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Theoretical part
Ergometry

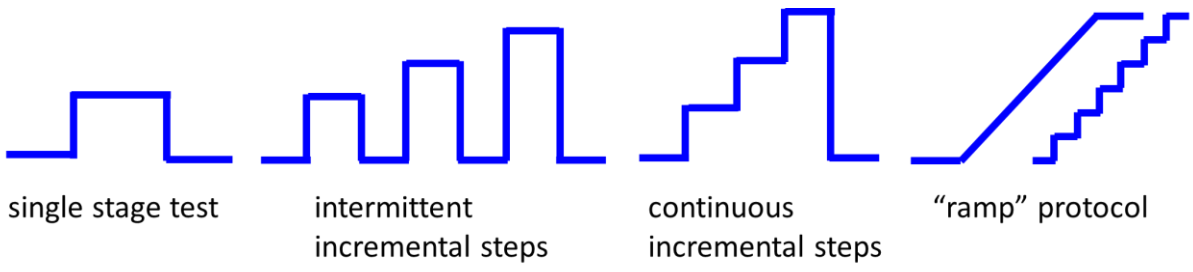
In order to evaluate how bodies react to stress, we can use dynamic, static, electric, pharmacological, cold, hypoxic, and mental tests. In most cases we use the dynamic test (exercise).

Exercise

- Static (isometric) – muscle develops force, but muscle length does not change
  - Dynamic (isotonic) - muscle length varies continuously, but force does not change
    - Positive - muscle shortens against constant or rising resistance, some of the energy in the muscle is converted into kinetic or potential energy
    - Negative - muscle during contraction is driven by external force, the bulk of the energy is converted into heat
  - Auxotonic - strength and muscle length are changing

There are several phases in each dynamic test (ergometry):

- preparatory phase: preparation for the test, connecting to equipment
- resting phase: recording of resting values
- "warmup" phase: application of low workload in order to increase tissue perfusion and improve joint mobility
- load phase: exposure of examined person to graduated physical effort
- "cooldown phase": low-intensity workout supporting removal of catabolites (lactic acid), helping heart rate recovery, reducing vertigo and collapses (due to hypotension following effort)
- recovery phase: follow-up exercise



Indications for examination

- Basic medical examination of athletes
- Indication of preventive cardiology
- Indications of differential diagnosis

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- Assessment indication
- Pharmacotherapeutic indications

Contraindications

Absolute contraindications:

- Systolic blood pressure decreases by more than 10 mmHg with increase in work rate, or drops below baseline in the same position, with other evidence of ischemia
- Increase in nervous-system symptoms: dizziness, ataxia or near syncope
- Moderate to severe anginal pain (above 3 on standard 4-point scale)
- Signs of poor perfusion, e.g. cyanosis or pallor
- Request of the test subject
- Technical difficulties (e.g. difficulties in measuring blood pressure or ECG)
- ST Segment elevation of more than 1 mm in aVR, V_1 or non-Q wave leads
- Sustained ventricular tachycardia

Relative indications:

- Systolic blood pressure decreases by more than 10 mmHg with increase in work rate, or drops below baseline in the same position, without other evidence of ischemia
- ST or QRS segment changes, e.g. more than 2 mm horizontal or downsloping ST segment depression in non-Q wave leads, or marked axis shift
- Arrhythmias other than sustained ventricular tachycardia e.g. premature ventricular contractions, both multifocal or triplet; heart block; supraventricular tachycardia or bradyarrhythmias
- Intraventricular conduction delay or bundle branch block or that cannot be distinguished from ventricular tachycardia
- Hypertensive response (systolic blood pressure > 250 mmHg or diastolic blood pressure > 115 mmHg)

Complications

- In 0.05% of cases – acute myocardial infarction or malignant arrhythmia
- The risk of sudden death in patients is approximately 0.01%
- Other risks are potential muscle or joint injuries as the result of excessive load (this risk is particularly high in patients of retirement age)
- There may also marginal complications such as dizziness, weakness and persistent fatigue

Break indication of stress test

- Typical symptoms of angina pectoris
- Dyspnoea
- Ischemic ECG signs, especially typical ischemic ST depression progressing with increasing workload
- Rise of SBP above 240 mmHg or DBP above 120 mmHg

Occurrence of the following changes on ECG: various forms of sudden tachycardia, atrial fibrillation, blockade in connection with the workload

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Preparation for examination

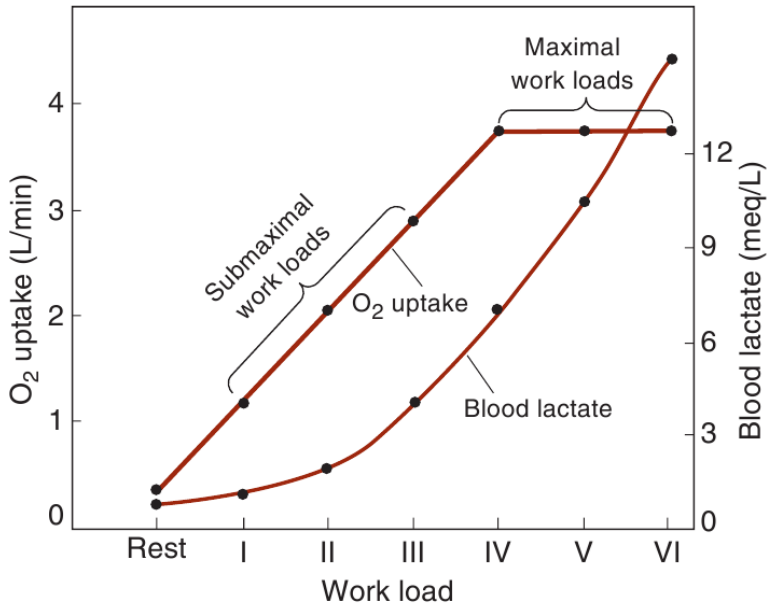
It is recommended that for at least 3 hours before the examination, the patient does not eat or drink in large quantities, or smoke, and at least 12 hours before examination the patient should refrain from unusual physical exercise.
After consulting with a doctor, the following medications should be discontinued:

- beta-blockers (eg. Vasocardin, Betaloc, Egiloc, Tenormin, Concor, Lokren, Sectral, etc.)
- nitrates (Cardiket, Isomer, Iso-Mack, Mycor, Nitro-Mack, Mack Mono, Olicard, Sorbimon, Corvaton, Molsihexal, etc.)

Effects of exercise

Changes in ventilation

During exercise, the amount of O_2 entering the blood in the lungs increases, because the amount of O_2 added to each unit of blood and the pulmonary blood flow per minute are increased. The Po_2 of blood flowing into the pulmonary capillaries falls from 40 to 25 mmHg or less, so that the alveolar–capillary Po_2 gradient is increased and more O_2 enters the blood. Blood flow per minute increases from 5.5 L/min to as much as 20–35 L/min. The total amount of O_2 entering the blood therefore increases from 250 mL/min at rest to values as high as 4000 mL/min. The amount of CO_2 removed from each unit of blood is increased, and CO_2 excretion increases from 200 mL/min to as much as 8000 mL/min. The increase in O_2 uptake is proportional to work load, up to a maximum. Above this maximum, O_2 consumption levels off and the blood lactate level continues to rise (Figure 1). The lactate comes from muscles in which aerobic resynthesis of energy stores cannot keep pace with their utilization, and an oxygen debt is incurred.



Ventilation increases abruptly with the onset of exercise, which is followed after a brief pause by a further, more gradual increase (Figure 2). With moderate exercise, the increase is due mostly to an increase in the depth of respiration; this is accompanied by an increase in the respiratory rate when the exercise is more strenuous. Ventilation abruptly decreases when

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exercise ceases, which is followed after a brief pause by a more gradual decline to pre-exercise values. The abrupt increase at the start of exercise is presumably due to mental stimuli and afferent impulses from proprioceptors in the muscles, tendons, and joints. The more gradual increase is presumably humoral, even though arterial pH, P co_2, and Po_2 remain constant during moderate exercise. The increase in ventilation is proportional to the increase in O_2 consumption, but the mechanisms responsible for the stimulation of respiration are still the subject of much debate. It can be caused by an increase in body temperature, increase in the plasma K^+ level, or changes of CO_2 and O_2 in blood.

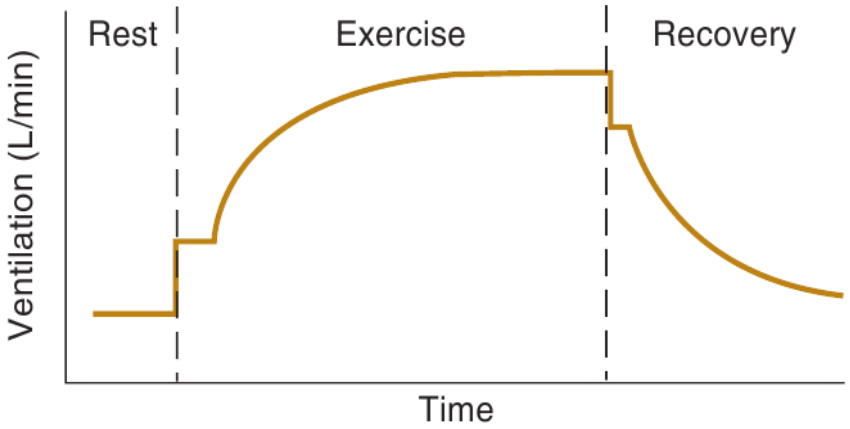

Figure1: relationship between work load, blood lactate level, and O 2 uptake.

When exercise becomes more vigorous, buffering of the increased amounts of lactic acid that are produced frees up more CO_2, and this further increases ventilation. With increased production of acid, the increases in ventilation and CO_2 production remain proportional, so alveolar and arterial CO_2 change relatively little. Because of the hyperventilation, alveolar Po_2 increases. With further accumulation of lactic acid, the increase in ventilation outstrips CO_2 production and alveolar Pco_2 falls, as does arterial Pco_2. The decline in arterial Pco_2 provides respiratory compensation for the metabolic acidosis produced by the additional lactic acid. The additional increase in ventilation produced by the acidosis is dependent on the carotid bodies and does not occur if they are removed.

The respiratory rate after exercise does not reach basal levels until the O_2 debt is repaid. This may take as long as 90 minutes. The stimulus to ventilation after exercise is not the arterial Pco_2, which is normal or low, nor the arterial Po_2, which is normal or high, but the elevated arterial H^+ concentration due to the lactic acidemia. The magnitude of the O_2 debt is the amount by which O_2 consumption exceeds basal consumption from the end of exertion until the O_2 consumption has returned to preexercise basal levels. During repayment of the O_2 debt, the O_2 concentration in muscle myoglobin rises slightly. ATP and phosphorylcreatine are resynthesized, and lactic acid is removed. Eighty per cent of the lactic acid is converted to glycogen and 20% is metabolized to CO_2 and H_2O.

Cardiovascular system in exercise

Cardiovascular control during exercise involves systemic regulation (cardiovascular centers in the brain, with their autonomic nervous output to the heart and systemic resistance vessels) in tandem with local control. Increased sympathetic drive elevates the heart rate and cardiac contractility, resulting in increased cardiac output; local factors in the coronary vessels

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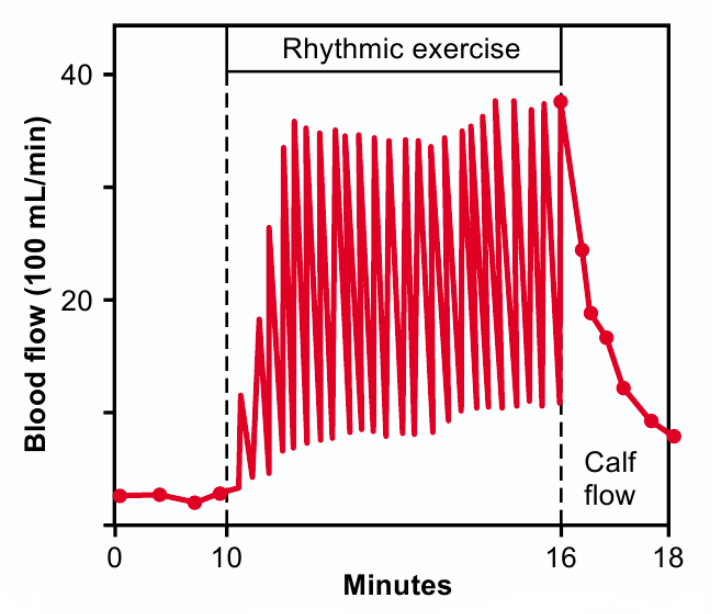
mediate coronary vasodilation. Increased sympathetic vasoconstrictor tone in the renal and splanchnic vascular beds, and in inactive muscle, reduces blood flow to these tissues. Blood flow to these inactive regions can fall by 75% if exercise is strenuous. Increased vascular resistance and decreased blood volume in these tissues helps maintain blood pressure during dynamic exercise. In contrast to blood flow reductions in the viscera and in inactive muscle, the brain autoregulates blood flow at constant levels independent of exercise. The skin remains vasoconstricted only if thermoregulatory demands are absent.

Muscle blood flow

A key requirement of cardiovascular function in exercise is delivering the required oxygen and other nutrients to the exercising muscles. For this purpose, the muscle blood flow increases drastically during exercise. Figure 3 shows a recording of muscle blood flow in the calf of a person for a period of 6 minutes during moderately strong intermittent contractions. Note not only the great increase in flow—about 13-fold—but also the flow decrease during each muscle contraction. Two points can be made from this study: (1) the actual contractile process itself temporarily decreases muscle blood flow because the contracting skeletal muscle compresses the intramuscular blood vessels; therefore, strong tonic muscle contractions can cause rapid muscle fatigue because of lack of delivery of enough oxygen and other nutrients during the continuous contraction; (2) the blood flow to muscles during exercise increases markedly. The following comparison shows the maximal increase in blood flow that can occur in a well-trained athlete.



Thus, muscle blood flow can increase a maximum of about 25-fold during the most strenuous exercise. Almost one half of this increase in flow results from intramuscular vasodilation caused by the direct effects of increased muscle metabolism. The remaining increase results from multiple factors, the most important of which is probably the moderate increase in arterial blood pressure that occurs in exercise, usually about a 30 per cent increase. The increase in pressure not only forces more blood through the blood vessels but also stretches the walls of the arterioles and further reduces the vascular resistance.

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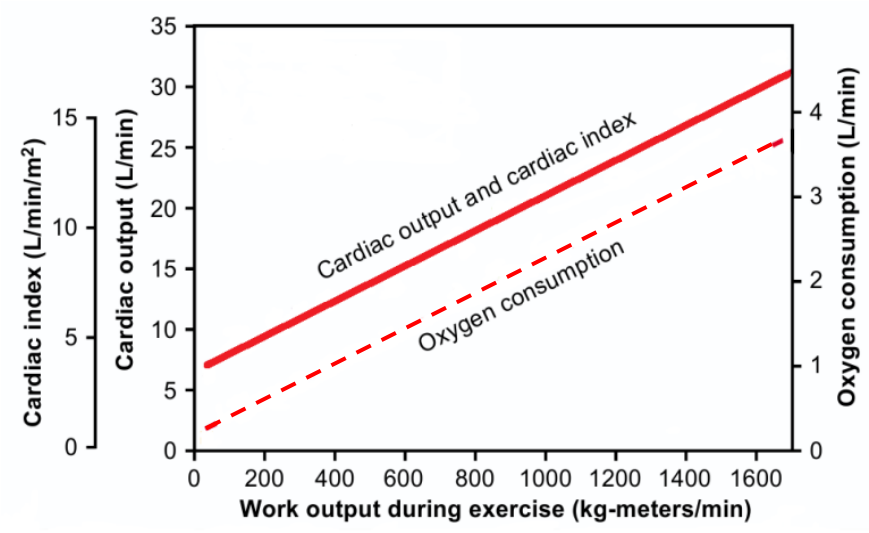
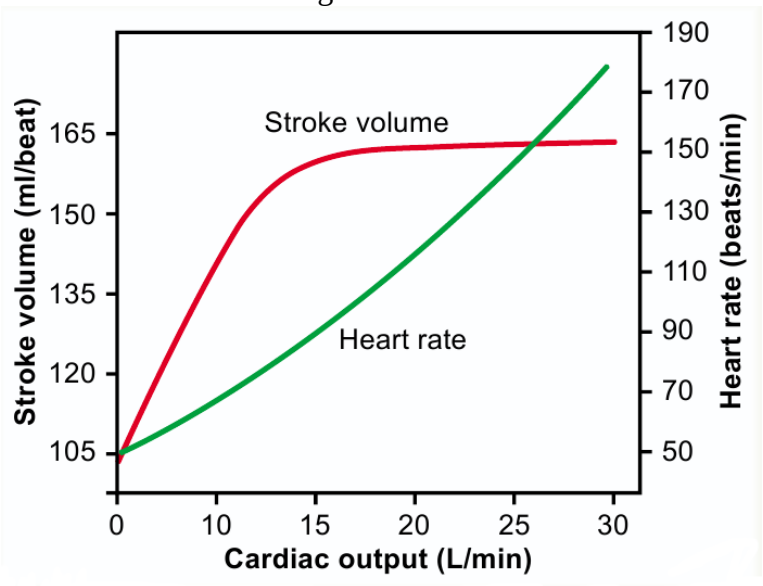


han double the blood flow; this multiplies the great increase in flow already caused by the metabolic vasodilation at least another twofold.

Work output, oxygen consumption, and cardiac output during exercise.

Figure 4 shows the interrelations among work output, oxygen consumption, and cardiac output during exercise. It is not surprising that all these are directly related to one another, as shown by the linear functions, because the muscle work output increases oxygen consumption, and oxygen consumption in turn dilates the muscle blood vessels, thus increasing venous return and cardiac output. Typical cardiac outputs at several levels of exercise are the following:



Thus, the normal untrained person can increase cardiac output a little over fourfold, and the well-trained athlete can increase output about sixfold (ɯndividual marathoners have been clocked at cardiac outputs as great as 35 to 40 L/min, seven to eight times normal resting output).

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Role of stroke volume and heart rate in increasing the cardiac output.

Figure 5 shows the approximate changes in stroke volume and heart rate as the cardiac output increases from its resting level of about 5.5 L/min to 30 L/min in the marathon runner. The stroke volume increases from 105 to 162 milliliters, an increase of about 50 per cent, whereas the heart rate increases from 50 to 185 beats/min, an increase of 270 per cent. Therefore, the heart rate increase accounts by far for a greater proportion of the increase in cardiac output than does the increase in stroke volume during strenuous exercise. The stroke volume normally reaches its maximum by the time the cardiac output has increased only halfway to its maximum. Any further increase in cardiac output must occur by increasing the heart rate.

Body heat in exercise

Almost all the energy released by the body's metabolism of nutrients is eventually converted into body heat. This applies even to the energy that causes muscle contraction for the following reasons: First, the maximal efficiency for conversion of nutrient energy into muscle work, even under the best of conditions, is only 20 to 25 per cent; the remainder of the nutrient energy is converted into heat during the course of the intracellular chemical reactions. Second, almost all the energy that does go into creating muscle work still becomes body heat because all but a small portion of this energy is used for (1) overcoming viscous resistance to the movement of the muscles and joints, (2) overcoming the friction of the blood flowing through the blood vessels, and (3) other, similar effects—all of which convert the muscle contractile energy into heat.
Now, recognizing that the oxygen consumption by the body can increase as much as 20-fold in the well-trained athlete and that the amount of heat liberated in the body is almost exactly proportional to the oxygen consumption, one quickly realizes that tremendous amounts of heat are injected into the internal body tissues when performing endurance athletic events. Next, with a vast rate of heat flow into the body, on a very hot and humid day so that the sweating mechanism cannot eliminate the heat, an intolerable and even lethal condition called heatstroke can easily develop in the athlete.

Thermoregulation and exercise

- Mean body temperature
  $T_{body} = (0.6 \times T_{core}) + (0.4 \times T_{skin})$
  This equation gives the average body temperature at any given time such that:
  - 60% is accounted for by the core
  - 40% is accounted for by the skin

What regulates body temperature?

- Hypothalamus:
  - contains the central coordinating center for temperature regulation
  - receives input from:
    - thermal receptors in the skin provide information
    - temperature of the blood (as it flows by the hypothalamus) provides information
  - anterior hypothalamus – stimulates heat loss
  - posterior hypothalamus – stimulates heat conservation

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Mechanisms of temperature regulation

- When it is hot (need for heat loss):
    - there is vasodilation of subcutaneous blood vessels; more sweating (↑ heat loss)
    - there is decreased muscle activity; decreased secretion of thyroxine and epinephrine (↓ heat production)
- When it is cold (need for heat retention):
    - there is vasoconstriction of skin blood vessels; also curling up to stay warm (↓ heat loss)
    - shivering and increased voluntary muscle activity; increased secretion of thyroxine and epinephrine (↑ heat production)

Heat Loss Mechanisms

- Radiation – emission of electromagnetic heat waves
- Conduction – direct transfer of heat through a liquid, solid, or gas (direct contact)
- Convection – transfer of heat via air currents over surface of skin
- Evaporation – vaporization of water from respiratory passages or surface of skin (2–4 million sweat glands)

Factors affecting heat loss

- Increased ambient temperature (reduces effectiveness of heat loss particularly by radiation, conduction, and convection)
- Increased relative humidity (reduces effectiveness of heat loss by evaporation)
- Decreased wind velocity (reduces both convective and evaporative effectiveness)
- Reduced surface exposed to environment (reduces effectiveness of all heat loss mechanisms)

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