Influence of exercise on selected cardiovascular variables in medical students

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INFLUENCE OF EXERCISE ON SELECTED CARDIOVASCULAR VARIABLES IN MEDICAL STUDENTS

Diploma thesis

Academic year:

2018/2019

Mentor:

Prof. Zoran Valić, MD, PhD

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LIST OF ABBREVIATIONS

ADP – adenosine diphosphate

ANS – autonomic nervous system

ATP – adenosine triphosphate

bpm – beats per minute

CO – cardiac output

CO_2 – carbon dioxide

DAP – diastolic arterial pressure

HR – heart rate

MAP – mean arterial pressure

pCO_2 – partial pressure of carbon dioxide

Q – flow

R – vascular resistance

SAP – systolic arterial pressure

SBP – systolic blood pressure

SNS – sympathetic nervous system

SV – stroke volume

TPR – total peripheral resistance

1. INTRODUCTION

1.1. The Circulatory system

1.1.1. Function

The circulatory system serves to maintain an optimal environment in the tissues of the body in order to ensure appropriate function. This is achieved by transporting nutrients and oxygen to and waste products away from the tissues (1). Furthermore the circulatory system is involved in maintaining homeostasis by participating in temperature regulation, fluid balance and hormone delivery (1).

1.1.2. Division

The circulatory system can be divided into the systemic and the pulmonary circulation. The systemic circulation, also referred to as the greater or peripheral circulation, serves blood to all tissues except the lungs, which are supplied by the pulmonary circulation (2).

1.1.3. Blood vessels

Arteries, capillaries and veins serve as tubes for distribution and collection of blood throughout the body and eventually as the site of metabolite exchange. Each vessel type differs in structure according to their function in the circulation (2).

The aorta, having to accommodate the blood ejected from the left ventricle, is primarily an elastic structure. Arteries have strong vascular walls because their main role is to transport blood to tissue sites under high pressure. The last branches of the arterial system are named arterioles. They represent the site of blood flow control as their thick muscular walls allow them to reduce their lumen (vasoconstriction) or to relax (vasodilation) (2).

Capillaries are characterized by thin fenestrated vessel walls and thus serve as the site of exchange of nutrients, electrolytes, waste products and other substances (2).

The venous circulation begins with the venules, which collect blood from the capillaries and converge into larger veins. Their role is to transport blood back to the heart. As the venous system is a low pressure conduit with vessels composed of thin walls, it additionally functions as a reservoir for extra blood (2).

1.1.4. Heart

The heart is a muscular organ whose task is to pump blood through the circulation. In humans it is composed of two atria and two ventricles. The right atrium receives deoxygenated blood from the superior and inferior vena cava and pumps it into the right ventricle for propulsion into the pulmonary circulation. From the lungs oxygenated blood returns to the left atrium via the pulmonary veins ready to be ejected into the aorta by the left ventricle (3).

1.1.5. Systemic circulation

The systemic circulation is responsible for the transport of oxygenated blood from the heart through the aorta to the rest of the body, and returns deoxygenated blood back to the heart. Pressure generated by heart contraction represents the driving force for blood flow.

Ejection of blood from the heart is intermittent. Nevertheless flow to body tissues must be continuous. This is achieved by distention of the aorta and its branches during systole. During ventricular relaxation elastic recoil of the arterial walls ensure forward propulsion of blood (4).

Influenced by time of the day, level of activity or fluid status fine adjustments regarding pressure, blood flow, heart rate, cardiac output and resistance must be made (5). These functions are discussed in detail in the following sections.

1.2. Cardiovascular Variables

1.2.1. Interdependency of Pressure, Flow and Resistance

Blood flow through a vessel is mainly affected by two factors. The first being the pressure gradient ΔP (P1–P2) between two ends of a blood vessel which describes the force pushing blood through the vessel. The other factor called vascular resistance (R) is the impedance to intravascular flow caused by friction of flowing blood and the vessel wall.
Flow (Q) can be calculated by Ohm's law (2):

$$Q = \frac{\Delta P}{R} \qquad (2)$$

The formula indicates that flow is directly proportional to pressure difference and inversely proportional to the resistance.
Other algebraic forms include:

$$R = \frac{\Delta P}{Q} \qquad (2)$$

$$\Leftrightarrow \quad \Delta P = Q \times R \qquad (2)$$

Considering cardiac output (CO) as the flow of blood from the heart, resistance occurring in the periphery (TPR) and replacing pressure difference with the average blood pressure (MAP) it follows:

$$MAP(\Delta P) = CO(F) \times TPR(R) \qquad (6)$$

1.2.2. Arterial Pressure

Systolic blood pressure (SBP) represents the maximum pressure in the aorta during a ventricular contraction. It usually measures around 120 mmHg (7).

The diastolic blood pressure (DBP) represents the minimum pressure in the aorta when the heart is relaxed. It commonly measures around 80 mmHg (7).

Mean arterial pressure (MAP) represents the average blood pressure during one cardiac cycle. It can be calculated from systolic and diastolic blood pressure as follows:

$$\text{MAP (mmHg)} = [1/3 \times \text{SBP (mmHg)}] + [2/3 \times \text{DBP (mmHg)}] \quad (6)$$

1.2.3. Blood Flow, Heart Rate and Cardiac Output

Blood flow is the amount of blood that passes by a given point at a given time. It is usually measured in liters or milliliters per minute. In the aorta this volume can be referred to as cardiac output because it equals the volume the heart pumps in the aorta each minute (2). Cardiac output is directly proportional to the heart rate (HR), measured in beats per minute and stroke volume (SV) in mililiters.

$$CO=SV \times HR$$

From this formula we can deduct that in order for cardiac output to increase stroke volume or heart rate must increase.

In concordance with a study from Giles N. Cattermole *et al.* cardiac output in 18 to 30 year old adults measures about 5.42 l/min. Normal values for heart rate average about 72 bpm (8).

1.2.4. Total peripheral resistance

Total peripheral resistance (TPR) provides information about elastic and vasoconstrictive properties of the peripheral blood vessels (6). It is a calculated value dependent on cardiac output and mean arterial pressure, measured in mmHg/l/min.

For calculation an analogous form of the before mentioned Ohm's law can be applied:

$$MAP(\Delta P) = CO(Q) \times TPR(R) \qquad (6)$$

$$\Leftrightarrow \quad TPR = \frac{MAP}{CO}$$

This formula suggests that total peripheral resistance increases if mean arterial pressure increases or cardiac output decreases.

1.3. The Nervous system and the Circulation

Blood flow in the body is mainly regulated by local tissue control mechanisms. Regarding the nervous regulation of the circulation it is primarily controlled by the autonomic nervous system (ANS) (2).

The ANS is important for maintaining physiological homeostasis and management of acute stress (9).

1.3.1. The Effect of the Sympathetic Nervous System

The primary neurotransmitter of the sympathetic nervous system (SNS) at postganglionic nerve endings in the majority of tissues is norepinephrine.

The response of the target tissues depends on the receptor being activated. The heart is mainly supplied with beta receptors. Activation of these receptors causes an increase in heart rate, atrioventricular node conduction velocity and force of contraction (10,11).

Alpha receptors however are widely located on vascular smooth muscle where sympathetic nerves directly innervating the smooth muscle layer of the vessel wall cause marked vasoconstriction leading to an increase in resistance and blood pressure (9, 12).

Norepinephrine mainly causes vasoconstriction by activation of alpha receptors but activates beta receptors to a lesser extent as well (2).

Epinephrine excites both adrenoceptors in a dose-dependent fashion. At low doses epinephrine excites vasodilating beta receptors but in high doses also excites the vasoconstricting alpha receptors (13).

1.3.2. The Adrenal Medulla

Sympathetic activation causes the adrenal gland to release norepinephrine and epinephrine. The continued process of methylation of norepinephrine in the adrenal medulla leads to a higher amount of epinephrine compared to norepinephrine. Epinephrine constitutes 80% and norepinephrine 20% of the secretion (2, 13).

In contrast to direct stimulation of target tissues by sympathetic nerves, the effect of these circulating hormones in the blood stream lasts 5 to 10 times longer because removal from the circulation takes more time (2).

1.3.3. The Effect of the Parasympathetic Nervous System

The largest nerve and main parasympathetic component in the body is the vagus nerve. It uses acetylcholine as its primary neurotransmitter. The right and left vagal nerve innervate the sinoatrial node and the atrioventricular node, respectively. Cholinergic stimulation to the heart leads to slowing of the heart rate and a decrease of conduction velocity (9).

Cholinergic nerves innervate the muscular as well as the endothelial layers of the vessel wall (12). Stimulation of M3 receptors in the vascular endothelium causes release of NO leading to vasodilation. In the tunica media acetylcholine causes contraction of smooth muscle via M2 and M3 receptors and thus vasoconstriction if NO production is impaired (12).

1.4. Exercise

Exercise leads to increased oxidative metabolic activity of the contracting muscles which hence demand for increased oxygen delivery by means of increased blood flow.
The response to exercise is mediated by different mechanisms (13).

At first the mechanical mechanism comes into play. When exercise is initiated cardiac output increases because increased venous return is provided by the action of the skeletal muscle pump.(13) As metabolites accumulate in the exercising muscle metabolic vasodilation occurs. This causes a fall in systemic vascular resistance (13). Considering Ohm's law (MAP=CO x TPR) (6) we would expect a drop in pressure. However this is limited by the increase in cardiac output (13).

At the same moment signals from the hypothalamus mediate increased sympathetic tone and decreased parasympathetic tone (13). In the heart sympathetic mass discharge and parasympatholysis lead to an increase in heart rate and inotropy augmented by the release from vagal stimulation (2). This further increases cardiac output when recalling that CO=SV x HR (2).

In the peripheral circulation sympathetic activity causes vasoconstriction in the nonexercising muscles as well as in the gastrointestinal tract and kidneys. Consequently the fall in total peripheral resistance is thwarted and flow to the active muscle is increased (13).

1.4.1. Static Exercise vs Dynamic Exercise

Muscular activity can be distinguished into two types. Static or isometric physical activity causes a change in muscle tension but a small change in muscle length. Dynamic or isotonic exercise leads to a change in length but small change in tension (14).

Riding the bicycle, swimming and running are primarily dynamic activities, whereas weight lifting and bearing weight against a fixed object are primarily static exercises (14).

2. OBJECTIVES

AIM:

The aim of this study is to investigate and compare the effect of static and dynamic exercise on selected cardiovascular variables such as mean arterial pressure, systolic pressure, diastolic pressure, heart rate, cardiac output and total peripheral resistance in healthy volunteers.

HYPOTHESIS:

We expect that static exercise leads to an increase in mean arterial pressure, systolic arterial pressure, diastolic arterial pressure and total peripheral resistance.

In dynamic exercise we expect an increase in mean arterial pressure, systolic arterial pressure, heart rate and cardiac output, but a drop of total peripheral resistance.

3. MATERIALS AND METHODS

3.1. Ethics

The Research Ethics Committee of the University of Split School of Medicine approved the study.

The experiments with students from the University of Split School of Medicine were conducted in Split in April 2019 on 6 students and on 49 students between 2011 and 2018. All experimental procedures were thoroughly explained to all participants with the possibility to withdraw at any point.

3.2. Subjects

Healthy students aged in their 20s were recruited. Regarding participants, 49 students were students in their second year of medical studies at the University of Split School of Medicine, 6 students were in their sixth year at the time of the experiment.

3.3. Experimental procedures

All experiments were performed at the University of Split School of Medicine. Upon arrival subjects were obligated to rest for 5 minutes in order to reach physiological baseline levels.

A photoplethysmograph cuff was placed around the middle finger of the non-dominant hand in order to measure heart rate and arterial pressure continously and noninvasively (Finometer, Finapress Medical Systems, Arnhem, Netherlands).

Arterial pressure and heart rate measurements were digitalized and stored on a computer (Apple eMac PC) equipped with a Power Lab16S data acquisition system (ADInstruments) at a sampling rate of 100Hz.

A dynamometer was used to simulate static exercise. For each participant isometric maximal grip strength was determined by squeezing the device to the individual's maximum. The subject was then instructed to squeeze the dynamometer at 30 percent of the previously determined maximum. Three minutes of exercise were followed by one minute of rest.

Thereupon dynamic exercise was to be performed. An ergometer was used to conduct dynamic exercise. The subject was instructed to ride the ergometer at 100Watts for a total of three minutes, followed by one minute of rest. While exercising the arm wearing the pressure cuff had to be held in a constant position to eliminate artefacts from movement.

3.4. Data analysis

Data were obtained from 6 exercising students in April 2019. Other data were obtained from 49 students exercising between 2011 and 2018.

Average value, standard deviation and standard error of the mean were calculated for each minute of exercise of static as well as dynamic exercise. Additionally relative changes for mean arterial pressure, systolic arterial pressure, diastolic arterial pressure, heart rate, cardiac output and total peripheral resistance from the first to the third minute were calculated in percentages.

3.5. Statistical analysis

The results presented in text and figures are shown as the mean value and standard error of the mean. One-way ANOVA with repeated measures on ranks was conducted followed by a *post hoc* Tukey test using SigmaPlot 12.5 software (Systat software, San Jose, CA, USA). Statistical significance value was set at $P<0.05$.

For statistics of relative changes paired T-tests were conducted using SigmaPlot 12.5 software (Systat software, San Jose, CA, USA).

4. RESULTS

Figure 1 illustrates the typical response regarding pulse pressure, systolic arterial pressure, diastolic arterial pressure, heart rate, cardiac output and resistance of an individual during exercise. On the left half of the graph responses to static exercise are shown. On the right half responses to dynamic exercises can be seen.

Start denotes the onset of exercise. Values before the start show resting values (baseline to start). After 3 minutes of exercise follows one minute of rest (3 min to 4 min) after which the recording was stopped.

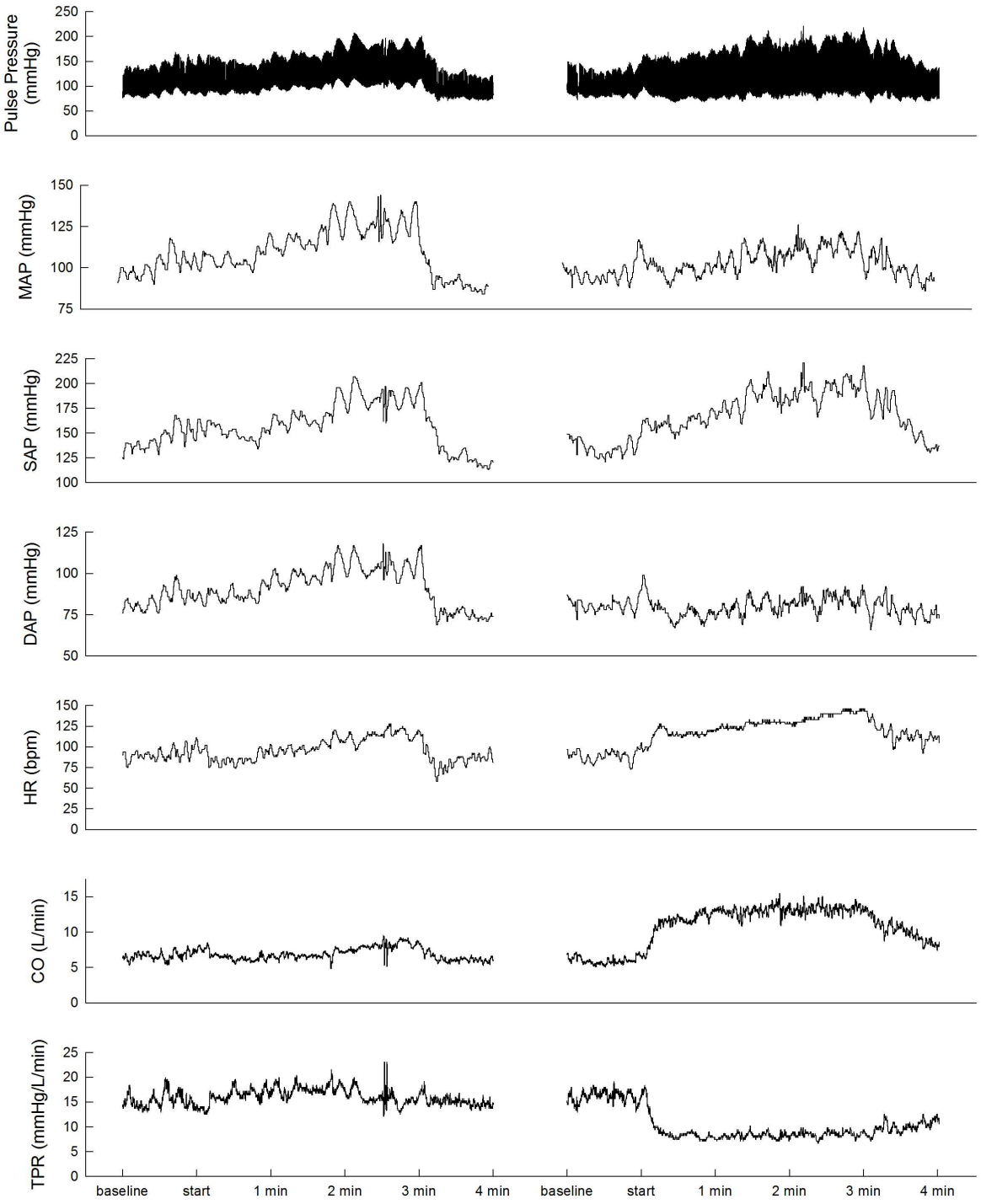

Figure 1. Raw data of pulse pressure, MAP – mean arterial pressure, SAP – systolic arterial pressure, DAP – diastolic arterial pressure, HR – heart rate, CO – cardiac output, TPR – total peripheral resistance during static and dynamic exercise

Mean arterial pressure increased during static and dynamic exercise. In static exercise the increase was significant in the first, second and third minute. In dynamic exercise a significant increase was seen in the second and third minute. There was no difference from baseline in the recovery period (Figure 2).

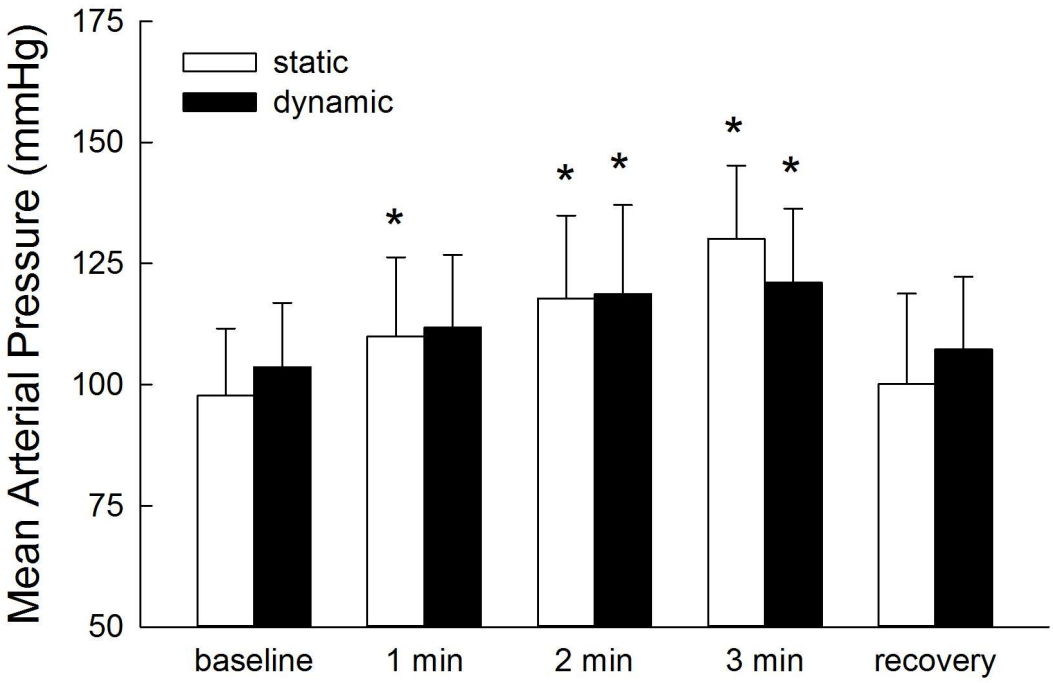

Figure 2. Mean Arterial Pressure measurement during static and dynamic exercise
Data are presented as mean ± standard error of the mean
* Tukey-test, $P<0.05$

Systolic arterial pressure increased significantly during the first, second and third minute of exercise in both types of physical activity. A significant difference from baseline was also noted during the recovery period from dynamic exercise but not during recovery from static exercise (Figure 3).

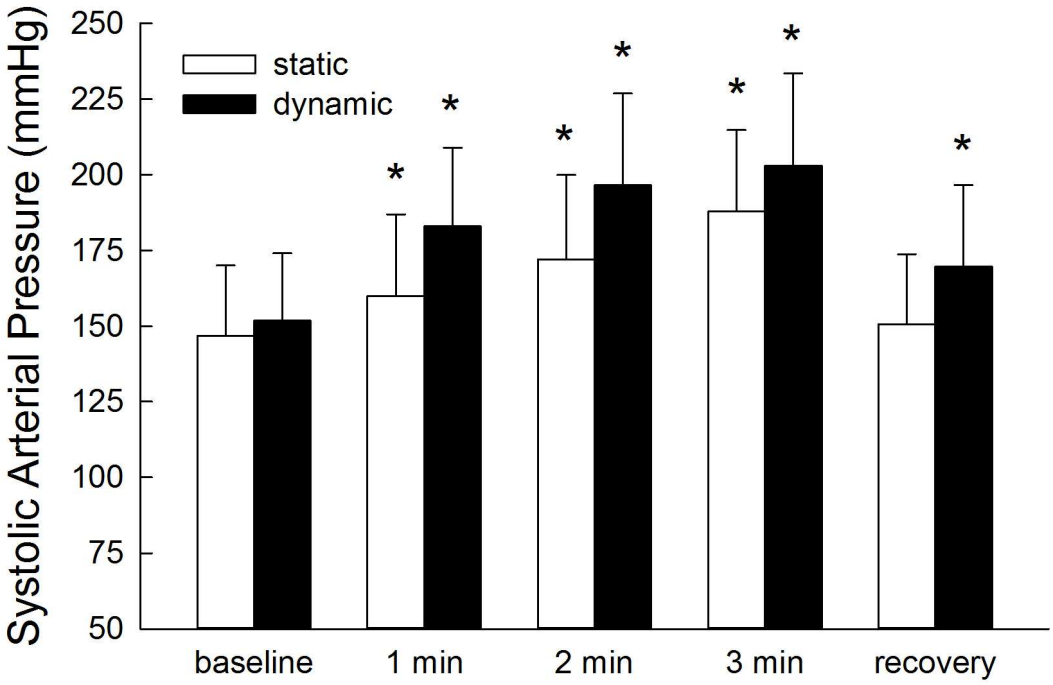

Figure 3. Systolic arterial pressure measurement during static and dynamic exercise
Data are presented as mean ± standard error of the mean
* Tukey-test, $P<0.05$

Diastolic arterial pressure during static exercise increased significantly in the first, second and third minute. In dynamic exercise a significant increase from baseline is noted in the second and third minute. There was no difference from baseline in the recovery period in both types of exercise (Figure 4).

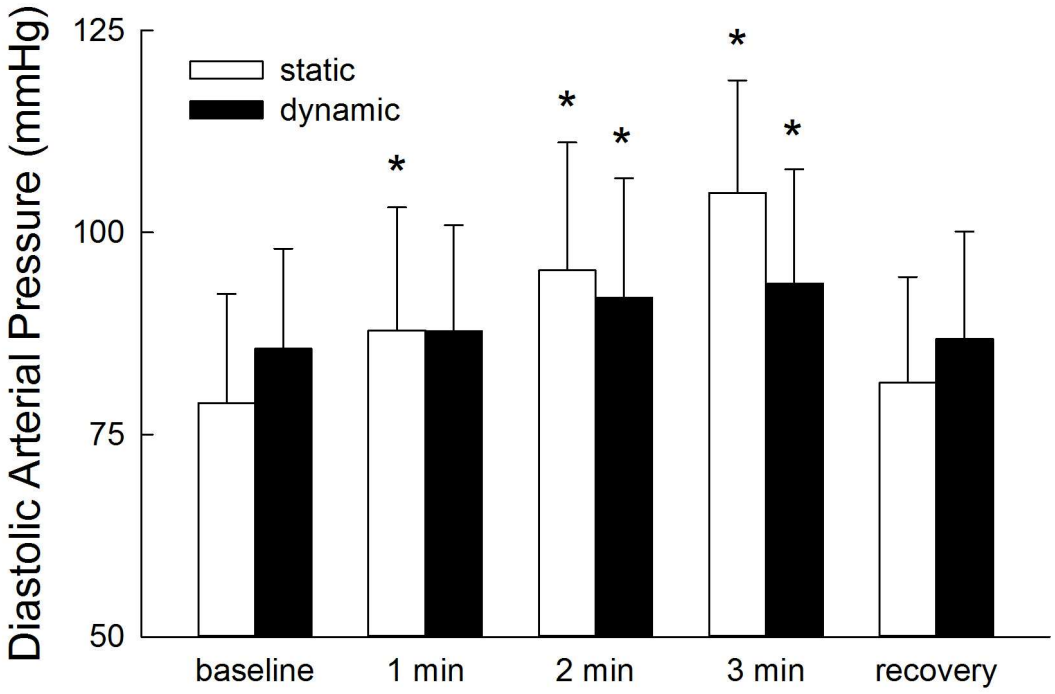

Figure 4. Diastolic arterial pressure measurement during static and dynamic exercise
Data are presented as mean ± standard error of the mean
* Tukey-test, $P<0.05$

Heart rate increased significantly in the first, second and third minute in both types of exercise. During the recovery period a decrease in heart rate was observed (Figure 5).

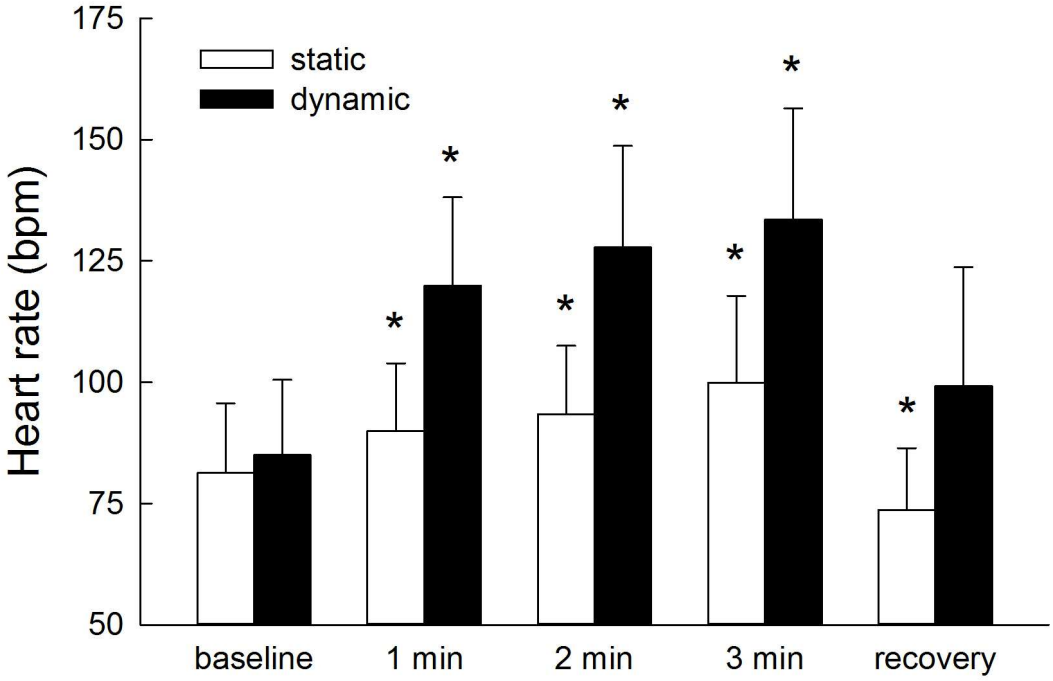

Figure 5. Systolic arterial pressure measurement during static and dynamic exercise
Data are presented as mean ± standard error of the mean
* Tukey-test, $P<0.05$

Cardiac output increased significantly compared to baseline in the first, second and third minute in static as well as dynamic physical activity. A significant difference was also shown in the recovery period during static exercise but not during recovery from dynamic exercise (Figure 6).

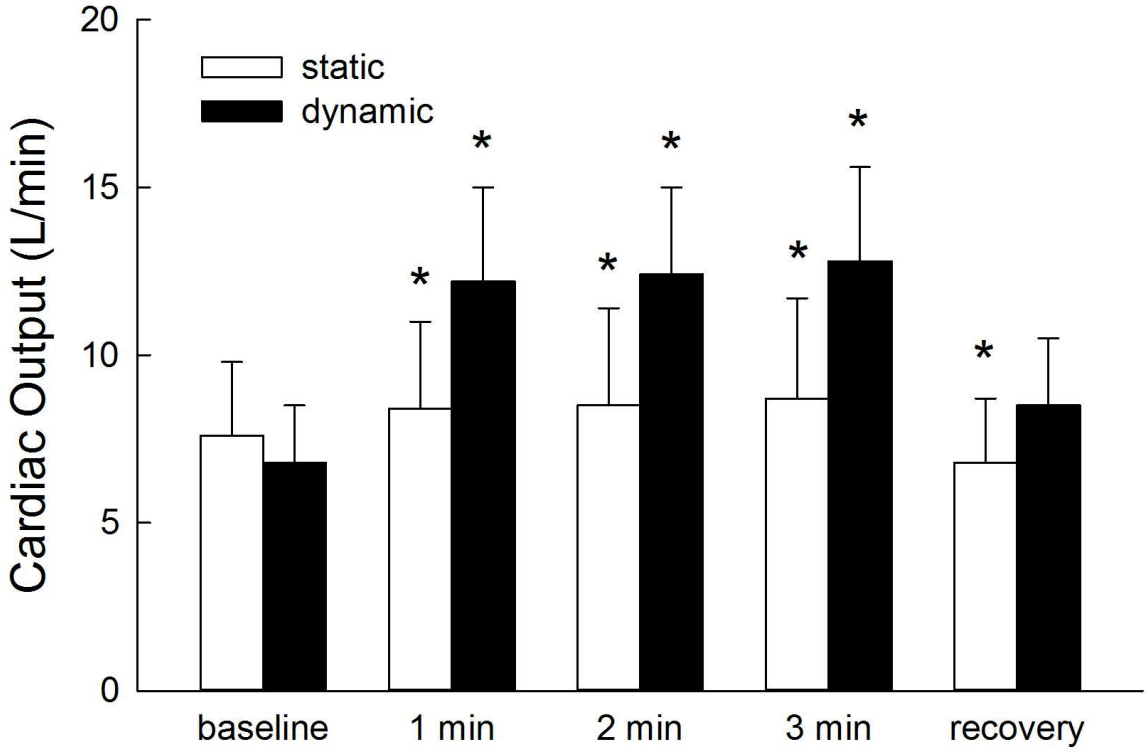

Figure 6. Cardiac output measurement during static and dynamic exercise

Data are presented as mean ± standard error of the mean

* Tukey-test, $P<0.05$

Total peripheral resistance increased significantly compared to baseline in the second and third minute during static exercise, as well as during the recovery period. During dynamic exercise a significant decrease of total peripheral resistance is shown in the first second and third minute, as well as during the recovery period (Figure 7).

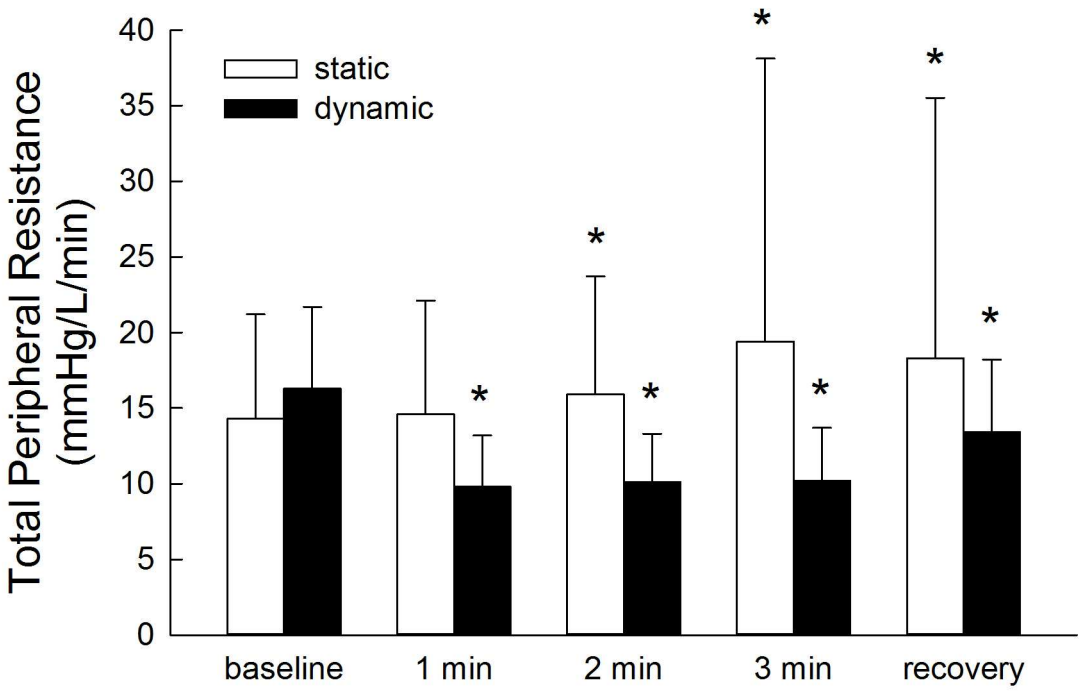

Figure 7. Total peripheral resistance measurement during static and dynamic exercise
Data are presented as mean±standard error of the mean
* Tukey-test, $P<0.05$

The relative change in pressure between baseline and the third minute of exercise is illustrated in Figure 8. Mean arterial pressure increased by 35% in static exercise. In dynamic exercise a significant increase of 18% was seen. Increase was statistically greater ($p<0.05$) in static exercise.

Systolic arterial pressure increased in both types of exercise by 29 and 35% respectively (Figure 8).

Diastolic arterial pressure increased by 36% during static exercise. In dynamic physical activity an increase of 10% was noted. Increase was statistically greater in static exercise ($p<0.05$) (Figure 8).

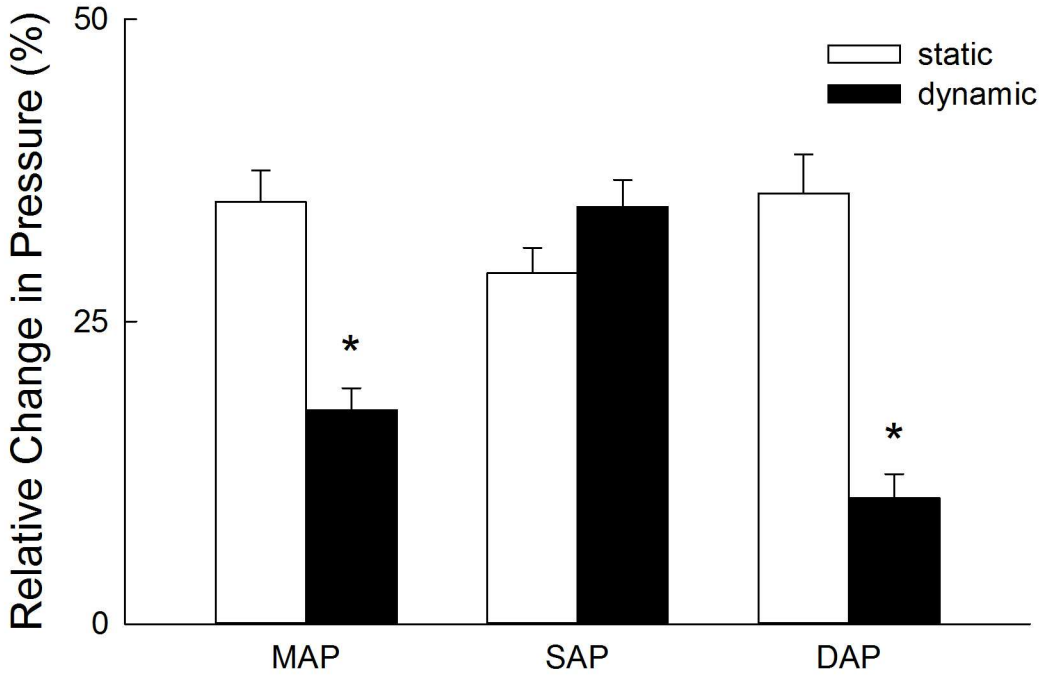

Figure 8. Relative change in pressure (%) MAP – mean arterial pressure, SAP – systolic arterial pressure, DAP – diastolic arterial pressure

Data are presented as mean ± standard error of the mean

* Paired T-Test, $P<0.05$

Heart rate increased in both types of exercises. In static exercise an increase of 25% was seen, in dynamic exercise heart rate increased by 60%. Increase was statistically greater in dynamic exercise ($p<0.05$) (Figure 9).

Measurements of cardiac output showed an increase of 16% during static exercise and an increase during dynamic physical activity by 94%. Increase was statistically greater in dynamic exercise ($p<0.05$) (Figure 9).

Total peripheral resistance increased by 42 percent during isometric exercise, however it decreased during dynamic exercise (-35 percent). Decrease was statistically significant compared to relative change during static exercise ($p<0.05$) (Figure 9).

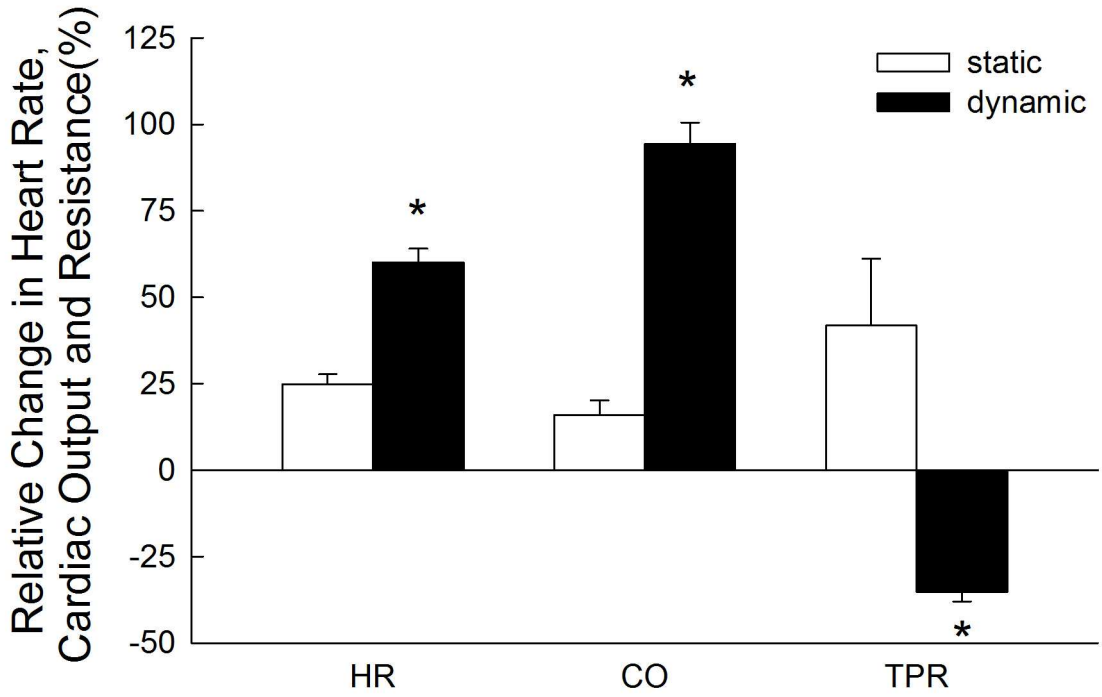

Figure 9. Relative change in heart rate, cardiac output and resistance, HR – heart rate, CO – cardiac output, TPR – total peripheral resistance

Data are presented as mean±standard deviation

* Paired T-Test, $P<0.05$

5. DISCUSSION

In this study we investigated the effect of static and dynamic exercise on the cardiovascular system. Measurements included mean arterial pressure, systolic and diastolic arterial pressure, heart rate, cardiac output and total peripheral resistance.

It is well known that the effect of isometric versus isotonic exercise is significantly different (15).

For simulation of static exercise we used a handgrip dynamometer. We observed a significant increase in mean arterial pressure, systolic arterial pressure as well as diastolic arterial pressure. Heart rate, cardiac output and total peripheral resistance also increased significantly during the exercise. Explanation of these responses to static exercise lies in the physiology of the cardiovascular system and the effect of the autonomic nervous system.
Increased sympathetic activity leads to vasoconstriction in the nonexercising muscles in order to provide adequate blood flow to the active muscles. Stroke volume remains fairly unchanged in static exercise but in combination with a slight increase in heart rate from adrenergic excitation and parasympatholysis the net result is a modest increase in cardiac output (15).

The increase in total peripheral resistance from widespread vasonconstriction and increased cardiac output results in an increase of pulse pressure, systolic and diastolic pressure.

Significant difference from baseline of heart rate, cardiac output and resistance may have ocurred because not enough time was given to allow these parameters to return back to resting values.

For simulation of dynamic exercise we used a cyclic ergometer, the stationary equivalent of a bicycle. We observed a significant increase in mean arterial pressure, systolic arterial pressure and diastolic arterial pressure. Heart rate and cardiac output also increased significantly. Total peripheral resistance decreased significantly. Again these responses to exercise can be explained looking at the physiology and the impact of the autonomic nervous system.

Dynamic exercise greatly increases oxygen consumption. Significant increase in heart rate as well as increased stroke volume increase cardiac output and thus enable increased flow to the active muscles (16). Once again these changes result from sympathetic excitation and decreased parasympathetic tone.

Increased sympathetic output leads to vasoconstriction in order to redirect cardiac output away from inactive muscles and splanchnic tissues to the exercising muscles (17). In the active muscle the vasoconstrictive effect from excitation of adrenergic receptors is overcome by metabolic vasodilation. Classic mediators originating from muscles include increased pCO_2, lactate, potassium and adenosine (18). Skeletal muscle activity leads to increased glycolysis and lipolysis accounting for an increased turnover of the citrate cycle which yields augmented production of CO_2. Lactic acid is generated from anaerobic metabolism in the muscle. Potassium ions are released from muscle cells for any action potenial. When the frequency of action potentials is high, reuptake by the $Na\text{-}K^+$-ATPase is overwrought and interstitial K^+ concentrations increase (18). Increased adenosine triphosphate (ATP) breakdown leads to increased concentration of adenosine diphosphate (ADP) during the resynthesis (18).

Another mechanism known as "functional sympatholysis" is the capability of skeletal muscle to blunt sympathetic vasoconstriction through specific signalling (17).
Thus during dynamic exercise contracting skeletal muscle is able to increase blood flow by almost 100-fold (17). In our recording this was observed by the tremendous drop of total peripheral resistance.

Static exercise predominantly causes a pressure load on the heart (14). In our study we concluded this on the basis of the observation that change in mean arterial pressure and diastolic pressure was significantly greater in static exercise compared to relative change of mean arterial pressure and diastolic arterial pressure during dynamic physical activity.
On the other hand dynamic exercise predominantly causes a volume load on the heart (14). We inferred this because the relative increase in heart rate and cardiac output was statistically greater in dynamic exercise compared to static exercise.

Limitations in our study included that we allowed one minute only for the recovery period. Concerning some measurements this lead to statistically significant differences of baseline values compared to values in the recovery period. Furthermore the order of the exercises was not randomized and static exercise always preceded dynamic exercise. Moreover we did not determine a maximum value for each individual before performance of dynamic exercise.

6. CONCLUSION

1. Static exercise leads to an increase in mean arterial pressure, systolic arterial pressure, diastolic arterial pressure, heart rate, cardiac output and total peripheral resistance.
2. Dynamic exercise leads to an increase in mean arterial pressure, systolic arterial pressure, diastolic arterial pressure, heart rate, cardiac output and a decrease in total peripheral resistance.
3. Relative change in mean arterial pressure and diastolic arterial pressure is greater during static exercise suggesting a pressure load on the heart.
4. Relative change in heart rate and cardiac output is greater during dynamic exercise suggesting a volume load on the heart.
5. Relative change in total peripheral resistance during dynamic exercise is greater compared to change during static exercise.

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8. SUMMARY

Objectives: The aim of this study is to investigate and compare the effect of static and dynamic exercise on selected cardiovascular variables such as mean arterial pressure, systolic pressure, diastolic pressure, heart rate, cardiac output and total peripheral resistance

Materials and Methods: 55 medical students in their twenties performed static exercise using a handgrip dynamometer and dynamic exercise on an ergometer. Arterial pressure and heart were measured noninvasively and recorded with a computed device for subsequent data analysis.

Results: We observed a significant increase in mean arterial pressure, systolic arterial pressure as well as diastolic arterial pressure during isometric exercise. Heart rate, cardiac output and total peripheral resistance also increased significantly.
During dynamic exercise we observed a significant increase in mean arterial pressure, systolic arterial pressure and diastolic arterial pressure. Heart rate and cardiac output also increased significantly while total peripheral resistance decreased.
Relative changes of mean arterial pressure and diastolic pressure were greater in static exercise. Relative changes of heart rate, cardiac output and total peripheral resistance were greater in dynamic exercise.

Conclusion: Exercise causes an change in certain cardiovascular variables due to activation of the autonomic nervous system. Static exercise induces a pressure load on the heart while dynamic exercise causes a volume load.

1. CROATIAN SUMMARY

Naslov: UTJECAJ TJELOVJEŽBE NA ODABRANE KARDIOVASKULARNE PARAMETRE U STUDENATA MEDICINE

Cilj: Svrha ove studije je istražiti i usporediti učinke statičke i dinamičke vježbe na odabrane kardiovaskularne parametre kao što su srednji arterijski tlak, sistolički i dijastolički tlak, srčanu frekvenciju, srčani minutni volumen i ukupni periferni otpor.

Materijali i metode: 55 studenata medicine, u njihovim dvadesetim godinama, sudjelovalo je u izvođenju statičke tjelovježbe koristeći ručni dinamometar, te dinamičke tjelovježbe na bicikli ergometru. Arterijski tlak i srčana frekvencija kontinuirano su neinvazivno mjereni i pohranjivani na osobno računalo za naknadnu analizu.

Rezultati: Zamijetili smo statistički značajan porast u vrijednostima srednjeg, sistoličkog i dijastoličkog tlaka tijekom izometričke tjelovježbe. Frekvencija srca, srčani minutni volumen i ukupni periferni otpor također su značajno porasli.
Tijekom izvođenja dinamičke tjelovježbe došlo je do statistički značajnog porasta vrijednosti srednjeg, sistoličkog i dijastoličkog tlaka. Frekvencija srca i srčani minutni volumen također su značajno porasli, dok su se vrijednosti ukupnog perifernog otpora smanjile.
Relativan porast srednjeg i dijastoličkog arterijskog tlaka bio je statistički značajnije izražen u statičkoj tjelovježbi u usporedbi s dinamičkom. Relativne promjene frekvencije srca, srčanog minutnog volumena i ukupnog perifernog otpora bile su značajno veće tijekom dinamičke tjelovježbe.

Zaključak: Tjelovježba uzrokuje promjenu određenih kardiovaskularni parametara prvenstveno aktivacijom autonomnog živčanog sustava. Statička tjelovježba dovodi do pretežito tlačnog opterećenja srca, dok dinamička tjelovježba izaziva pretežito volumno opterećenje.

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