as it passes through the capillary bed. This difference rises from approximately 4 mL of oxygen per 100 mL of blood at rest to approximately 18 mL of oxygen per 100 mL of blood during high-intensity aerobic exercise.

### **Redistribution of blood flow during exercise**

At rest, 15-20% of blood goes to skeletal muscle; during exercise, this amount increases to 80-85%. The percentage of blood to the brain decreases, but the absolute amount increases. The same percentage of blood goes to cardiac muscle, but the absolute amount increases. Blood flow to visceral tissues and inactive skeletal muscle

reduces. In addition, the cutaneous blood flow initially decreases, but it later increases during the course of exercise.

The redistribution of the blood is brought about by several mechanisms. During exercise, generalized vasodilatation occurs because of the accumulation of vasodilatory metabolites. This leads to a decrease in the peripheral resistance, which, in turn, elicits a strong increase in the sympathetic activity through the activation of baroreceptors. The increase in sympathetic activity leads to vasoconstriction in the visceral organs, while the vasodilatation predominates in the blood vessels of the muscles and the coronary circulation because of the local vasodilatory metabolites. The cutaneous blood vessels initially respond to the sympathetic activity by vasoconstriction. As the exercise continues, temperature reflexes are activated and cause cutaneous vasodilatation to dissipate the heat produced by the muscle activity, resulting in an increase in the cutaneous blood flow.

### **Regulation of blood flow at the local level**

The local blood flow is controlled by chemical factors, metabolites, paracrines, physical factors such as heat or cold, stretch effects on endothelial membrane, active hyperemia, and reactive hyperemia. The paracrine regulation is mainly regulated by nitric oxide, histamine release, and prostacyclin. Nitric oxide diffuses to smooth muscle and causes vasodilation by reducing  $\text{Ca}^{+2}$  entry into smooth muscle.

### **Regulation of cardiovascular function**

HR and blood flow are controlled by various centers in the brain. These centers receive input from receptors located throughout the body. The centers work to initiate the appropriate response from tissues and organs in the body.

Aerobic exercise requires oxygen to be present for the generation of energy from fuels such as glucose or glycogen. Aerobic exercise results in no buildup of lactic acid as a result of metabolism. This process is more efficient than anaerobic metabolism. During normal rest and aerobic exercise, carbohydrates and fats are used as fuels. A high degree of aerobic fitness requires a well-adapted ability to take in,

carry, and use oxygen. Laboratory measurements are most accurate, but they are expensive. An individual's fitness level may be estimated according to these measurements.

Anaerobic exercise produces lactic acid and usually is of short duration. Anaerobic exercise is high intensity and has a greater inherent risk of injury. Individuals who are unfit have a lower anaerobic threshold than athletes who are aerobically trained. The well-trained athlete may be able to approach 80% of the  $VO_{2max}$  aerobically without lactate production. The usual  $VO_2$  measurements are in L/min; however, if the size of the individual needs to be accounted for, the measurements may be in mL/kg/min. The values for the average person aged 20 years are 37-48 mL/kg/min. Male athletes who are highly trained may approach measurements in the high 70s to low 80s. Training enhances the ability of the body, in particular the muscle cells, to better handle oxygen. Muscle must be able to use oxygen efficiently to keep anaerobic metabolism at a given level of effort to a minimum.

Cardiac output is a major determinant of oxygen uptake.  $VO_{2max}$  declines with age as the maximum HR declines. This is one of the major factors causing the approximately 7% decline with each decade of life after age 30 years. Muscle training and use of oxygen at the end organ, muscle, is the second factor that affects oxygen uptake. The arterial-venous oxygen difference comes about as a combination of arterial oxygen content, shunting of blood to muscles, and the muscle extraction of oxygen. Training results in a more efficient heart and an increase in the maximum SV. An increase in  $VO_2$  results in an ease in the stress of a given workload. When maximum SV is increased, the heart can work more efficiently at a given pulse rate. This lessens the necessity of an increased pulse at a given workload. Resting pulse is lower, as is the pulse at any given workload.

One metabolic unit (MET) equals the  $VO_2$  at rest. The estimate of the value of one MET is 3.5 mL of oxygen per kg/min. Conversion of  $VO_2$  measurements may be obtained by dividing the value of the  $VO_2$  in mL of oxygen per kg/min by the value



of one MET or 3.5. For example, a  $\text{VO}_2$  measurement of 35 mL of oxygen per kg/min is equivalent to an output of 10 METs.

### **Cardiovascular changes with isometric exercise**

Cardiovascular changes during isometric exercise differ from those during dynamic exercise. Static exercise causes compression of the blood vessels in the contracting muscles, leading to a reduction in the blood flow in them. Therefore, total peripheral resistance, which normally falls during dynamic exercise, does not fall and may, in fact, increase, especially if several large groups of muscles are involved in the exercise. The activation of the sympathetic system with exercise thus leads to an increase in HR, cardiac output, and BP. Because the total peripheral resistance does not decrease, the increase in HR and cardiac output is less and an increase in the systolic, diastolic, and mean arterial pressure is more compared with those seen with dynamic exercise. Because BP is a major determinant of after load, the left ventricular wall stress, and thus the cardiac workload, is significantly higher during static exercise compared with the cardiac workload achieved during dynamic exercise.

### **Cardiac changes following training**

In most cases, the SV plateaus at a  $\text{VO}_2$  of approximately 40-60% of the maximum. This applies to both trained and untrained males and females. The SV for untrained males may approach 100-120 mL/beat/min. For trained males, this value is 150-170 mL/beat/min. For highly trained athletes, maximal SV may reach or even exceed 200 mL/beat/min. The values for women are lower than those for men. Maximal SV for untrained and trained women usually is between 80 mL/beat/min and 100 mL/beat/min, respectively. These changes translate into an increase in the circulation blood volume and in cardiac output, with a corresponding decrease in the resting HR and the resting and exercise BP.

The heart undergoes certain morphological changes in response to chronic exercise, commonly seen via echocardiography. These morphological changes define

what is commonly referred to as an "athletic heart." Athletic heart syndrome is characterized by hypertrophy of the myocardium (ie, an increase in the mass of the myocardium). Although the hypertrophy in athlete's heart is morphologically similar to that seen in patients with hypertension, several important differences exist. In contrast to the hypertension-induced hypertrophy, the hypertrophy in the athletic heart is noted in absence of any diastolic dysfunction, with a normal isovolumetric relaxation time, with no decrease in the peak rate of left ventricular filling, and with no decrease in the peak rate of left ventricular cavity enlargement and wall thinning. Because the wall stress in the athlete's heart is normal, sometimes the hypertrophy seems to be disproportionate to the level of resting BP.

In addition, the rate of decline in the left ventricular hypertrophy and mass is much more rapid when the training is stopped compared with the regression in the same parameters in treated hypertension. On average, the decline in these parameters is seen 3 weeks after stopping exercise, and these morphologic changes can be seen on echocardiograms.

Sometimes, these morphological changes are confused with the changes seen in patients with hypertrophic cardiomyopathy (HCM). A few important morphological differences exist. In athletic heart syndrome, the hypertrophy is usually symmetrical, as opposed to the asymmetric hypertrophy in HCM. Also, the left ventricular size generally is normal or increased and the left atrial size is normal, as opposed to a small left ventricular cavity with a larger left atrial cavity size (usually >4.5 cm) in HCM. Despite these differences, sometimes making a distinction between 2 conditions is a challenge.

## GENERAL EFFECTS OF PHYSICAL ACTIVITY

All of the body's tissues and our genetic material generally look like they did in our ancestors 10,000 years ago. The human body is built for movement. Body and mind benefit from physical activity. Most organs and tissues are affected by physical activity and adapt to regular exercise. Regular physical activity significantly reduces the risk of premature death.

Physical activity can be carried out at different levels of intensity. The more intense, the greater the immediate impact on various bodily functions. Oxygen consumption, which is directly linked to energy expenditure, increases from 0.25 liters per minute at rest to slightly more than 1 liter per minute during a relaxed walk. During maximum exertion, it increases to 2–7 liters per minute, i.e. up to 10–25 times the resting rate.

During physical exertion, the pulse rises and cardiac output increases. Ventilation multiplies, blood pressure increases, body temperature rises, perfusion in the heart and muscles increases, more lactic acid is formed and the secretion of hormones such as adrenaline, growth hormone and cortisol increases.

Maximum oxygen uptake capacity depends on body size, gender, age, fitness level, genetics and more. The factors that limit performance capacity in full-body exertion differ depending on the length of the session. The durations stated in the following text shall only be viewed as approximate for an “average”, middle-aged person – major differences exist. In maximal exertion that lasts 5–15 minutes, central circulation (the heart) is generally considered to comprise the most important limitation of the performance capacity (by limiting the maximum oxygen uptake capacity). The longer the exertion continues, the more performance capacity is limited by properties of the engaged skeletal muscles (mitochondria, capillaries, some transport molecules, buffer capacity, etc.), which affect the so-called anaerobic threshold (see below for definition). In terms of long-term endurance (more than 30–60 minutes), the muscles' carbohydrate deposits (glycogen) also comprise a

limitation.

*What determines the response to exercise training?*

Several factors determine how much a person improves if the degree of physical activity increases. One important factor is the fitness/performance level when the period of exercise training begins. A person who is inactive and in poor shape improves more in relative terms than a person who is well trained. The effects of exercise are specific to the organs and tissues that are exercised – only the muscles that are used adapt and only the parts of the skeleton that are loaded are strengthened. The length of the period of exercise training also plays a major role. Although some effects from exercise can be seen after a surprisingly short time of one to a few weeks, the effects are considerably greater if training continues for several months to years. Of course, the effect of exercise gradually “levels off” and eventually a considerable amount of training is required just to maintain the prevailing level of fitness.

Three other important factors are frequency (how often the person exercises), duration (how long a session is) and intensity (how hard/intense the session is). These three factors determine the combined “exercise dose”. In other words, the higher the dose, the greater the effect. It should be pointed out that low doses also have an effect, although to a more limited extent.

*Frequency*

For physical activity to have the maximum performance and health effects, it must be pursued often and regularly. The effect that an exercise session has can affect the body for several days, and then subside. Consequently, for low-intensity physical activity, a daily “dose” is recommended.

*Duration*

As a rule, the longer the activity continues, the greater the effect it has. In many

cases, the daily activity session can be divided up into several separate 10–15 minute periods, as long as the total time is sufficient. One common recommendation with regard to time is 30 minutes of physical activity per day.

### *Intensity*

The harder an exercise session is, the greater its performance and health effects usually are, although excessively intense exercise can lead to deteriorations. Good health-related effects often seem to be achieved at a lower intensity, although a higher intensity is important to be able to improve fitness and to maintain an improvement in condition.

Moreover, there are of course a large number of factors that affect the outcome of the exercise training. For example, exercise can be conducted with relatively constant or with varying intensity (interval training) and with varying size of the engaged muscle mass (arm, abdomen and leg muscles compared with just leg muscles, for example). Genetics also seem to play a relatively large role in how large the response to exercise training is, perhaps accounting for around a third to one half of the variation between people. There is some evidence in the literature that individuals who increase their performance capacity at a certain exercise dose more than others appear to activate key genes in a stronger way (7, 8). It has not been established whether differences in exercise response are only due to genetic mechanisms (9). Age can be of significance, although older persons do not generally appear to have a worse ability to increase their relative performance. The composition of the diet may also play a role; a deficient diet lessens the response to exercise training. Dietary supplements provide no proven effect, however.

## **EFFECTS OF ACUTE EXERTION AND REGULAR EXERCISE**

When discussing the effects of physical activity on the bodily organs and organ systems, it is necessary to differentiate 1) what happens in the body during (and after) a session of physical activity compared with the situation at rest, and 2) what

differences are achieved (at rest or under exertion) after a certain period of exercise training compared with an untrained condition. In this text, the former is referred to as “Effects of acute exertion” and the latter as “Effects of exercise training”. The effects of acute exertion are due to a number of factors and differ between different tissues. The time for achieving different effects from exercise training varies from function to function, some processes start immediately in connection with the first exercise session, others take weeks to months before they are noticeable.

From a physiological perspective, a physical activity is called either aerobic or anaerobic, depending on which form of metabolism is dominant. One rule of thumb is that physical activity is aerobic (dependent on oxygen) if the maximum time one can perform the activity exceeds two minutes (3). Then the muscles mainly obtain their energy from the oxygen-dependent degradation of carbohydrates or fat. If one has the energy to carry out the activity for two minutes, but no longer, the metabolism is probably approximately 50 per cent aerobic and 50 per cent anaerobic (not oxygen-dependent). In short-term, intense physical activity, the muscles work without a sufficient oxygen supply (anaerobic metabolism) and the dominant energy-providing process is the splitting of glycogen and glucose into the degradation product lactic acid. Consequently, it is natural that aerobic exercise and anaerobic exercise provide different effects of exercise training. Aerobic exercise performed for a sufficient period of time stimulates the adaptation of the heart and the aerobic systems of the skeletal muscles, which is why regular exercise leads to the heart increasing its capacity accompanied by an increased mitochondrial volume in the engaged skeletal muscle cells. The exercise time in pure anaerobic exercise (such as sprint training) is too short to provide these exercise responses in the heart and muscles. Such anaerobic training instead leads to improved conditions for greater lactic acid production and lactic acid tolerance.

Daily physical activity often has elements of both aerobic and anaerobic activities, such as walking in hilly terrain. Strength training, especially with heavy weights, is an extreme form of anaerobic exercise. In interval training (such as interspersed 10–15 second periods of hard exertion and equally long periods of rest),

aerobic and normally anaerobic exercises are combined so that the total period of exercise at heavy loads can be kept sufficiently long to provide an exercise effect on both the heart and the muscles' aerobic systems (81).

### *Measurement of exercise dose and exercise effects*

Measurement of heart rate or perceived exertion (10) are methods to adjust the exercise dose to the person's own capacity. This is discussed in depth in a separate chapter. Pedometers, or step counters, that measure vertical movements, are good aids for measuring the total number of steps when walking and running, but are relatively insensitive to many other movements. To obtain objective measurements of an individual's physical activity during a certain period of time, tri-axis accelerometers are used instead, measuring frequency, intensity and duration of movements on horizontal, sagittal and vertical planes (11).

Training effects in aerobic exercise are often measured as the change in the maximum oxygen uptake capacity. This is the highest oxygen consumption a person can achieve and is measured when the individual works with a maximum pulse during e.g. running. In short-term exercise (5–15 minutes), the maximum performance capacity is largely dependent on the maximum oxygen uptake capacity (12). This can be improved by 20–50 per cent in 2–6 months if exercise is of sufficient intensity. However, there are very large individual differences in the response to exercise. From the maximum oxygen uptake capacity, the individual's maximum energy expenditure can be calculated, since every liter of consumed oxygen corresponds to an amount of energy of approximately 20 kJ (5 kCal). Since direct measurement of the maximum oxygen uptake capacity is relatively difficult, and requires both special equipment and nearly maximum exertion by the individual, an indirect approach is often used where the maximum oxygen uptake capacity is calculated based on heart rate measurements at lower levels of exertion (3). The reliability of the indirect methods is limited, however.

Determination of the anaerobic threshold (lactic acid threshold test) can be achieved in a reliable manner from blood lactic acid samples taken during non-

maximum exertion (13). The anaerobic threshold is defined as “the highest sustained exertion intensity at which lactic acid appearance in the blood will be equal to the rate of its disappearance”. In terms of percentages, it increases more than the maximum oxygen uptake capacity after a period of endurance training. The economy of motion can also be improved with exercise. Accordingly, less energy is consumed for the same amount of work performed (measured as lower oxygen consumption), efficiency is improved. This may be very noticeable for many activities such as running while for others, such as cycling, efficiency is generally identical for all individuals. Besides the aforementioned effects of exercise training, which are significant to performance capacity, important exercise effects can be measured that are primarily of significance to one’s metabolism (and risk of disease). Among these are glycosylated hemoglobin (HbA1C) – an integrated measure of blood sugar increases over an extended period; glucose tolerance – a measure of plasma glucose two hours after the consumption of 75 grams of glucose, inverse measure of insulin sensitivity and blood glucose control; and fat tolerance (14) – a measure of blood lipids in the hours after a fat- rich meal, inversely related to insulin sensitivity. Other methods to measure the effects of regular exercise include underwater weighing, skin-fold, DXA, BOD POD (15) – methods to measure the body’s fat content; and immunoglobulin A in saliva (16) – method of measuring immunity in connection with physical activity.

## **HEART**

### *Acute exertion*

During exercise, which involves large muscle groups, there is a high demand on the heart and blood circulation. The skeletal muscles’ requirement of a supply of oxygen and nutrients and the need for greater removal of carbon dioxide and other waste products as well as the need for temperature and acid-base balance demands greater blood circulation. With help from the autonomic nervous system, this increased need can be felt, which leads to an increase in pulse and the contractile



force of the heart. An increase from a resting heart rate, usually 60–80 beats per minute, up to a pulse of around 120 beats per minute is accompanied by an increase in the heart's stroke volume, meaning the amount of blood pumped out with each heartbeat. Under exertion, heart frequency increases from a resting heart rate of approximately 60–80 beats per minute to the maximum pulse, which can be 150–230 beats per minute depending mainly on age, but also on individual factors. The greater contractile force of the heart increases pressure in both chambers of the heart. Pulse rate and contractile force increase in proportion to the work load and the amount of blood the heart pumps out increases from 4–5 liters per minute at rest to 20–40 liters per minute at maximum exertion intensity. The greater cardiac exertion increases demand on the heart's own circulation (coronary circulation), which increases 5–8 fold.

#### *Effects of exercise training*

The effects of exercise training on the heart, like the majority of organs and tissues, depends on the frequency, intensity and duration of the exercise sessions. After a few months of sufficiently intensive exercise, it is typical for the resting heart rate and the pulse during submaximal exertion to be 5–20 beats per minute lower, for the stroke volume to increase by 20 per cent or more, and for the heart's contractility to improve, which is reflected in a greater ejection fraction (the percentage of the heart's blood volume that is pumped out in one heartbeat) at maximum exertion intensity. The maximum heart rate is unaffected or only slightly decreased. Structurally, the heart's internal volume increases significantly and its wall thickness increases somewhat, which altogether means greater cardiac muscle mass, primarily due to the individual cardiac muscle cells increasing in size. In addition, the number of capillaries and mitochondria increases. The expansion capacity of the coronary vessels is improved with exercise training (17). The extent to which the occurrence of and sensitivity of receptors to signal substances and hormones in the cardiac muscles change with regular exercise is not fully established (18).

## **SKELETAL MUSCLES**

### *Acute exertion*

Acute muscle exertion demands greater activation of individual motor units and the recruitment of more motor units. In low intensity exertion, mainly motor units with slow- twitch muscle fibres (type I) are recruited and in more intense exertion, fibres in fast- twitch motor units (type IIa and IIx) are also recruited.

Muscle exertion demands a great deal of energy in the form of adenosine triphosphate (ATP). The primary sources of energy for ATP production are carbohydrates and fatty acids. Carbohydrates are stored in the form of glycogen in the liver and skeletal muscles. These stores are limited and must be refilled daily. Fatty acids are mostly stored in adipose tissue in virtually unlimited amounts.

Several factors affect the choice of energy sources during exertion, such as:

### *Exertion intensity*

Energy expenditure is proportional to the exercise intensity. At rest, 60 per cent of the energy needed is provided by fats and, in low-intensity exertion, roughly the same proportion of the energy is extracted from fat as from carbohydrates. In more intense exertion, relatively more carbohydrates are used. This is due to several factors, including the fact that the fast, less oxidative and more glycolytic muscle fibers are involved to a greater extent and that the muscle cells' access to oxygen (oxygen pressure) gradually decreases. With higher workloads, the need for carbohydrates per unit of time increases sharply and can reach above 200 grams per hour. In maximum aerobic exertion, virtually only carbohydrates are considered to be burned and in even more strenuous, so-called supramaximum exertion, large additional amounts of carbohydrates are split to lactic acid. The higher the load, the more lactic acid is formed, which makes both muscle tissue and blood more acidic (lower pH). However, the fatty acid need levels out with increasing work load and rarely exceeds 20–30 grams per hour. If exertion continues for several hours, 50 grams per hour may be burned.

The highest level of fat burning in skeletal muscles (grams per minute) is achieved at exercise intensities approximately corresponding to 50 per cent of the maximum oxygen-uptake capacity in the general population and to slightly more than 60 per cent of the maximum oxygen-uptake capacity in very fit individuals (19). However, it should be added that the total fat expenditure (during and after the exertion) is primarily dependent on the total energy expenditure, which is why the fat expenditure in high-intensity exertion is greater overall than in low-intensity exertion lasting the same amount of time. However, the duration of high-intensity exercise is often very short and, consequently, fat expenditure is limited.

#### *Fitness level*

A well-trained person uses more fat for energy extraction and thereby saves carbohydrates with each work load, which means that it is possible to maintain a higher intensity of exertion for a longer period of time.

#### *Duration of exercise*

The longer a session of exercise of sub-maximal intensity lasts, the greater is the proportion of fat used. This is partially related to the gradual emptying of the body's carbohydrate deposits.

#### *Diet*

The composition of the diet also affects which energy sources are used. When fasting or on a fat-rich/carbohydrate-poor diet, fatty acids are used to a greater extent. After so-called carbohydrate loading, carbohydrates are used to a greater extent during exercise, but in spite of this glycogen (the storage form of carbohydrates) also lasts longer.

#### *Body temperature*

In strong hypothermia or heat loading, relatively more carbohydrates are used.

### *Oxygen supply*

When oxygen availability is limited, such as at high altitudes, and when the blood flow to the working arm or leg is restricted, carbohydrates are used to a greater extent. An example of the latter situation is exercise with the arms when held above the heart. In acute exercise, the blood flow increases sharply (by 50–100 times) in the working muscles. This improves the oxygen supply and is mainly due to the vasodilatation (expanding of the vessels) brought about by various factors in the muscles.

### *Muscle fatigue*

Fatigue upon exertion can be due to many different factors in multiple tissues. The local fatigue in the muscles can, for example, be caused by the accumulation of products from ATP decomposition or a lack of glycogen. Dehydration affects the circulating blood volume and can cause fatigue.

## ***Effects of exercise training***

The skeletal muscles comprise a very adaptable tissue. Endurance training affects its structure and function significantly. In terms of the muscles' contractile function, some studies have found increased activation and increased recruitment of motor units after a longer period of exercise training (20). Local fatigue in the working muscles decreases after a period of exercise training.

### *Structure*

The size of the muscle fibers changes only slightly in endurance training, while it can of course increase sharply if the endurance training has elements of strength training. The slow-twitch fibers (type I) can become somewhat larger. In terms of the distribution between fiber types within the type II group (fast-twitch fibers), the proportion of type IIx decreases after around one week at the same time that the proportion of type IIa increases. The transition from type II to type I (slow-twitch)

fibers is very limited in the short term, although the proportion of slow protein increases in many fibers.

The amount of mitochondria increases markedly (see below). The small capillaries also increase in number. This improves the blood flow and extends the perfusion time of the tissue, which facilitates the exchange of oxygen and nutrients. However, in contrast to some animals, it appears to be difficult to affect myoglobin content with exercise in humans.

#### *Transport capacity*

The occurrence of so-called glucose transporters (GLUT-4) in the skeletal muscle cell membrane increases immediately in connection with an exercise session and even more after a longer period of exercise training. This increases sensitivity to insulin and tolerance to sugar (glucose). The occurrence of fatty acid-binding proteins increases on the capillary wall and in the muscle cell. Exercise training also improves the occurrence of special transport molecules for fatty acids into the muscle cell and its mitochondria, and transport molecules for lactic acid out of the muscle cell. This raises the transport capacity considerably. Furthermore, as early as within a few days, the ATPase activity of the sodium- potassium pumps (enzyme rate) increases, which probably improves the ability to restore the ion balance (contractility) after the end of muscle exertion (21).

#### *Nutrient deposits*

The amount of deposited carbohydrates (in the form of glycogen) and fatty acids (in the form of triglycerides) in skeletal muscle cells can be more than doubled (glycogen is tripled or quadrupled) with exercise training.

#### *Fat and carbohydrate use*

The amount of enzymes that break down fatty acids increases very rapidly after exercise training, which facilitates fatty acid use. The mitochondrial density, and consequently the muscle's fat and carbohydrate burning capacity, increases relatively

quickly with exercise training. Within just 4–6 weeks, a 30–40 per cent increase can be noted. The very well- trained have 3–4 times higher mitochondrial densities in trained muscles than those that do not exercise.

After just a few weeks' training, a higher “metabolic fitness” is achieved, meaning that carbohydrates are “saved” and fat is used to a greater extent for energy extraction under exertion at a given, sub-maximal load level. These tangible differences in substrate selection are due in part to higher mitochondrial density and a greater transport capacity for fatty acids. The production and concentration of lactic acid is lower at a given load. The buffer capacity for lactic acid is also improved (22). At maximum exertion (higher exertion intensity possible after exercise training), the lactic acid concentration is significantly higher.

### *Mechanisms*

During acute exertion, the external and internal environments of the skeletal muscle cells change. Hormones and growth factors surround and are bound to the cells to a greater extent. One example is Vascular Endothelial Growth Factor (VEGF), which affects the new formation of blood vessels (23). Inside the cells, the temperature, calcium content and occurrence of the ATP molecule's decomposition products increase. At the same time, pH and oxygen pressure decrease. These and other factors directly and/or indirectly affect a number of proteins in the skeletal muscles. For example, the degree of phosphorylation of so-called mitogen-activated protein kinases (24) and mitochondrial factors (25, 82) is affected, which in turn affects processes that control the adjustment to exercise training, such as the activity degree of some genes. It should also be noted that only the muscles used/exercised adapt, hence, the exercise is specific, which is of crucial significance.

## **BLOOD**

### *Acute exertion*

Upon exertion, the working muscles swell up somewhat, which is due to all of the metabolites formed in the muscle cells osmotically “extracting” fluid from the blood. This fluid withdrawal to the working muscles directly decreases blood volume somewhat, which means that the concentration of hemoglobin (blood value) increases by 5–15 per cent. After the exertion, this increase subsides.

The occurrence of leukocytes also rises sharply under exertion, largely because leukocytes are released from lymphatic tissue, such as lymph nodes and the spleen.

### *Effects of exercise training*

Regular endurance training entails a significant increase in both plasma and blood cells, which together increase blood volume by 10–15 per cent or more (26). The plasma expansion begins as early as the first week after the first exercise session. A change in the number of red blood cells, which is relatively less, can first be observed after a few weeks. Because the plasma volume expands more, the percentage of the blood comprised by red blood cells will drop due to dilution. This is why, paradoxically, a person often has a lower Hb value (blood count) after a period of exercise training despite a higher total amount of red blood cells. The increased blood volume increases the venous return of blood to the heart and consequently the end-systolic volume of the heart, which contributes to increasing the stroke volume and lowering the heart rate at rest and in sub-maximal exertion.

### *Mechanisms*

The increased plasma volume is probably due to hormonal factors that increase fluid retention and to an increased synthesis of albumin that binds more fluid in the plasma. The increased formation of red blood cells is probably due to an increase in the erythropoietin (EPO) concentration.

## LIPOPROTEINS

### *Acute exertion*

Significant changes in blood lipid levels (lipoproteins) are seen after individual exercise sessions, with higher levels of HDL cholesterol (4–43%), especially subfractions 2 and 3 associated with a reduction of the levels of triglycerides and very low density lipoproteins (VLDL). These changes can last 24 hours after the end of exertion (27). A work load corresponding to five kilometres of running at an intensity corresponding to the anaerobic threshold has also been given as a threshold value for achieving these changes (28).

An exercise session, such as a one-hour long brisk walk, done within 24 hours before a fat-rich meal means that the increase in lipoproteins in connection with the meal will be significantly lower than if no exercise session had been done (29). This can probably in part be explained by the enzyme lipoprotein lipase in the skeletal muscles' capillaries being activated by the exertion and increasing its decomposition of the fat molecules (triglycerides) that flow through the capillaries, and in part by a decreased secretion of triglycerides from the liver. Both of these effects are probably connected to the lack of energy (reduced levels of energy substrate) that occurs in the skeletal muscles and liver after an exercise session of a sufficient length (30).

### *Effects of exercise training*

Changes in the composition of lipoproteins with exercise are among the changes that are thought to underlie the reduced risk of cardiopulmonary diseases among fit individuals. Physically fit individuals normally have higher levels of HDL cholesterol (high-density lipoproteins) and lower levels of triglycerides than untrained individuals. The increase in HDL cholesterol is considered to be especially important due to its role in the process (reverse cholesterol transport) whereby the body extracts cholesterol from peripheral tissues for transport to the liver and excretion.

Other changes in prolonged training, although not as constantly occurring,



include lower overall cholesterol and LDL cholesterol (low-density lipoproteins) (83) as well as lower concentrations of Apolipoprotein B. The approximate exercise volumes required to obtain these positive effects from exercise have been given as the equivalent of 25–30 km jogging or fast walking per week, or in other words an exercise-related energy expenditure of 1,200–2,200 kcal per week. Higher exercise volumes entail additional positive effects. With this level of exercise, people of both genders can expect the HDL cholesterol level to rise by 10–20 per cent and the triglyceride level to decrease by 10 to 30 per cent (31).

The significance of relatively extensive physical exercise to influence the composition and concentrations of lipoproteins is clearly exemplified by a large U.S. study (32) in which overweight men and women were divided into four groups, one control group and three exercise groups, that were monitored for eight months. Group A exercised (ergometer cycling, jogging) with an energy expenditure that corresponded to 32 km jogging per week and with a strenuous load (65–80% of maximum oxygen uptake capacity), group B exercised at the same intensity, but with a shorter distance (corresponding to an energy expenditure of 19 km jogging per week), while group C underwent the same amount of exercise as group B, but at a lower intensity (corresponding to 40–55% of the maximum oxygen uptake capacity). After eight months of exercise, the concentration of HDL cholesterol had only increased in group A (+9%), together with several other beneficial lipoprotein changes (such as lower LDL concentrations with an increase in the LDL particles' size). Some changes were also noted in groups B and C (primarily an increase in the size of the LDL and VLDL particles), but to a much lower extent (32).

### ***Blood coagulation factors and platelet characteristics***

#### ***Acute exertion***

An exercise session leads to a significant increase in the number of platelets in the blood. This cannot be explained by the decrease in the plasma volume that occurs in acute exertion (see above), but rather is probably due to a release of blood platelets from various organs, such as the spleen, bone marrow and from pulmonary

circulation. An activation of blood platelets has also been observed during acute exercise sessions, primarily among untrained individuals. Such an activation is reflected in an increased occurrence of the protein, P-selectin, on the platelets' surface, with greater aggregation tendency of platelets and greater formation of thrombin and fibrin leading to a shortened coagulation time. It is mainly higher intensity exertion that provides these potentially negative effects of physical exercise. Studies of individuals with coronary disease provide clear support for the theory that platelet aggregation and activation increase through physical activity. Interestingly, this increase does not appear to be inhibited by acetylsalicylic acid, which is normally an effective treatment for conditions of increased blood clot tendency (33).

These results can be compared with what is known about the risk of being afflicted by a cardiac infarction in connection with physical exertion. It should be noted that it is relatively uncommon for a heart attack to be triggered by physical exertion; only around

5 per cent of cardiac infarctions occur during or within one hour of physical exertion. Of these cases, 70 per cent can be related to coronary occlusion by a platelet-rich blood clot. The definition of physical exertion that is commonly used in these contexts is an energy expenditure that is six times higher than the energy expenditure at sitting rest (six metabolic equivalents or 6 MET), which can normally be said to correspond to light jogging or shovelling snow, for example.

In various studies, the risk of being struck by a heart attack during a randomly selected hour was compared with the same risk during and within one hour after physical exertion.

In several studies, large increases in risk were reported in connection with physical exertion (34, 35). However, the results only apply to untrained men who rarely (less than once a week) subjected themselves to this degree of exertion. Regular exercise constitutes strong protection against the increased risk of cardiac infarction in connection with physical exertion, and the risk has been estimated to only be 2.5 times (34) and 1.3 times (35) greater, respectively, than at rest for men who exercise regularly (>6 MET at least 4–5 days per week). For women, the risk of

being struck by a heart attack during and in connection with physical exertion is very small (compared with the risk during a randomly selected hour without physical exertion), and the small risk that has been reported appears to vanish with regular exercise. For both men and women who exercise regularly, the risk of having a heart attack at all (that is at any hour of the day) is less than half of that among untrained individuals (36).

### *Effects of exercise training*

The sharply reduced risk of sudden death or acute cardiac infarction during an exercise session among people who exercise regularly indicates that prolonged exercise gives rise to changes that counteract the increased aggregation tendency of platelets under exertion. This has also been shown (37). Another important explanation is that the mechanism for the dissolution of blood clots, fibrinolysis, is enhanced among individuals who exercise regularly (38). Other important explanations of the reduced tendency for blood clots among fit individuals may be that regular exercise leads to higher levels of prostaglandin, in part as a result of higher levels of HDL cholesterol, and a higher nitrogen oxide content, both of which inhibit blood clot formation.

## ***VESSELS, BLOOD PRESSURE AND BLOOD FLOW DISTRIBUTION***

### *Acute exertion*

Arterial blood pressure depends on the cardiac output (volume of blood per minute) and the peripheral resistance in the vessel tree. During a session of acute exercise, the cardiac output increases significantly at the same time that peripheral resistance decreases sharply, although not to the same extent that the cardiac output increases. This means that the mean blood pressure increases, almost entirely dependent on a systolic blood pressure increase in proportion to the exertion intensity. At maximum intensity, the blood pressure, measured over the brachial artery, is 180–240 mm Hg. The change in resistance is not uniform in the various

tissues of the body.

In the vessels of the working muscles and the heart, a marked vasodilation occurs with a resulting decrease in resistance. This results in the skeletal muscles' share of the blood flow at rest increasing from approximately one fifth (of 4–5 liters per minute) to approximately four fifths (of 20–30 liters per minute) under exertion. In other groups of vessels, such as in the digestive tract, kidneys and resting skeletal muscles, there is a constriction of the resistance vessels, which decreases the relative perfusion in these areas. Under strenuous exertion, particularly if the exertion is prolonged and occurs in a high ambient temperature, the skin receives a relatively larger share of the blood flow. In the hours after a session of acute exertion, blood pressure drops by 5–20 mm Hg below the normal resting blood pressure.

### *Effects of exercise training*

Up to just two decades ago, researchers believed that the vessels, except the capillaries, were relatively passive tubes that could not change with exercise. However, it turns out that regular exercise improves the function and structure of vessels that supply the engaged skeletal muscles and those in the heart muscle.

The dilation capacity of arterioles is improved and the inner volume of large arteries increases. Thanks to this and the increased capillarisation, an even larger proportion of the blood flow (84) can be guided to the working skeletal muscles. These and other mechanisms, such as increased parasympathetic activity and decreased release of adrenaline and noradrenaline (see the section on the hormone system), contributes somewhat to lowering resting blood pressure among those with normal blood pressure despite an increased blood volume.

### *Mechanisms*

Through the “friction” exerted by the flow of blood against the vessel wall (shear stress), nitric oxide synthase, the enzyme that catalyses the formation of nitric oxide (NO), is induced. NO makes the smooth muscles relax and the vessels open. Capillary formation is probably induced through the formation of so-called

angiogenic factors, of which vascular endothelial growth factor (VEGF) is the most studied. Together with anti-inflammatory mechanisms and better antioxidant activity, the increased NO formation contributes to counteracting arteriosclerosis/atherogenesis (hardening of the arteries/plaque formation).

## **IMMUNE SYSTEM**

### *Acute exertion*

The prevalent view is that regular physical activity of moderate to average intensity improves the function of the immune system and can reduce sensitivity to infections. However, hard or prolonged exercise in endurance sports can lead to immunosuppression and greater infection sensitivity (85).

During sessions of acute exertion, there is an increase in the majority of leukocyte populations in the blood, primarily neutrophil granulocytes and natural killer (NK) cells, and there is also a marked lymphocyte mobilisation to the blood. These changes have been related to the immune-stimulating function of moderate to intense physical exertion (39). However, in the process after a session of intense exertion, there is often a period of weakened immune function with reduced NK-cell activity and reduced lymphocyte proliferation (39). This immunosuppression is also clear in several organs, such as the skin, mucous membranes of the upper respiratory passages, lungs, blood and muscles, and appears to comprise an “open window” of diminished immunity through which viruses and bacteria can enter and gain a foothold in the body. This immunosuppression can last from 3 to 72 hours after an intense exercise session, depending on which immunological markers are measured (40). Among athletes, the risk of developing a clinical infection in this situation can be particularly large upon exposure to new pathogenic flora when travelling, and to a lack of sleep, mental stress, poor food, etc. It has been shown that the concentrations of immunoglobulin A (IgA) and M (IgM) in saliva decrease immediately after an intense exercise session, after which they return to normal levels within 24 hours (41). Prolonged intense exercise can, however, result in a chronic reduction of these

levels. This increases the risk of respiratory infections and it has been proposed that measurements of IgA and IgM in saliva during periods of intense training may be a way of keeping the risk of infection somewhat under control.

Macrophages are important cells in the immune system for phagocytosis, the elimination of microorganisms and tumour cells, and for T-lymphocyte-mediated immunity. There are preliminary indications that physical exertion can stimulate the macrophages' function in the former two areas, and possibly reduce T-lymphocyte-mediated immunity. Hard exercise has also been shown to give rise to raised levels of several both pro- inflammatory and anti-inflammatory cytokines, cytokine inhibitors and chemokines. The increase in the IL-6 cytokine after an exercise session is particularly sharp, but the significance of this increase is still unknown. It is well-known that physical activity leads to an activation of endogenous opioid peptides (86), which probably also plays a role in the immune system (42).

#### *Effects of exercise training*

Research regarding the relationship between the degree of physical exercise, the immune system and sensitivity to infections and other diseases is still in an early phase, and it is often difficult to draw definite conclusions from the results available in the literature. At rest, trained and untrained individuals appear to show relatively small differences in their immune system, with the exception of the activity of NK cells, which are usually higher among well-trained individuals. Besides these changes, reduced function of neutrophil granulocytes and reduced concentrations of NK cells have also been observed among over-trained individuals as a result of prolonged intense exercise (40).

## **SKELETON**

Maximum bone mass is reached at the age of 20–30 years for both men and women, and then slowly decreases with rising age. Besides women having relatively greater bone- mass loss with age, there is also a sharp drop in connection with

menopause. This means that post-menopausal women have significantly less bone mass than men (43). In the majority of studies of the significance of physical activity, bone mass refers to the bone's mineral density or mineral content. Mineral density can be measured through quantitative computed tomography, ultrasound densitometry or dual-energy x-ray absorptiometry (DXA) on selected parts of the skeleton or the entire body. It is also known that the bone's size (volume), structure and protein content are affected by mechanical loading (44, 45), and it has been observed that these variables can often be more informative, since bone density measurements can underestimate the strengthening of the bone structure as a result of exercise (46).

Bone tissue is continuously reformed through resorption and synthesis, whereby the balance between these two processes determines if a net formation or decomposition occurs. How large the bone mass ultimately becomes is also dependent on the initial conditions and time. The common perception is that the balance is determined by the mechanical load the bone cells are subjected to (micro-strain), and that deficient loading leads to decreased bone mass and excess loading to increased bone mass. Important factors include how often the load is repeated and the direction, duration, speed, etc. of the load. For example, dynamic loading with higher speed is more significant than static loading or loads with a low speed. There is also data that indicates that varying types of loading can be more effective than repeating the same loading (47, 48). What in a given situation constitutes the threshold load for bone synthesis to exceed resorption depends on multiple factors, such as the levels of calcium, vitamin D and hormones. When mechanical loading is combined with an increase in oestrogen or androgen levels, the effect on new bone formation is greater than if each factor is allowed to act separately (49). For women, normal ovarian function is therefore very important to the development of bone.

In light of this, it can be expected that it is the intensity of exercise rather than its duration that is important to achieving greater bone density, and that types of exercise with high, temporary loading of the bone (high impact) are particularly effective. The greatest mineral density among athletically active men and women is

also found in sports that involve impact loading, such as weightlifting, aerobics, squash, volleyball and football. The difference in bone density among trained and untrained persons, or between the hitting arm and the non-hitting arm among tennis and squash players, is usually on the magnitude of 10–20 per cent, while changes in bone tissue volume and strength may be larger (44).

It is known that intense physical exercise in adolescence, meaning mechanical loading on the skeleton, results in larger, stronger and more mineral-dense bones and that this effect is more pronounced if the exercise is begun early (50). If the exercise starts at an adult age, only small improvements in bone density are achieved. In spite of this, it has been clearly shown that the risk of a hip fracture is lower among trained individuals, while the proof that exercise at an adult age would reduce other types of fractures related to osteoporosis is not currently as strong (51). On the other hand, monotonous repetitions of the same load over time can cause microscopic damage that weakens the bone and eventually gives rise to so-called stress fractures (52). An interesting finding is that veteran cyclists, with many years of training behind them, have significantly lower bone density than control persons of the same age and, although very physically fit, they therefore have a higher risk of being affected by brittle bones with increasing age (53). Among women, intense exercise training such as long-distance running can also lead to diminished bone density, probably due to hormonal changes, possibly in combination with low energy consumption (49). The state of proof is relatively weak with regard to training with low to moderate intensity exercise also providing a positive effect on the skeleton. In terms of non-weight-bearing activity, such as swimming, such activities do not normally lead to greater bone density.

In light of the fact that half of all women and one third of all men will be affected by a brittle bone fracture in their lifetime (51), it is of great interest to know if it is possible to build up a strong skeleton during adolescence that can protect against fractures later in life. However, available data does not conclusively indicate the existence of any such lasting protective effect (51) and it has been reported that the risk of fractures among former athletes is not lower than among those previously



not athletically active (51). On the other hand, in recent years, studies have been published that support the hypothesis that physical activity in the teenage years and as young adults really is linked to higher bone density late in life, such as for lumbar vertebra (54) and proximal femurs after menopause among women (54, 55), as well as for lumbar vertebra and femurs among older men (56), which in turn could reduce the risk for a fracture in a long-term perspective.

### *Cartilage*

Isolated cartilage cells respond to mechanical loading and an increased strain of a cyclically varying type leads to greater net synthesis of extracellular matrix in cartilage in organ baths. However, static loading commonly leads to decreased matrix production. In animal experiments, both intense physical activity on one hand and total immobilization on the other have proven to lead to osteoarthritis-like changes. Intense and prolonged physical activity in human beings is also probably associated with osteoarthritis in hips and knees (57). The function of cartilage tissue is linked to the interaction between tissue matrix and the extra-cellular fluid that is bound to proteoglycan molecules in the cartilage tissue. Loading leads to deformation of the cartilage with fluid outflow from the tissue matrix to the surroundings, which normalizes in the hours after the exertion. After 100 knee bends in people, this normalization is reported to take more than 90 minutes (58). Consequently, it can be assumed that the balance between deformation and restitution is an important factor, and if this is kept at an appropriate level, damage to the cartilage in connection with physical activity may be avoided (87). What constitutes an appropriate level can probably vary significantly depending on different joint anatomy, joint mobility, etc. (59). There are research results that indicate that kinesiotherapy and passive motion training have positive effects on cartilage tissue by speeding the restitution phase (57).

## *Connective tissue*

Connective tissue responds to strain with increased collagen synthesis, while immobilization has the opposite effect. It is believed that a session of physical activity may in fact lead to increased decomposition of connective tissue as a result of the activation of protease enzymes. Analogous to what was reported above for cartilage, it therefore appears as if the actual exercise session leads to a degradation, meaning a reduction of the synthesis rate, while synthesis markedly increases in the restitution phase in the ensuing days. Consequently, it is the balance between the effect on synthesis and decomposition that determines if a certain training programme leads to improved ligament strength or to a degradation with ruptures or damage as a result (60). It has also been shown that several hormonal growth factors and inflammatory mediators play a role in this balance. An interesting observation is that a considerable net synthesis of new connective tissue often requires several weeks or months of exercise, because the enhanced decomposition is most pronounced at the beginning of a period of exercise and can counteract the increased new formation of connective tissue (60). The strong ligaments that characterize well-trained individuals provide greater sustainability because the load per cross-sectional area decreases.

## **LUNGS AND GAS EXCHANGE**

### *Acute exertion*

In low-intensity exertion, it is mainly the size of each breath (tidal volume) that increases. In more high-intensity exertion, the respiratory rate increasingly rises. Altogether, this means that the pulmonary ventilation increases from 6–8 liters per minute at rest to up to 150 liters per minute among the untrained and up to 200 liters per minute among well-trained persons under maximum exertion.

Under exertion, large amounts of oxygen is consumed and roughly the same amount of carbon dioxide is formed. Despite the sharply increased carbon dioxide formation, the content in arterial blood and exhalation air decreases at maximum

exertion. This is due to the pulmonary ventilation increasing by 15–30 times at the same time that carbon dioxide formation increases by only 10–15 times. The extraction of oxygen from arterial blood increases from around 25 per cent at rest to more than 75 per cent under strenuous exertion.

### *Effects of exercise training*

The pulmonary ventilation under maximum exertion increases. Under sub-maximum exertion, the respiratory rate, tidal volume and consequently pulmonary ventilation is significantly lower after exercise training. Exercise training improves the endurance of the respiratory muscles. This occurs by adaptations in the same way as in other skeletal muscles that are regularly exercised (see above). The lungs' blood flow distribution changes and there is a lesser degree of mismatch between perfusion and air ventilation; the upper parts of the lungs in particular receive a greater blood flow. The lungs' gas diffusion improves.

### *Mechanisms*

The probable underlying causes behind the training-induced changes in the respiratory muscles are the same as for other muscles (see above). In terms of the improved blood flow distribution in the lungs, it may be due to the increased blood volume combined with changes in the vessels of the lungs.

## **BODY COMPOSITION AND ADIPOSE TISSUE**

The sharp increase in overweight and obesity that has occurred in the Western World in the past 15–20 years has been associated with growing inactivity, although the relative significance of decreased physical activity compared with altered caloric intake and meal- time patterns is unknown (61).

The energy expenditure when running on level ground is on a magnitude of 1 kcal per kg of body weight and kilometer, while the corresponding value for walking is 20–25 per cent lower. Accordingly, one hour of walking corresponds to 1/10 of the

energy expenditure per day of a standard man (2,800 kcal per day) or woman (2,100 kcal per day). It being difficult and nearly impossible to predict on an individual level how more physical activity will affect body weight and body composition is illustrated by the fact that three glasses (of 2dl each) of a soft drink that may be consumed in connection with training also corresponds to 10 per cent of the daily energy needs. It has been said that the increase in the average weight of 20–40 year-olds in the U.S. in the 1990s (approximately 7–8 kg in eight years) could have been avoided if 100 kcal more of energy on average had been expended (or 100 kcal less food consumed) per day. This corresponds to just 15–20 minutes of walking or one glass of a soft drink (62).

The appetite can also be affected by physical activity in various ways. Low energy levels and low levels of insulin in plasma, which is often observed after an exercise session, stimulates the appetite through neuropeptide Y-releasing neurons in the central nervous system. On the other hand, intense exertion can lower the appetite through the release of corticotropin-releasing hormone (CRH) from the hypothalamus with an anorectic effect. At the population level, knowledge about how regular physical activity affects body composition is more certain, and several major studies with observation times of approximately 3–4 months show that various exercise programmes can be expected to provide a decrease in fat weight of an average of 0.1 kg per week. As a rule, the decrease in fat weight is always larger than the decrease in body weight, and body weight often does not change at all due to increased muscle mass (63). The decrease in fat weight is seen in both genders. Although a tendency of larger decreases are seen in men, it cannot be said for certain that any gender difference exists.

There is support from studies of rats that exercise training-induced changes occur in adipose tissue similar to those seen in skeletal muscles, with increases in both mitochondrial enzyme activity and in the level of glucose transport protein (GLUT-4). One difference from the skeletal muscles (where exercise training provides a decrease) is that there is an increase in adipose tissue of the hormone-sensitive lipase enzyme (HSL) with training, that is to say the enzyme responsible for

the release of fatty acids (lipolysis) into the blood. This agrees with studies in organ baths of fat cells from humans and rats, where it could be shown that adrenaline (which stimulates HSL) gives rise to significantly greater release of fatty acids in fat cells that were taken from trained individuals than in fat cells from untrained individuals. It is known that being overweight leads to lower HSL concentrations in the adipose tissues, but that the concentration increases in connection with periods of fasting. The increased adrenaline effect on the release of fatty acids among trained individuals can, however, also be due to a higher level of the adenylate cyclase enzyme in the fat cells. Adenylate cyclase conveys the effects of adrenaline by giving rise to the messenger molecule cyclic adenosine monophosphate, cAMP. The number of receptors for adrenaline on the surface of the fat cells is probably not affected by exercise, however. To some extent, the increased fat degradation activity in adipose tissue from trained individuals can be seen as a compensation for a lower overall adipose tissue mass in a trained individual (64).

In the past decade, it has been discovered that adipose tissue is significantly more metabolically active than was previously known. Today, it is known that several potent peptides are released from adipose tissue and have important effects on other organs in the body. Two such peptides are leptin, which has an anorectic effect on the energy balance and also affects sugar metabolism, and adiponectin, which stimulates fat burning. Adipose tissue also releases pro-inflammatory proteins such as tumour-necrosis factor alpha (TNF- $\alpha$ ) and other cytokines and acute phase proteins. Angiotensinogen (AGT) formed in adipose tissue affects blood pressure, and can play a role in the blood pressure increase seen in overweight individuals. It has not been established how physical activity and exercise training affect these factors, but the decreased fat mass seen with exercise training can be expected to decrease the significance of these factors (88). Leptin has been examined in several studies, but there does not appear to be any unambiguous effect of exertion or exercise training on leptin levels. However, lower plasma levels of TNF- $\alpha$  have been observed among well-trained people, which is of interest because TNF- $\alpha$  formed by adipose tissue is considered to result in diminished tissue sensitivity to insulin, primarily in skeletal

muscles. A decreased level of TNF- $\alpha$  could therefore contribute to the greater insulin sensitivity that exercise training entails (65).

## **NERVOUS SYSTEM**

Much of the knowledge that applies to the effects of acute exertion and exercise training on the nervous system is gathered from studies of animals, but growing numbers of human studies of cognition and learning are being published.

### *Acute exertion*

During exertion, the brain has a total metabolism and total blood flow that do not significantly differ from that at bodily rest. However, during exertion, the activity, metabolism and blood flow in the areas that take care of motor activity increase measurably. The glucose concentration increases interstitially in the central nervous system (CNS) regardless of the blood sugar concentration. Besides glucose, the brain uses lactic acid as an energy substrate under intense exertion. The release of neurotransmitters (signal substances) such as dopamine, serotonin and glutamate in various parts of the brain are also affected during physical exertion.

### *Effects of exercise training*

Regular physical activity affects several different functions in the human nervous system (89). Functions connected more directly to physical activity improve, such as coordination, balance and reaction ability. This increases the functional ability, which can contribute to the increased well-being which is tied to regular physical activity. Moreover, cognitive ability (especially planning and coordination of tasks) is retained better, sleep quality is improved, depression symptoms decrease and self-esteem improves. Experiments in animals have shown that growth factors significant to cells in the central nervous system are affected by physical activity (66). In the hippocampus (important to memory formation), the gene expression of a large number of factors increases. For example, the occurrence of IGF-1, a very

important growth factor, increases. The occurrence of noradrenaline increases in the brain. There are also studies that indicate that the new formation of brain cells increases in animals that are allowed to run (67). These animals also show better learning ability. Other studies have shown that the new formation of vessels increases in the cerebral cortex after exercise training, which can be of significance to the supply of nutrients. In cells in the peripheral nervous system, studies in animals have shown that markers for oxidative capacity/aerobic capacity increase. In addition, there are findings that indicate that cell size can increase with regular physical activity.

### *Mechanisms*

The increased metabolism associated with more activity in parts of the cells of the brain, spinal cord and peripheral nervous system entails an effect on gene activity, in part caused by increased production of growth factors such as brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF) and galanin (68). Local hypoxia may potentially drive the formation of new blood vessels around the brain cells.

## **SKIN**

### *Acute exertion*

Under acute exertion, especially prolonged exertion in heat, perfusion of the skin increases sharply and the degree of sweating can be multiplied many times over. A well-trained person can excrete 2–3 liters of sweat per hour under extreme conditions. Various hormones affect the sweat glands so that salt is largely saved.

### *Effects of exercise training*

Exercise improves sweating function and thereby heat-regulation capacity. Therefore, a well-trained person has better heat tolerance at rest and under exertion. Among other factors, this is due to altered perfusion and changed gene expression in the cells of the sweat glands. Regular physical activity reduces the amount of

subcutaneous fat.

## **GASTROINTESTINAL TRACT AND LIVER**

### *Acute exertion*

The gastrointestinal tract is affected in many ways during and after acute exertion (69). Under strenuous exertion, symptoms occur such as stomach aches, diarrhoea, etc. It is not easy to determine the degree to which such symptoms are related to stress, dietary and fluid intake, or the physical exertion. The frequency at which the stomach empties decreases, at most during strenuous exertion. Besides motility, digestion and absorption, the gastrointestinal tract's blood flow, its secretion of hormones and other factors are affected. The stomach's lymphoid tissue and the mucous membranes' immunological functions, such as IgA, are also affected. Under exertion, the liver increases its glycogenolysis, which contributes to maintaining blood sugar (see below under "Hormone system").

### *Effects of exercise training*

A well-trained person has a higher gastric emptying rate. The risk of the formation of gall stones is also reduced.

## **HORMONE SYSTEM**

### *Acute exertion*

Several hormone systems are activated under exertion, and physical activity entails increased plasma concentrations of multiple hormones, such as adrenaline/noradrenaline, adrenocorticotrophic hormone (ACTH), cortisol, beta endorphin, growth hormone, renin, testosterone, thyroid hormone and several gastrointestinal hormones. The levels of glucagon in arterial blood is only affected to a small degree by physical exertion, while the concentration of insulin decreases (70). The decrease in the insulin level in plasma during exertion, which can be very sharp



(a drop to half or less of the resting level), is probably mediated by the increased activity in sympathetic nerves and by small reductions in the blood glucose level during the exertion. The latter explains why the insulin decrease under exertion is counteracted or even converted to an insulin increase upon sugar intake during the exercise session. Because glucagon, like insulin, exerts a significant part of its effect on the liver, one is at risk of underestimating the significance of glucagon under exertion when measuring arterial concentrations since the concentration in the portal vein, which is the concentration “seen” by the liver, is significantly higher and probably significantly raised under exertion (71).

The catecholamines adrenaline and noradrenaline increase sharply and exponentially with increasing work load. The source of the circulating adrenaline is the adrenal medulla and the increase in plasma adrenaline in physical exertion is due to increased sympathetic nerve activation of this organ. Although the blood’s noradrenaline also comes in part from the adrenal medulla, the most important cause of the sharp rise in the plasma content of noradrenaline under exertion is a “flood” of noradrenaline from the sympathetic nerves. The most important sympathetic nerves in this respect are those stimulating a higher heart rate and cardiac contractile force during exertion, as well as those which innervate the liver and adipose tissue. It is believed that a lower glucose concentration in the portal vein is an important cause of the strong activation of the sympathetic nervous system under exertion. The increase in noradrenaline starts at a lower work load than the increase in adrenaline, and noradrenaline also increases more sharply when exertion intensity increases. These hormones can increase 10–20 fold in strenuous or prolonged exertion. The noradrenaline content of the blood is often raised for several hours after the end of exertion, while the adrenaline concentration goes back to resting values within a few minutes (72).

The liver’s greater release of glucose is one of the most important metabolic changes under exertion and compensates for the muscles’ increased glucose uptake without the blood glucose level dropping too much. It is practically entirely caused by the changes in insulin and glucagon (73). The reduction of the plasma insulin level

that takes place with exertion is believed to make the liver more sensitive to the glycogen-degrading effect of glucagon. The increased activation of the sympathetic nervous system during physical exertion appears to lack any direct significance to the liver's increased glucose release. However, under prolonged exertion, when the adrenaline levels are at their highest, adrenaline can have some stimulatory effect on the liver's glucose release in addition to glucagon. Adrenaline and noradrenaline are mainly of significance to the carbohydrate metabolism at the muscle level by making the muscle's glycogen degradation process sensitive to the stimulatory effect that the contraction process (actually the calcium ions that are released) has. However, if prolonged exercise leads one to "hit the wall" due to a blood glucose reduction, a crisis reaction is triggered, whereby adrenaline is released, which leads to an increase in the liver's glucose release. The liver's limited glycogen deposits mean that new synthesis of glycogen in the liver (so-called gluconeogenesis) becomes important in prolonged exertion (in addition to the sugar consumed by drinking). Here, the hormone cortisol plays an indirect role by increasing the capacity of the enzymatic machinery that takes care of this process.

Another crucial enzymatic process during physical exertion is the release of free fatty acids from the body's fat deposits, since free fatty acids are the body's other important nutrient during exertion. Here, noradrenaline, released by the sympathetic nerves that innervate adipose tissue, plays the most important role. Insulin has an inhibitory effect on the release of fatty acids, although this effect is diminished by its plasma concentration dropping sharply during exertion.

Increased levels of beta endorphines during prolonged exercise can be of significance to well-being and blood pressure reduction in connection with an exercise session (74).

### *Effects of exercise training*

Naturally, lower hormone responses at a given work load are observed among well-trained than among untrained individuals. This applies to the increases in noradrenaline, adrenaline, growth hormone, ACTH and glucagon as well as the

reduction in insulin. The reduced hormonal activation during exertion among well-trained persons is particularly notable with regard to the sympathetic nervous system, where the change occurs rapidly, normally during the first two weeks of exercise (75). The physiological mechanism behind this rapid change is unknown, but the activation of stress hormones that occurs with other stress stimuli is not reduced among fit individuals. It is also well-known that the adrenal medulla's capacity to excrete adrenaline is greater among well-trained individuals (sports adrenal medulla).

The so-called hypothalamus-pituitary-adrenal (HPA) axis is a messenger for the body's responses to various states of stress. The resting state of the HPA system is affected by regular endurance training so that the daily rhythm is shifted (the morning peak comes earlier) and the release of the pituitary glands control hormone ACTH is increased. Although this can be interpreted as a hormonal stress state in the trained body, the effector hormone of the HPA axis, cortisol, does not change its resting level as a result of regular exercise, however. This apparent paradox seems to be explained by the fact that cortisol provides less effective feedback inhibition of the pituitary and possibly of the hypothalamus in well-trained individuals, which leads to an increased level of ACTH (76, 77). This is suspected to be one of several different explanations of the menstruation disruptions that occur in female athletes. Disturbances of the reproductive system in male athletes are rarely discussed, but may also exist (90).

Well-trained individuals have lowered insulin concentrations in plasma, both basally and after sugar intake, due to both a reduced release of insulin from the islets of Langerhans (78) and an increased tissue sensitivity to insulin (79). The increased insulin sensitivity is strongly linked to the reduced risk of having cardiovascular disease that is characteristic of physically trained individuals. As described above in the section on adipose tissues, regular exercise leads to an increased capacity for lipolysis in the adipose tissue. This contributes to a well-trained person being able to maintain a sufficient fat release during physical exertion even though the activation of the sympathetic nervous system, which controls lipolysis, is sharply reduced. Regular exercise has a carbohydrate-saving effect by a large part of the energy need

being met with the burning of fat. This is registered by the liver and, after just 10 days of exercise, the liver's glucose release during a two-hour exercise bout can be reduced by 25 per cent (80). In spite of this, regular physical exercise leads to a greater capacity for gluconeogenesis in the liver.

## **EXERCISE PRESCRIPTION**

Substantial data are available on the benefits of physical activity. For primary preventative benefits, physical activity patterns should begin in the early school years and continue throughout an individual's life. Schools must specifically designate physical education programs with aerobic activities for children at early ages. Programs should include recreational sports (eg, running, dancing, swimming). Support at home for an active lifestyle for children helps to promote healthy physical activity patterns.

In the clinical setting, discuss physical activity and provide exercise prescriptions for patients and their families. In some instances, suggestions could be made about implementing physical activity recommendations at the work site.

Consider intensity, duration, frequency, mode, and progression in all types of physical activity programs. As children and adolescents become adults and discontinue the athletic endeavors of school and college, primary prevention must include a plan for a lifetime of appropriate physical activity. Ideally, this activity should be performed for at least 30-60 minutes, 4-6 times weekly or 30 minutes on most days of the week. The frequency, duration, and intensity of activity should be individualized to personal satisfaction, mode, and progression.

Subjects may use individual end points of exercise, such as breathlessness and/or a fatigue level ranging from somewhat hard to hard on the Borg perceived exertion scale described above. Standardized charts that designate heart rates may help by providing heart rate end points that can be measured immediately after exercise, but these are not necessary. Exercise should include aerobic activities, such as bicycling (stationary or routine), walk-jog protocols, swimming, and other active recreational or leisure sports. Shoes and clothing should be appropriate for extremes of heat, cold, and humidity.

Resistive exercises using free weights or standard equipment should be performed 2-3 times per week. These exercises should include 8-10 exercise sets

consisting of 10-15 repetitions per set (including arms, shoulders, chest, trunk, back, hips, and legs), performed at a moderate intensity. If free weights are used, 15-30 lb is generally adequate or resistance that requires a perceived effort that is relatively hard, ie, an RPE 15-16. Resistive exercises tend to complement aerobic exercise in that some training effect is realized. However, development of muscle tone and strengthening of body musculature is more important as adults age.

The long-term effect of any physical activity program is affected by compliance. In today's mobile society, an exercise plan must include activities for business trips and vacations. Exercise facilities may not be convenient in such settings, which may mean improvising. For example, a walk-jogger should bring walking or running shoes and find a safe place to walk or run at a pace that approximates the usual activity level. Many hotels or motels have exercise facilities with a track or treadmill, exercise cycle, and weights, enabling travelers or others away from their usual routine to maintain an exercise program.

Physical activity measured in total time or kilocalories (or kilojoules) per week is appropriate and may be achieved with various combinations of scheduling, such as 10-15 minutes in the morning, at noon, and/or an afternoon/evening session. Many persons may schedule longer, less frequent periods of exercise. As intensity decreases, frequency and duration should increase and vice versa. The dosage or total energy (calorie) expenditure per week must be individualized.

Persons with influenza syndromes or respiratory illnesses should decrease or stop exercise until they have recovered. If the recovery time is greater than 2-3 weeks, activity should be resumed at a lower level to compensate for the slight loss in training level. Maintenance of the cardiovascular training effects of exercise has been shown to be more related to the exercise intensity than to exercise frequency or duration. In other words, if the intensity is maintained, even though the exercise sessions are less frequent or shorter in duration, transient reductions in conditioning from the decreased exercise appears to be minimized.

Various exercise testing measures of functional capacity should be used in special populations but are not necessary for primary prevention. Traditionally, many

athletically inclined persons like to have periodic  $\text{VO}_2$  measurements to assess their level of training. However, recent technologic advances have made cardiopulmonary (CPX) or metabolic (CMET) testing more commonplace among medical practices and have become increasingly popular as a part of a routine physical and stress test evaluation, especially for individuals who are considered at high risk for cardiovascular disease (eg, those with hypertension, shortness of breath, chest discomfort, or abnormal blood lipid levels).

A lifestyle of physical activity from childhood throughout the adult years fosters health and longevity. Even brisk walking as a physical activity/exercise habit promotes health benefits. This is the simplest program for most individuals and has clear benefits. This improved state of health is enhanced by weight control, restricted intake of saturated fat and cholesterol, abstinence from cigarette smoking, and control of high blood pressure and glucose intolerance.

### **Benefits of exercise**

Routine exercise improves tissue oxygen uptake, improves insulin sensitivity, decreases blood pressure, increases high-density lipoprotein levels, decreases low-density lipoprotein and triglyceride levels, improves glycemic control in persons with type 2 diabetes, and, hence decreases overall mortality. Considerable data also support that exercise may decrease the prevalence of colon cancer and endometrial cancer. Exercise also helps with osteoarthritis and obesity. Exercise has also been reported to benefit persons with migraine headaches and fibromyalgia.

Middle-aged men and women who work in physically demanding jobs or perform moderate-to-strenuous recreational activities have fewer manifestations of coronary artery disease than their less active peers. Meta-analysis studies of clinical trials reveal that medically prescribed and supervised exercise can reduce mortality rates for persons with coronary artery disease.

Several studies suggest that relatively small amounts of physical activity show considerable reductions in mortality and improved health outcomes among participants when compared with sedentary control subjects. These findings imply

that a minimal activity (ie, exercising once per week) may have positive health benefits even though fitness may not be measurably improved. Some have suggested that a threshold of physical activity may be necessary for maintaining optimal health and that future investigation of this should use controls subjects who participate in at least minimal activity levels rather than comparing exercise treatment groups to the control subjects who are completely sedentary.

In addition to the physical benefits of exercise, both short- and long-term aerobic exercise training are associated with improvements in various indexes of psychological functioning. Cross-sectional studies reveal that compared with sedentary individuals, active persons are more likely to be better adjusted, to perform better on tests of cognitive functioning, to exhibit reduced cardiovascular responses to stress, and to report fewer symptoms of anxiety and depression.

In one report, persons who increased their activity levels from 1965-1974 were at no greater risk for depression than those individuals who were active all along; however, persons who were active and became inactive were 1.5 times as likely to become depressed by 1983 compared with those who maintained an active lifestyle. Longitudinal studies have also documented significant improvement in psychological functioning. Exercise training reduces depression in healthy older men and in persons with cardiac disease or major depression.

Exercise also improves self-confidence and self-esteem, attenuates cardiovascular and neurohumoral responses to mental stress, and reduces some type A behaviors. Although exercise training generally has not been found to improve cognitive performance, short bouts of exercise may have short-term facilitative effects.

Despite the positive physical and mental health benefits of exercise, long-term adherence to exercise programs remains problematic. Overall physical activity levels decrease with aging, in minority populations, in females, in disabled persons, and in those with chronic disease. Only an estimated 50% of all persons who initiate an exercise program continue the habit for more than 6 months. The issue of nonadherence is particularly important because exercise is only beneficial if it is



maintained for extended periods. Thus, developing strategies to improve exercise initiation and adherence, especially for persons who are among the least active (eg, 75% of African American women; less educated, obese, elderly persons), is important.

### **Different types of exercise**

Exercise has been defined as an activity for the express purpose of improving fitness or health. Physical activity includes all forms of activity (eg, occupational, recreational, sports-related) performed without the specific purpose of fitness or health. Different types of exercise are as follows:

- Aerobic (eg, walking, swimming)
- Anaerobic (eg, sprinting)
- Isometric (eg, lifting weights)
- Resistance training: This involves providing some form of resistance to the contracting muscles to stimulate the body to increase strength. Multiple types of equipment are used for resistance training, including hand weights, cam machines, pulleys, and hydraulic, elastic, rubber, fiberglass, and magnetic equipment.

- Strength or resistance training is very important to improve functionality and reduce the risk of injury. As people age, the lean tissue (ie, muscle) declines more from lack of use than from aging itself. Performing some type of resistance training regularly is imperative.

- Because the demand on the heart is generally less while strength training than while walking at a moderate pace, resistance training is regarded as safe for patients with many heart conditions. Patients should never strain or hold their breath while attempting to lift something. Straining can adversely affect blood flow to the heart.

### **Resistance and repetitions**

- Resistance: The appropriate resistance may be provided by hand weights, elastic resistance, calisthenics, or machines and should be no more than what

one can lift for approximately 15-20 repetitions. Perceived effort should only be moderate or somewhat hard.

- Repetition: A set is a group of repetitions, such as 2 or 3 sets of 15 repetitions. The number of sets depends on several factors, including time constraints, motivation, and personal goals. One to 3 sets are adequate for strength development. Add 1 set per week, increasing up to 3 sets.

- Progress: Progression can be made as one finds that the weight being used can be lifted more than 20-25 times. One should then increase the resistance slightly (eg, add 1-5 lb) and resume the training. As one reaches muscle fatigue, more stimulation of the muscle tissue results in protein being added to the muscle groups. Significant strength changes generally occur within 6 weeks.

- Other: Stretching should also be part of the exercise plan.

### **Type of exercise (mode)**

- Intensity: This should range from low to moderate for healthy individuals.

- Duration: Continuous aerobic activity for 20-60 minutes is recommended.

- Frequency

- Individuals with a less than 3-MET capacity should engage in multiple short sessions each day.

- Individuals with a 3- to 5-MET capacity should engage in 1-2 sessions per day.

- Individuals a greater than 5-MET capacity should engage in 3-5 sessions per week.

### **Energy (caloric) expenditure**

- Per-minute calculation: To calculate kilocalories per minute, multiply the METs times 3.5 times body weight in kilograms and divide by 200 (METs X 3.5 X kg body weight/200). For example, the energy (caloric) expenditure of a 70-kg

individual at a prescribed 6-MET capacity with a weekly goal of 1000 kcal per week is calculated as  $6 \times 3.5 \times 70 \text{ kg}/200$ , which equals 7.35 kcal/min (30.87 kJ/min). To convert kilocalories to kilojoules, note that 1 kcal equals 4.2 kJ.

- Per-week calculation: This determines the exercise duration per week. Using the numbers from the example above, divide 1000 kcal (4200 kJ) by 7.35 kcal/min (30.87 kJ/min), which equals 136 min/wk or approximately 20-30 minutes, 6 d/wk.

### **Lifetime activities**

- Vary the type of activity. Pick an activity that is enjoyable. The activity can be any activity that uses most muscles, elevates the heart rate, and may be sustained for 20 minutes or longer. For example, one may find stationary cycling boring but enjoy playing tennis or racquetball.

- Vary the duration and intensity within the guidelines. Some days, decrease the intensity of the activity but increase the duration. On other days, warmup and then increase the intensity to the upper range of the guidelines but decrease the duration.

- Contract with a friend (buddy system) or participate in group classes.

- Use music for rhythm. If an activity is boring, either change it or find another one that is enjoyable. For example, if one is stationary cycling, videos that show outdoor scenery are available. Also, new saddles are available that make cycling much more comfortable.

- Make exercising enjoyable by selecting at least 2 activities that are enjoyable.

- Conditioning may be realized from many activities if applied correctly.

- Walk the dog daily, whether one has a dog or not.

### **Selecting the right physical activities**

- Select physical activities that are enjoyable, use most of the muscles, are rhythmic, and may be sustained for several minutes to an hour.

- Plan to exercise every other day until more adequately adapted the activity.

- Think of the frequency, intensity, time, and type (ie, FITT) plan.

- Frequency: This is how often per week one will perform the exercise.

Plan on most days of the week.

- Intensity: This is how hard one exercises. Moderate effort is appropriate.

- Time: This is the duration of each session. Start off with as little as needed (10 min if necessary).

- Type: This is the choice of physical activity, which can include recreational activities and domestic or occupational activities. A short list of each follows:

- Recreational activities - Aerobic activity classes, backpacking, badminton, baseball, basketball, body building, bowling, boxing, calisthenics, canoeing, climbing hills, cricket, cycling, dancing, fencing, fishing, playing catch (eg, Frisbee), gardening, golf, gymnastics, handball, hiking, horseback riding, hunting, in-line skating, lacrosse, low-impact aerobics, martial arts, orienteering, racquetball, rope skipping, rugby, running, sailing, scuba-diving, shuffleboard, skating, skiing, snow shoeing, stair climbing, swimming, table tennis, tennis, volleyball, walking, water polo, water activities, weightlifting, and windsurfing

- Domestic or occupational activities - Cleaning windows, housework, mowing, packing and unpacking, plowing, sanding, sawing, sweeping, stocking shelves, pushing a wheelbarrow, yard work, and many others

- Set goals, which may include health, improving physical capacity, or performance.

- Motivation may be helpful for compliance. See the following tips:

- Join a class or facility or contract with a friend (buddy system).

- Listen to one's body (eg, slowing down or skipping if tired or ill). Start at the present level to prevent soreness.

- Exercise at the same time each day.

- Make sure to have good quality nutrition.

- Make exercising a priority; scheduling a time benefits the individual.
- Get advice if help is needed.

## CLASSIFICATION OF SPORTS BY CONTACT LEVEL

<b>Contact/Collision Sports</b>	<b>Limited-Contact Sports</b>	<b>Noncontact Sports</b>
Basketball	Baseball	Archery
Boxing	Bicycling	Badminton
Diving	Cheerleading	Bodybuilding
Field hockey	Canoeing/kayaking (white water)	Canoeing/kayaking (flat water)
Football, flag or tackle	Fencing	Crew/rowing
Ice hockey	Field events: high jump, pole vault	Curling
Lacrosse	Floor hockey	Dancing
Martial arts	Gymnastics	Field events: discus, javelin, shot put
Rodeo	Handball	Golf
Rugby	Horseback riding	Orienteering
Ski jumping	Racquetball	Powerlifting
Soccer	Skating: ice, inline, roller	Race walking
Team handball	Skiing: cross-country, downhill, water	Riflery
Water polo	Softball	Rope jumping
Wrestling	Squash	Running
	Ultimate Frisbee	Sailing
	Volleyball	Scuba diving
	Windsurfing/surfing	Strength training
		Swimming
		Table tennis
		Tennis
		Track
		Weightlifting

## SPORTS OF HIGH TO MODERATE INTENSITY

<b>Sports With High to Moderate Dynamic and Static Demands</b>	<b>Sports With High to Moderate Dynamic and Low Static Demands</b>	<b>Sports With High to Moderate Static and Low Dynamic Demands</b>
Boxing	Badminton	Archery
Crew/rowing	Baseball	Auto racing
Cross-country skiing	Basketball	Diving
Cycling	Field hockey	Equestrian
Downhill skiing	Lacrosse	Field events (jumping)
Fencing	Orienteering	Field events (throwing)
Football	Table Tennis	Gymnastics
Ice hockey	Race walking	Karate or judo
Rugby	Racquetball	Motorcycling
Running (sprinting)	Soccer	Rodeo
Speed skating	Squash	Sailing
Water polo	Swimming	Ski jumping
Wrestling	Tennis	Water skiing
	Volleyball	Weightlifting

## TESTS

1. Bradycardia concerns, when pulse more small after:

- A. 70 per min;
- B. 60 per min;
- C. 50 per min;
- D. 20 per min;
- E. There is not a right answer.

2. By a simple spirometry it is possible to define the index of pulmonary ventilation:

- A. Index of Tiphno;
- B. Speed of exhalation;
- C. Volume of the forced exhalation;
- D. All answers are correct;
- E. All answers are not correct.

3. Changes of adaptations of the respiratory system are during the physical training:

- A. Decline of minute volume of breathing;
- B. Decline of respiratory coefficient;
- C. Multiplying a respiratory volume;
- D. A and C;
- E. There is not a right answer.

4. Characterizes static coordination:

- A. Test of Yarotskyj;
- B. Rotation in an arm-chair Ram;
- C. The test of Romberg is complicated;
- D. Finger-nasal test;



E. There is not a right answer.

5. Diastolic noise concerns:

- A. At once after the first tone;
- B. Between the first and second tones;
- C. Immediately in front of by the first tone;
- D. After the second tone;
- E. There is not a right answer.

6. Direct ECG-signs of hypertrophy of myocardium is:

- A. Multiplying amplitude of indent of P;
- B. Multiplying duration of indent of P;
- C. Multiplying amplitude of indents of R and P;
- D. Multiplying the width of complex QRS more than 0,12 s;
- E. There is not a right answer.

7. During the peak of sporting form develops:

- A. Physiology hypotension;
- B. Physiology hypertension;
- C. An arterial pressure does not change;
- D. B and C;
- E. There is not a right answer.

8. Early systolic noise concerns:

- A. At once after the second tone;
- B. At once after the first tone;
- C. Between the first and second tones;
- D. Before the second tone;
- E. Before the first tone.

9. Factors which reduce the level of arterial pressure under act of the physical training:

- A. Increase of shock volume of heart;
- B. Decline of common peripheral resistance of vessels;
- C. Multiplying the retractive function of myocardium;
- D. A and C;
- E. There is not a right answer.

10. For a sportsman-athlete (there are broad jumps) marked the index of mass of body, even 24,0. It means:

- A. Norm;
- B. Deficit of mass of body;
- C. Surplus mass of body;
- D. B and C;
- E. All answers are faithful.

11. For a sportswoman-sprinter marked the index of mass of body 18. It is considered as:

- A. Norm;
- B. Deficit of mass of body;
- C. Surplus mass of body;
- D. All answers are correct.
- E. All answers are not correct.

12. For an athlete-decathlon competitor marked the index of mass of body 32. It is considered as:

- A. Norm;
- B. Deficit of mass of body;
- C. Surplus mass of body;
- D. But also In;

E. All answers are faithful.

13. For description of the vegetative nervous system use:

- A. Test of Yarotskyj;
- B. Temp-test;
- C. Test of Ashner;
- D. Genucalcaneal test;
- E. Test of Bare.

14. For determination of what pathology ultrasonic research of heart can help for a sportsman:

- A. Dystrophy of myocardium;
- B. Valvular defects;
- C. Violation of rhythm of heart;
- D. Chronic aneurysm of heart;
- E. There is not a right answer.

15. For sportsmen distinguish the types of circulation of blood, after an exception:

- A. Hypokinetic;
- B. Hyperkinetic;
- C. Macrokinetic;
- D. Eukinetic;
- E. There is not a right answer.

16. For visual description of degree of development of skeletal musculature does not use a criterion:

- A. Volume;
- B. Force;
- C. Relief;

- D. Resiliency;
- E. There is not a right answer.

17. Function of myocardium, which it cannot find out on ECG:

- A. Conductivity;
- B. Activity;
- C. Automatism;
- D. Contractility;
- E. There is not a right answer.

18. High cardiac production characteristic at development:

- A. Physiology hypertrophy of myocardium;
- B. Pathological hypertrophy of myocardium;
- C. Physiology dilatation of ventricles;
- D. A and B;
- E. There is not a right answer.

19. In the first phase of myogenic leucocytoses number of leucocytes of peripheral blood:

- A. Diminishes;
- B. Does not change;
- C. Increased;
- D. Deviates;
- E. There is not a right answer.

20. In the norm of oscillation of relative closeness of urine for a sportsman makes:

- A. 1004-1010;
- B. 1006-1020;
- C. 1015-1027;

- D. 1030-1040;
- E. 1035-1045.

21. In the types of sport on endurance of pulse in a space hold can be less after:

- A. 70 per min;
- B. 60 per min;
- C. 50 per min;
- D. 30 per min;
- E. There is not a right answer.

22. In urine of healthy sportsman at peace in a period rest from muscular activity of squirrel:

- A. Present;
- B. Absent;
- C. Present sometimes;
- D. A and C;
- E. There is not a right answer.

23. Registration of vibrations of arterial wall at distribution of pulse wave for carries the name vessels:

- A. Tachoscylography;
- B. Reography;
- C. X-ray kimography;
- D. Ballistic cardiography;
- E. Sphygmography.

24. The size of arterial pressure in most sportsmen is registered at level:

- A. High bound of norm;
- B. Low bound of norm;
- C. Does not differ from a norm;

- D. Higher statistical norm;
- E. Below statistical norm.

25. To description of the vegetative nervous system of sportsman does not belong:

- A. Quality of sleep;
- B. External secretion;
- C. Vestibular firmness;
- D. Dermography;
- E. Thermal resistance.

26. Vital lungs volume most for sportsmen in the types of sport:

- A. Ski double-event;
- B. Heel-and-toe walk;
- C. Handball;
- D. Water-polo;
- E. Pentathlon.

27. Volume of air, which directly takes part in the pulmonary interchange of gases is in a pulmonary capacity:

- A. Vital capacity of lights;
- B. Functional capacity of lights;
- C. General capacity of lights;
- D. Capacity of exhalation;
- E. Capacity of inhalation.

28. Volume of air, which fizzles out from lungs during 1 minute named:

- A. Respiratory volume;
- B. Pulmonary ventilation;
- C. Vitally lungs volume is forced;

- D. Maximal ventilation of lights;
- E. There is not a right answer.

## REFERENCES

1. Shephard RJ, Balady GJ. Exercise as cardiovascular therapy. *Circulation* 1999;99:963- 72.
2. Saltin B, Gollnick PD. Skeletal muscle adaptability. Significance for metabolism and performance. In: Peachey L, Adrian R, Gaiger S, eds. *Handbook of physiology*. Section 10. Skeletal muscle. Baltimore: Williams & Wilkins Company; 1983. pp. 555- 631.
3. Astrand P-O, Rodahl K, Dahl HA, Stromme SB. *Textbook of work physiology. Physiological bases of exercise*. 4. edn. Champaign (IL): Human Kinetics; 2003.
4. Wilmore JH, Costill DL. *Physiology of sport and exercise*. 3. edn. Champaign (IL): Human Kinetics; 2004.
5. McArdle WD, Katch FI, Katch VL. *Exercise physiology. Energy, nutrition, and human performance*. 5. edn. Philadelphia: Lippincott Williams & Wilkins; 2001.
6. Booth FW, Chakravarthy MV, Gordon SE, Spangenburg EE. Waging war on physi- cal inactivity. Using modern molecular ammunition against an ancient enemy. *J Appl Physiol* 2002;93:3-30.
7. Timmons JA, Jansson E, Fischer H, Gustafsson T, Greenhaff PL, Riddén J, et al. Modulation of extracellular matrix genes reflects the magnitude of physiological adap- tation to aerobic exercise training in humans. *BMC Biol* 2005;3:19.
8. Timmons JA, Larsson O, Jansson E, Fischer H, Gustafsson T, Greenhaff PL, et al. Human muscle gene expression responses to endurance exercise provide a novel per- spective on Duchenne muscular dystrophy. *FASEB J* 2005;19:750-60.
9. Rankinen T, Bray MS, Hagberg JM, Perusse L, Roth SM, Wolfarth B, et al. The human gene map for performance and health-related fitness phenotypes. The 2005 update. *Med Sci Sports Exerc* 2006;38:1863-88.



10. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982;14:377-81.
11. Crouter SE, Churilla JR, Bassett Jr DR. Estimating energy expenditure using accelerometers. *Eur J Appl Physiol* 2006;98:601-12.
12. Bassett Jr DR, Howley ET. Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc* 2000;32:70-84.
13. Svedahl K, MacIntosh BR. Anaerobic threshold. The concept and methods of measurement. *Can J Appl Physiol* 2003;28:299-323.
14. Herd SL, Kiens B, Boobis LH, Hardman AE. Moderate exercise, postprandial lipidemia, and skeletal muscle lipoprotein lipase activity. *Metabolism* 2001;50:756-62.
15. Fields DA, Goran MI, McCrory MA. Body-composition assessment via air-displacement plethysmography in adults and children. A review. *Am J Clin Nutr* 2002;75:453-67.
16. Gleeson M, Pyne DB, Callister R. The missing link in exercise effects on mucosal immunity. *Exerc Immunol Rev* 2004;10:107-28.
17. Bowles DK, Woodman CR, Laughlin MH. Coronary smooth muscle and endothelial adaptations to exercise training. *Exerc Sport Sci Rev* 2000;28:57-62.
18. Zanesco A, Antunes E. Effects of exercise training on the cardiovascular system. Pharmacological approaches. *Pharmacol Ther* 2007;114:307-17.
19. Achten J, Jeukendrup AE. Optimizing fat oxidation through exercise and diet. *Nutrition* 2004;20:716-27.
20. Doherty TJ. Effects of short-term training on physiologic properties of human motor units. *Can J Appl Physiol* 2000;25:194-203.
21. Green HJ. Adaptations in the muscle cell to training. Role of the Na<sup>+</sup>-K<sup>+</sup>-ATPase. *Can J Appl Physiol* 2000;25:204-16.
22. Hawley JA. Adaptations of skeletal muscle to prolonged, intense endurance training. *Clin Exp Pharmacol Physiol* 2002;29:218-22.
23. Gustafsson T, Rundqvist H, Norrbom J, Rullman E, Jansson E,

Sundberg CJ. The influence of physical training on the angiotensin and VEGF-A systems in human skeletal muscle. *J Appl Physiol* 2007;103:1012-20.

24. Widegren U, Wretman C, Lionikas A, Hedin G, Henriksson J. Influence of exercise intensity on ERK/MAP kinase signalling in human skeletal muscle. *Pflugers Arch* 441:317-22.

25. Bengtsson J, Gustafsson T, Widegren U, Jansson E, Sundberg CJ. Mitochondrial transcription factor A and respiratory complex IV increase in response to exercise training in humans.

26. Sawka MN, Convertino VA, Eichner ER, Schnieder SM, Young AJ. Blood volume. Importance and adaptations to exercise training, environmental stresses, and trauma/ sickness. *Med Sci Sports Exerc* 2000;32:332-48.

27. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. *Med Sci Sports Exerc* 2001;33:S438-45, discussion S452-3.

28. Park DH, Ransone JW. Effects of submaximal exercise on high-density lipoprotein cholesterol subfractions. *Int J Sports Med* 2003;24:245-51.

29. Petitt DS, Cureton KJ. Effects of prior exercise on postprandial lipemia. A quantitative review. *Metabolism* 2003;52:418-24.

30. Gill JM, Hardman AE. Exercise and postprandial lipid metabolism. An update on potential mechanisms and interactions with high-carbohydrate diets. Review. *J Nutr Biochem* 2003;14:122-32.

31. Durstine JL, Grandjean PW, Davis PG, Ferguson MA, Alderson NL, DuBose KD. Blood lipid and lipoprotein adaptations to exercise. A quantitative analysis. *Sports Med* 2001;31:1033-62.

32. Kraus WE, Houmard JA, Duscha BD, Knetzger KJ, Wharton MB, McCartney JS, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *N Engl J Med* 2002;347:1483-92.

33. El-Sayed MS. Exercise and training effects on platelets in health and disease. *Platelets* 2002;13:261-6.

34. Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ,

Muller JE. Triggering of acute myocardial infarction by heavy physical exertion. Protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators. *N Engl J Med* 1993;329:1677-83.

35. Willich SN, Lewis M, Lowel H, Arntz HR, Schubert F, Schroder R. Physical exertion as a trigger of acute myocardial infarction. Triggers and Mechanisms of Myocardial Infarction Study Group. *N Engl J Med* 1993;329:1684-90.

36. Sundberg CJ, Jansson E. Reduced morbidity and the risk of premature death. Regular physical exercise is beneficial for health at all ages. . *N Engl J Med*, 1998;95:4062-7.

37. Wang JS, Jen CJ, Chen HI. Effects of exercise training and deconditioning on platelet function in men. *Arterioscler Thromb Vasc Biol* 1995;15:1668-74.

38. Rauramaa R, Li G, Vaisanen SB. Dose-response and coagulation and hemostatic factors. *Med Sci Sports Exerc* 2001;33:S516-20, discussion S528-9.

39. Malm C, Celsing F, Friman G. Fysisk aktivitet bade stimulerar och hammar immunforsvaret. [Physical activity both stimulates and inhibits the immune defence.] *Ldkartidningen* 2005;102:867-73.

40. Nieman DC. Special feature for the olympics. Effects of exercise on the immune system. Exercise effects on systemic immunity. *Immunol Cell Biol* 2000;78:496-501.

41. Gleeson M, Pyne DB. Special feature for the olympics. Effects of exercise on the immune system. Exercise effects on mucosal immunity. *Immunol Cell Biol* 2000;78:536-44.

42. Gleeson M. Special feature for the olympics. Effects of exercise on the immune system. Overview. Exercise immunology. *Immunol Cell Biol* 2000;78:483-4.

43. Russo CR, Lauretani F, Bandinelli S, Bartali B, Di Iorio A, Volpato S, et al. Aging bone in men and women. Beyond changes in bone mineral density. *Osteoporos Int* 2003;14:531-8.

44. Heinonen A, Sievanen H, Kannus P, Oja P, Vuori I. Site-specific skeletal response to long-term weight training seems to be attributable to principal loading modality. A pQCT study of female weightlifters. *Calcif Tissue Int* 2002;70:469-74.
45. Saino H, Luther F, Carter DH, Natali AJ, Turner DL, Shahtaheri SM, et al. Evidence for an extensive collagen type III proximal domain in the rat femur. II. Expansion with exercise. *Bone* 2003;32:660-8.
46. Kontulainen S, Sievanen H, Kannus P, Pasanen M, Vuori I. Effect of long-term impactloading on mass, size, and estimated strength of humerus and radius of female racquetsports players. A peripheral quantitative computed tomography study between young and old starters and controls. *J Bone Miner Res* 2003;18:352-9.
47. Turner CH, Takano Y, Owan I. Aging changes mechanical loading thresholds for bone formation in rats. *J Bone Miner Res* 1995;10:1544-9.
48. Lanyon LE. Functional strain in bone tissue as an objective, and controlling stimulus for adaptive bone remodelling. *J Biomech* 1987;20:1083-93.
49. Balasch J. Sex steroids and bone. Current perspectives. *Hum Reprod Update* 2003;9:207-22.
50. Kannus P, Haapasalo H, Sankelo M, Sievanen H, Pasanen M, Heinonen A, et al. Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. *Ann Intern Med* 1995;123:27-31.
51. Karlsson M. Does exercise reduce the burden of fractures? A review. *Acta Orthop Scand* 2002;73:691-705.
52. Midtby M, Magnus JH. Normal bone remodelling. What can go wrong in osteoporosis? *Tidsskr Nor Laegeforen* 1998;118:552-7.
53. Nichols JF, Palmer JE, Levy SS. Low bone mineral density in highly trained male master cyclists. *Osteoporos Int* 2003;14:644-9.
54. Rideout CA, McKay HA, Barr SI. Self-reported lifetime physical activity and areal bone mineral density in healthy postmenopausal women. The importance of teenage activity. *Calcif Tissue Int* 2006;79:214-22.
55. Rikkonen T, Tuppurainen M, Kroger H, Jurvelin J, Honkanen R.

Distance of walking in childhood and femoral bone density in perimenopausal women. *Eur J Appl Physiol* 2006;97:509-15.

56. Lynch NA, Ryan AS, Evans J, Katzel LI, Goldberg AP. Older elite football players have reduced cardiac and osteoporosis risk factors. *Med Sci Sports Exerc* 2007;39:1124-30.

57. Karlsson MK, Nordqvist A, Karlsson C. Physical activity, muscle function, falls and fractures. *Food Nutr Res.* 2008; 52: Published online 2008 December 30.

58. Eckstein F, Tieschky M, Faber S, Englmeier KH, Reiser M. Functional analysis of articular cartilage deformation, recovery, and fluid flow following dynamic exercise in vivo. *Anat Embryol (Berl)* 1999;200:419-24.

59. Saxon L, Finch C, Bass S. Sports participation, sports injuries and osteoarthritis. Implications for prevention. *Sports Med* 1999;28:123-35.

60. Kjaer M, Langberg H, Magnusson P. Overuse injuries in tendon tissue. Insight into adaptation mechanisms. *Ugeskr Laeger* 2003;165:1438-43.

61. Eisenmann JC, Bartee RT, Wang MQ. Physical activity, TV viewing, and weight in U.S. youth. 1999 Youth Risk Behavior Survey. *Obes Res* 2002;10:379-85.

62. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment. Where do we go from here? *Science* 2003;299:853-5.

63. Ballor DL, Keeseey RE. A meta-analysis of the factors affecting exercise-induced changes in body mass, fat mass and fat-free mass in males and females. *Int J Obes* 1991;15:717-26.

64. Enevoldsen LH, Stallknecht B, Langfort J, Petersen LN, Holm C, Ploug T, et al. The effect of exercise training on hormone-sensitive lipase in rat intra-abdominal adipose tissue and muscle. *J Physiol* 2001;536:871-7.

65. Straczkowski M, Kowalska I, Dzienis-Straczkowska S, Stepien A, Skibinska E, Szelachowska M, et al. Changes in tumor necrosis factor-alpha system and insulin sensitivity during an exercise training program in obese women with normal and impaired glucose tolerance. *Eur J Endocrinol* 2001;145:273-80.

66. Cotman CW, Engesser-Cesar C. Exercise enhances and protects brain function. *Exerc Sport Sci Rev* 2002;30:75-9.
67. Van Praag H, Christie BR, Sejnowski TJ, Gage FH. Running enhances neurogenesis, learning, and long-term potentiation in mice. *Proc Natl Acad Sci USA* 1999;96:13427-31.
68. Dishman RK, Berthoud HR, Booth FW, Cotman CW, Edgerton VR, Fleshner MR, et al. Neurobiology of exercise. *Obesity* 2006;14:345-56.
69. Moses FM. The effect of exercise on the gastrointestinal tract. *Sports Med* 1990;9:159- 72.
70. Galbo H. The hormonal response to exercise. *Diabetes Metab Rev* 1986;1:385-408.
71. Wasserman DH, Lacy DB, Bracy DP. Relationship between arterial and portal vein immunoreactive glucagon during exercise. *J Appl Physiol* 1993;75:724-9.
72. Christensen NJ, Galbo H, Hansen JF, Hesse B, Richter EA, Trap-Jensen J. Catecholamines and exercise. *Diabetes* 1979;28:58-62.
73. Wasserman DH, Lickley HL, Vranic M. Interactions between glucagon and other counterregulatory hormones during normoglycemic and hypoglycemic exercise in dogs. *J Clin Invest* 1984;74:1404-13.
74. Jonsdottir IH, Hoffmann P, Thoren P. Physical exercise, endogenous opioids and immune function. *Acta Physiol Scand Suppl* 1997;640:47-50.
75. Winder WW, Hagberg JM, Hickson RC, Ehsani AA, McLane JA. Time course of sympathoadrenal adaptation to endurance exercise training in man. *J Appl Physiol* 1978;45:370-4.
76. Wittert GA, Livesey JH, Espiner EA, Donald RA. Adaptation of the hypothalamo- pituitary adrenal axis to chronic exercise stress in humans. *Med Sci Sports Exerc* 1996;28:1015-9.
77. Duclos M, Corcuff JB, Arsac L, Moreau-Gaudry F, Rashedi M, Roger P, et al. Corticotroph axis sensitivity after exercise in endurance-trained athletes. *Clin Endocrinol (Oxf)* 1998;48:493-501.
78. Wasserman DH. Regulation of glucose fluxes during exercise in the

postabsorptive state. *Ann Rev Physiol* 1995;57:191-218.

79. Henriksson J. Influence of exercise on insulin sensitivity. *J Cardiovasc Risk* 1995;2:303-9.

80. Mendenhall LA, Swanson SC, Habash DL, Coggan AR. Ten days of exercise training reduces glucose production and utilization during moderate-intensity exercise. *Am J Physiol* 1994;266:E136-43.

81. Gibala M. Molecular responses to high intensity interval exercise. *Appl Physiol Nutr Metab* 2009;34:428-32.

82. Hood DA. Mechanisms of exercise-induced mitochondrial biogenesis in skeletal muscle. *Appl Physiol Nutr Metab* 2009;34:465-72.

83. Vislocky LM, Pikosky MA, Herron Rubin K, Vega-LWF, Zern TL, Lofgren IE, Luz Fernandez M, Rodriguez NR. Habitual consumption of eggs does not alter the beneficial effects of endurance training on plasma lipids and lipoprotein metabolism in untrained men and women. *Journal of Nutritional Biochemistry* 2009; 20:26–34.

84. Laughlin MH, Roseguini B. Mechanisms for exercise training-induced increases in skeletal muscle blood flow capacity: differences with interval sprint training versus aerobic endurance training. *J Physiol Pharmacol* 2008;59 Suppl 7:71-88.

85. Moreira A, Delgado L, Moreira P, Haahtela T. Does exercise increase the risk of upper respiratory tract infections? *Br Med Bull.* 2009;90:111-31.

86. Mathur N, Pedersen BK. Exercise as a mean to control low-grade systemic inflammation. *Mediators Inflamm.* Epub 2009.

87. Hunter DJ, Eckstein F. Exercise and osteoarthritis. *J Anat.* 2009;214(2):197-207.

88. Van Praag H. Exercise and the brain: something to chew on. *Trends Neurosci.* 2009 May;32(5):283-90.

89. Van Praag H. Exercise and the brain: something to chew on. *Trends Neurosci.* 2009 May;32(5):283-90.

90. Hackney AC. Effects of endurance exercise on the reproductive system

of men: the "exercise-hypogonadal male condition". J Endocrinol Invest.  
2008;31:932-8.



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