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# Cardiovascular responses to static exercise

by Eino Hietanen, MD<sup>1</sup>

HIETANEN E. Cardiovascular responses to static exercise. *Scand J Work Environ Health* 10 (1984) 397—402. Cardiovascular responses and adaptation to stress are different in dynamic and static exercise. In light static exercise the heart rate and blood pressure increase much more than during dynamic exercise at the same oxygen uptake level. Heavy static exercise is characterized by a failure of the local blood flow to adjust to the oxygen demands of the exercising muscles. Respiratory and circulatory responses are dominated by an incompetence to obtain steady-state conditions, and thus the worktime is short. After the cessation of heavy static exercise a sudden compensatory increase occurs in cardiac output and oxygen uptake. Due to the higher increase in blood pressure, even light static exercise causes much higher strain on the heart than an equivalent amount of dynamic exercise. The heart responds to the increased afterload by increasing contractility and heart rate and thus improves cardiac output. In persons with a poor cardiac reserve a rise in the left ventricular end-diastolic pressure is seen, along with a fall in the stroke work index in response to the increased afterload caused by static exercise. It is possible that a discrepancy exists between work capacity during tasks demanding also isometric muscle work and a dynamic exercise test performance. The decreased cardiac reserve may first appear after the great increase in afterload, even in relatively light static work.

*Key terms:* cardiac adaptation, isometric exercise, static work, stress test.

In many work tasks there is a clear static component combined with dynamic work (14). In order to analyze the effect of individual work tasks on the cardiovascular system, one can divide the tasks by their dynamic and static components although in practical work situations this kind of division may prove to be artificial. The load of the dynamic component on the cardiovascular system can be evaluated through comparison with strain in dynamic exercise stress tests, but evaluating the cardiovascular strain of the static work component on the basis of laboratory stress tests is more difficult. Yet the testing of the cardiovascular strain due to static exercise is important for the prediction and prevention of excessive cardiac load on persons with lowered cardiac reserve in uncontrolled work conditions.

Although in both static and dynamic exercise the muscles transform chemical energy into mechanical energy, there are differences in these two forms. Common to both types of exercise is the loss of part of the energy as heat and the production of exercise force. In practice both isotonic and isometric components are involved in both dynamic and static exercise, but to different degrees, so that isotonic contraction dominates in the former and isometric contraction is predominant in the latter. The energy transformation in muscle contraction can be described by the equation  $E = A + kW$ , where  $E$  corresponds to the total energy transformed by the con-

traction,  $A$  is the energy connected with the production of work, and  $W$  is the energy needed to perform external work. In static exercise, as no external work is performed (isometric contraction),  $W$  is zero (2); in addition, at a given production of force, static exercise would need less energy. When oxygen uptake has been measured as an index of energy consumption, the same amount of tension time, which represents force, has been produced at much less energy in rhythmic isometric exercise than in dynamic exercise. However, direct comparisons are somewhat unjustified as rhythmic isometric exercise does not represent typical static exercise, which is usually long-lasting muscle contraction without relaxation intermissions.

During the static contraction intramuscular pressure increases due to the swelling and stiffening of the active fibers within their connective tissue sheaths, and thus a slight shortening of the muscle fibers takes place, even in static exercise. The increased pressure is transferred to the intramuscular blood vessels. It compresses them, squeezes blood out of the veins, and hinders blood from entering the intramuscular arteries in a way that may totally block the blood flow during the contraction period of maximal static exercise. The highest pressure is obtained when a maximum of muscle fibers is active. The length of time that static contraction can be maintained is dependent on the blood flow to the muscle, and this blood flow is dependent on the maximal voluntary contraction. Under a certain level of the maximal voluntary contraction blood flow is not hindered, and consequently then static exercise can be maintained almost indefinitely. The critical value which determines that static contraction can be maintained

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“indefinitely” is dependent on the type of muscle and the anatomical structure and placement of the muscle, all of which influence intramuscular pressure and blood flow. The muscle fiber type is important because a muscle with a large proportion of fast-twitch fibers (glycolytic) can tolerate less static contraction than muscles with a large proportion of oxidative slow-twitch fibers (2).

A physiological test to increase arterial pressure is the hand grip test. Although this test does not strain a large group of muscles and cannot be used to assess general muscle performance, it is useful in testing the cardiac response to increased load. The test performance is such that a person is first asked to squeeze a grip dynamometer at maximal instantaneous force, which is noted as maximal voluntary contraction. Then the patient is asked to maintain a fixed percentage, eg, 25 %, of this level for a short period of time. An electrocardiogram and heart rate and blood pressure are recorded. Further use of this test is apparent when one studies cardiovascular responses invasively or noninvasively.

A load corresponding to 10 % of the maximal voluntary contraction in a hand grip test yields a relatively moderate increase in oxygen uptake and minute ventilation (2). When oxygen uptake or minute ventilation in static exercise is compared to the respective values of dynamic exercise, a similar response is found (table 1), despite different mechanical conditions in the muscles or differences in the subjective feeling of effort (2).

### **Cardiovascular and related respiratory responses**

#### *Light static exercise*

Heart rate increases immediately after the beginning of light static exercise, and this increase is higher than expected from the increase in oxygen uptake. In addition the systolic and diastolic blood pressures increase immediately in the beginning of the exercise and increase slowly during the work period. When these data are compared to those of the corresponding responses to equal light dynamic exercise, it is found that the heart rate is somewhat higher and the blood pressure clearly higher during static exercise (2). The cardiovascular response differences between static and dynamic exercise are due to local, chemical, thermal, or mechanical factors or they may be neural in origin (24). Biochemical differences are to be expected due to the diminished local blood flow, which leads to anaerobic metabolism despite adequate oxygen supply to the whole body. This phenomenon may cause differences in the neurogenic regulation of cardiovascular responses, which do not reach steady state even in relatively light static exercise.

The signals from the mechanoreceptors in muscles and tendons are different in static and dynamic exer-

cise because of the much higher muscle tension in the former when the strength of the two is the same. The mechanoreceptors are activated immediately after the beginning of exercise, and this occurrence might explain the rapid increase in heart rate. Recruitment of new motor units to maintain muscle tension expands the excitation of the central nervous system also to the cardiovascular centers. Thus the voluntary activity increases the excitatory state of the central nervous system and results in a possible increase in sympathetic and a possible decrease in parasympathetic outflow, which explains the heart rate and blood pressure responses. Although this phenomenon applies to both static and dynamic exercise, the higher voluntary effort of the former might explain the stronger responses to it. The occlusion of the exercising muscles at the beginning or end of the exercise does not change the heart rate response even if it might prevent the action of humoral factors or muscle metabolites (3). The slow increase in heart rate during the progress of static exercise might be due to chemoreceptor signals from the ischemic muscles as this response can be reproduced in dynamic exercise when the blood flow to the muscles is blocked. As muscle fatigueness demands increasing voluntary effort to produce a certain force, the increasing effort might also contribute to the increasing stimulation of the central nervous system. As a result of the effect on the central nervous system vagal stimulation decreases and sympathetic stimulation increases and yields an increased heart rate and blood pressure. The number of afferent nerve impulses affecting autonomic centers seems to be the same from the large or small muscles and is determined by the level of exercise, as the number of muscle spindles (mechanoreceptors) is related to the number of motor nerves (about 10 % of the number of myelinated nerve fibers) and the number of motor nerves is relatively less in big than in small muscles. In addition to the neurogenic explanation also hemodynamic adaptation might partially explain the response. When large muscle groups are involved in static exercise, peripheral resistance also increases, due to the increased intramuscular pressure, and is followed by an increase in blood pressure.

#### *Heavy static exercise*

Cardiovascular adaptation is even farther from steady state in heavy than in light static exercise. Typical to heavy static exercise is the failure of the local blood flow to adjust to the oxygen demands of the exercising muscles, which must work totally under ischemic conditions, and thus energy demands must be met by the anaerobic metabolism. The work-time in heavy static exercise becomes short due to muscle fatigue. Exercise corresponding to about 30 % of the maximal voluntary contraction is already so demanding that it can be performed only a few minutes at a time. Oxygen uptake increases rather

moderately when one considers the heavy load in this kind of exercise (table 1). Pulmonary ventilation increases also only in ratio to the oxygen uptake, and arteriovenous oxygen difference is practically unchanged during heavy static exercise. Also the respiratory quotient remains unchanged (2). After the exercise period, during the first few minutes of recovery, a rebound effect is seen in the respiratory parameters. Oxygen uptake, minute ventilation, the arteriovenous oxygen difference, and the respiratory quotient all increase abruptly but show a decrease after a few minutes (2). The response is equal to that seen during and after ischemic exercise and can be explained by the blocked circulation to the exercising muscles, which is followed during the recovery period by hyperemia.

Typical to the circulatory adaptation is also that steady state is not reached during the period of static exercise. The cardiac output increases continuously to the end of the exercise period and thereafter shows an abrupt increase typical to the postischemic phenomenon. Heart rate, however, increases abruptly immediately after the beginning of the exercise and then stays at a relatively steady-state level during the exercise period. It begins to decrease immediately after the cessation of the exercise without any rebound effect. Blood pressure does not show any rebound effect either. Both the systolic and diastolic blood pressures increase continuously during heavy static exercise to relatively high levels as compared to the moderate increase in oxygen uptake. After the exercise has ceased, blood pressure falls as after the cessation of dynamic exercise.

The main differences between heavy static exercise and a short burst of equal dynamic exercise are in the time courses of oxygen uptake, minute ventilation, and cardiac output, which all show a rebound effect after the cessation of the exercise. This phenomenon is mainly due to anaerobiosis during exercise and postischemic hyperemia. The moderate increase in oxygen uptake after the exercise is probably due to the rather low oxygen demand during the exercise. At the onset of static exercise there is no rapid neurogenic increase in minute ventilation, in contrast to the rapid neurogenic increase in heart rate.

In dynamic exercise the increased cardiac output is directed to the working muscles with a following increase in arteriovenous oxygen difference. As the increased cardiac output cannot be directed through the exercising muscles during static exercise, it goes through the nonexercising parts of the body, the result being an unchanged arteriovenous oxygen difference. In heavy static exercise there is an immediate increase in the stroke volume, possibly due to the venous blood being squeezed from the muscles at the beginning of the exercise, a phenomenon which increases the venous return to the heart. As the heart rate remains fairly constant after the initial increase during the exercise but stroke volume increases con-

**Table 1.** Cardiovascular and related respiratory responses to static and dynamic exercise (2, 4). (— = no change, ± = no or slight change, + = small change, ++ = intermediate change, +++ = great change)

Response	Light exercise		Heavy exercise	
	Static	Dynamic	Static	Dynamic
Oxygen uptake	+	+	+	+++
Minute ventilation	+	+	+	+++
Arteriovenous oxygen difference	—	+	—	+++
Heart rate	+	+ <sup>a</sup>	+	+++
Cardiac output	+	+ <sup>a</sup>	+	+++
Blood pressure				
Systolic	+	+ <sup>a</sup>	++	+++
Diastolic	+	+ <sup>a</sup>	+++	±

<sup>a</sup> Change greater than that in static exercise of the same strength.

tinuously, the increase in cardiac output must be due to the increased stroke volume.

The mean arterial pressure (MAP) response can be regarded as a function of cardiac output (Q) and total peripheral resistance (TPR) as follows:  $MAP = k \times Q \times TPR$ , where k is a constant and Q equals heart rate (HR) times stroke volume (SV); thus  $MAP = k \times HR \times SV \times TPR$ . The increase in heart rate can thus explain some of the increase in mean arterial pressure. Another factor is total peripheral resistance, which may be higher during static exercise than during a corresponding amount of dynamic exercise. During heavy static exercise the peripheral vasodilatation of the working muscles does not decrease the total peripheral resistance because of the cut-off of circulation to the exercising muscles, while simultaneously the increased mechanical pressure increases the systemic resistance to some extent. Furthermore, because of the anaerobiosis that occurs during heavy static exercise, the metabolic effects cause sympathetic vasoconstriction in the nonexercising parts of the body. The mean arterial pressure is thus controlled during heavy static exercise by different factors, including mechanical factors related to muscle mass and absolute force, some factors related to the number of motor and sensory units in the active muscles, chemoreflexes and relative voluntary effort, and the excitatory state of the autonomic nervous centers. [All these factors control total peripheral resistance.] During static strain equal to 40 % of the maximal voluntary contraction the arterial pressure response has been found to be related to the mass of muscles involved in that the larger the muscle mass the stronger the response (20). The central control mechanism of arterial blood pressure response to static muscular contraction is related to the central activity for the recruitment of motor units, and the peripheral control mechanism is mediated by muscle afferents excited by metabolic changes in the contracting skeletal muscle. Probably both static and dynamic exercise activates corticomedullary reflexes with links between the motor cortex and the cardiovascular central control center and reflexes origi-

nating in skeletal muscle afferents (8, 19). In both types of exercise the reflex-induced tachycardia and consequent increase in cardiac output may produce a significant increase in mean arterial pressure during exercise involving small muscles (static), when vascular conditions in the active muscles have only minor effects on the vascular conductance. The metabolically induced vasodilatation that occurs in the large muscle groups during dynamic exercise increases the systemic conductance markedly, and thus the pressor response is lower.

Rhythmic, intermittent muscle activity causes only slight changes in mean arterial blood pressure (10), while tetanic or sustained contractions are accompanied by considerable pressor responses disproportionate to the increase in oxygen consumption (16).

The adaptation of the cardiovascular system to heavy or even moderate static exercise seems inadequate when compared to the adaptation that takes place during dynamic exercise, and this difference should be kept in mind when the stress of static exercise on the cardiovascular system is being evaluated.

There are three typical features of the cardiovascular response to isometric exercise, ie, (i) the conspicuous rise in systemic blood pressure, (ii) the reflex nature of the response, and (iii) the dependence of the response on the conditions of blood flow through the active muscles. The pressure response results from the combination of increased cardiac output and vasoconstriction in nonactive muscles (6, 16). Rhythmic static exercise leads to a considerable increase also in the stroke volume, but constant static exercise does not (1). A beta-blocking agent (propranolol, 80 mg, single dose) has been found to cause a reduction in heart rate response to hand grip, a decrease in stroke volume during activity, and a lower increase in cardiac output (21). The pressor response was, however, unchanged due to propranolol, as a sustained hand grip caused a significant increase in total peripheral vascular resistance. A shift in the vascular response occurred when the cardiac response was limited. A similar shift occurs in patients with congestive heart failure (18) and in patients with mitral stenosis (13).

Although there are regulatory differences between the cardiovascular responses to static and dynamic exercise and clear-cut hemodynamic differences, eg, in blood flow and vascular resistance, it is difficult to make direct comparisons because of the differences in the muscle masses active during static and dynamic exercise. It has been suggested that active muscle mass might be one of the major determinants of the cardiovascular response to exercise, both dynamic and static (4).

### **Cardiovascular diseases and static load**

The normal heart increases its performance in response to exercise. Heart rate may increase 2.5-fold

from the resting value and cardiac output may be three to five times higher than the resting value. In many cardiovascular diseases the resting heart function may be normal, but the response to exercise is impaired. This phenomenon has led to the use of various exercise stress tests in the study of the potential reserve of the heart to respond. Generally the exercise tests used are either of the treadmill or bicycle ergometer type, both of which measure dynamic work capacity despite the fact that many work tasks needing physical effort are at least partially static in nature.

Although the exact mechanisms may not as yet be totally clarified, it is apparent that the cardiovascular response to static exercise is different in nature than the corresponding response to dynamic exercise. Persons with cardiovascular disease may have a rather normal response to a dynamic exercise test, and this response would indicate that they should succeed in certain work tasks. In practice, however, there might be a discrepancy between the individual's performance on a work task and the results on an exercise test. This discrepancy may be of great importance if a patient has high blood pressure, coronary heart disease, stress-related arrhythmias, cardiomyopathies, or valvular diseases. The use of static exercise tests may help to evaluate individual performance in such cases.

The increase of the afterload is especially useful in evaluating cardiac reserve. In order to respond to increased arterial pressure, the heart must increase contractility and heart rate to maintain the appropriate cardiac output. The infusion of angiotensin has been used to increase arterial pressure, but the use of an isometric exercise test is a far more effective physiological means of increasing afterload. In such experiments patients with impaired ventricular reserve have shown a marked rise in the left ventricular end diastolic pressure, together with a fall in the left ventricular stroke volume (23). Patients with a normal ventricular reserve had a marked increase in stroke index and no or a slight increase in end-diastolic pressure. Patients with aortic regurgitation had a higher increase in systolic blood pressure than referents despite similar heart rate response to a hand grip test corresponding to 33 % of the maximal voluntary contraction (9).

During a hand grip test at 25 % of the maximal voluntary contraction for 3 min the coronary sinus blood flow has been shown to increase more than twofold, a finding clearly suggesting an increased oxygen demand on the part of the heart muscle (13). A 10 % increase in systemic vascular resistance and about a 40 % increase in a cardiac index were also found at the end of a 3-min hand grip test at 25 % of the maximal voluntary contraction level (13). When the cardiovascular response to the same test was evaluated with respect to the clinical classification of the New York Heart Association and the cor-

relation between left ventricular end-diastolic pressure and left ventricular stroke work, a good estimation of the cardiac reserve was observed (13, 22). In those patients with a poor cardiovascular reserve (according to category III of the New York Heart Association), no increase in the left ventricular stroke work was found, but the end-diastolic pressure of the left ventricle increased markedly during a hand grip test at 25 % of the maximal voluntary contraction. In those persons with a good cardiac reserve left ventricular stroke work increased markedly, but the left ventricular end-diastolic pressure did not show any marked increase. When utilizing the present noninvasive methods of clinical physiology (electrocardiography, pulse curves, echocardiogram), one is able to estimate the cardiovascular reserve of patients during a hand grip test, whereas the same estimation would not be possible during a dynamic stress test (22). Furthermore, the hand grip test is, in practice, easily applicable also in invasive patient examinations (11, 15). Preoperative response to the hand grip test has been shown to have prognostic significance with respect to the postoperative course of the disease (aortic insufficiency, aortic stenosis or combined), and ventricular dysfunction has been revealed during the cardiac catheterization of patients with various aortic valve diseases (11). Huikari et al (11) found that, for patients with aortic regurgitation, the ejection fraction in a hand grip test (30 % of the maximal voluntary contraction) remained low even after successful valve replacement. Furthermore, it was found that, although the postoperative isovolumic cardiac indices became normal at rest after aortic valve replacement in some patients, the response to hand grip remained pathological during cardiac catheterization (15). The isometric test has been found to be even safer for patients than dynamic testing (5). During isometric testing the heart can be examined noninvasively, eg, with an echocardiograph, which may reveal many dysfunctions not always apparent in electrocardiograms monitored during a dynamic test (7).

### Effects of static exercise training on the heart

The effects of dynamic exercise training on the cardiovascular system are well known. The heart volume increases due to the enlargement of cavities and also to some extent due to an increase in muscle mass.

The effects of long-term static exercise training on heart functions have been studied in power trainers such as weight lifters, hammer throwers, etc (12). During static exercise training the heart volume does not increase. Both absolutely and in relation to body weight, the heart volume of endurance trainers is higher than that of power athletes (12). Dynamic exercise training increases the volume of the heart relatively more than the thickness of the heart wall, while static training increases mainly the left ventric-

ular wall thickness and results in left ventricular hypertrophy. The stroke volume of endurance athletes is considerably higher than that of referents, while that of power athletes is lower than that of referents when related to heart volume (12). Interestingly, the heart rate of power athletes is not lower than the reference values of untrained persons at rest, while the heart rate of endurance athletes is. In addition the cardiac index of power athletes remains at the reference level, while that of endurance athletes increases despite the fall in the resting heart rate. After isometric training the relationship of the septal thickness to the posterior wall thickness increases (12). The ejection fraction does not increase significantly for endurance athletes, and it remains within reference values for power-trained athletes also, that is, until increased muscle growth decreases left ventricular cavity and the ejection fraction increases. In power training no enhancement of the pump capacity occurs.

To summarize, the stimuli initiating the adaptation processes are increased preload with the isotonic (dynamic) training and increased afterload with the isometric (static) training. Neither type of training produces any myocardial damage in a healthy heart. However, when these data are applied to the cardiac patient, it is essential to differentiate between dynamic and static training in, eg, rehabilitation programs because of the different amount of strain on the heart. Endurance training alters the cardiovascular response to static exercise. For example, endurance trainers have been shown to have relative bradycardia in comparison to referents during static hand grip exercise, while power athletes react in the same manner as their respective referents (17).

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