Physiological mechanisms, controlling cardiovascular responses to muscular static load

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PHYSIOLOGICAL MECHANISMS CONTROLLING CARDIOVASCULAR RESPONSES TO MUSCULAR STATIC LOAD

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Abstract:

A review synopsis of physiological mechanisms controlling cardiovascular responses to muscular static (isometric) load is presented. Muscular static load during work activity is associated with development of disorders of the musculoskeletal system known as repetitive strain injuries, cumulative trauma disorders or activity and work-related musculoskeletal disorders. One of the basic topics of occupational and physiological research is investigation of the mechanisms underlying induced cardiovascular responses to muscular static load. Static muscular load might affect most of the physiological cardiovascular responses, such as – heart rate, cardiac cycle length, arterial pressure. Cardiovascular responses vary with the intensity of muscular exertion and contraction, and are part of processes that occur to adapt the circulation to the skeletal muscles’ need of blood. Reflex mechanisms that mediate circulatory changes during static muscular contraction, stimuli that initiate the reflex mechanism, and nerve structures which compose the reflex arc are indicated.

Key words: static load; isometric contraction; cardiovascular responses; reflex mechanisms; musculoskeletal disorders; health risk
Research studies indicate that human activity is associated with performance of muscular static work. Several main demands must be met: moving the body or its parts, transporting or moving other objects, and maintaining the body posture. Muscular loading during intensive work activity has been linked to the development of disorders of the musculoskeletal system known as repetitive strain injuries, cumulative trauma disorders or activity and work-related musculoskeletal disorders. Static muscular load is associated with muscle fatigue, pain and myalgia. When exposed to demands of performing static muscular load the human body responds with complex series of reactions, leading to muscular exercise. The muscle contraction is the end point of reactions taking place in the sensory organs, the brain, nervous system, heart, blood vessels, lungs, and musculoskeletal systems.

One of the basic topics of occupational and physiological research is investigating of mechanisms underlying induced cardiovascular responses to muscular static load. Activities that require static force development occur in lifting, pushing, and grasping. Static muscular load might affect most of the physiological responses, such as cardiovascular responses – heart rate, cardiac cycle length, arterial pressure. Heart rate, cardiac cycle length, and arterial pressure are cardiovascular indices frequently used for studying of the response of cardiovascular function to static muscular exercise. Cardiovascular responses are controlled by neural, humoral, and biomechanical factors in order to adjust its function to the variable needs of the organism’s tissues. Among these, one of the most variable types of demands arises from the skeletal muscles – at isometric (static) muscular exercise. Cardiovascular responses vary with the intensity of isometric contraction and are among the processes developed to adapt the circulation to the muscles’ need of blood.
A major part of the physiological literature concerning the phasic reaction of heart rate and arterial pressure describes results from studies where static contractions were performed [4, 5, 11, 15, 26, 30, 33, 36, 39]. One of the purposes of our studies was to demonstrate the relation between the changes in arterial pressure and pulse wave velocity in a study where the independent variable – the arterial pressure is investigated at sustainment of muscular static effort performed at 25 % of Maximum Voluntary Contraction [2]. This model of increased arterial pressure revealed that the functional change of vascular wall (vasoconstriction) and the increased cardiac output induce decreasing of the Pulse Transit Time [1, 3].

Our studies in the filed of occupational medicine are associated with assessment the risk of static workload and work-related musculoskeletal disorders at following occupations – medical doctors and nurses, lawyers and magistrates, video-display operators, teachers, social workers, employees, and etc. (Hospital of Lung Diseases “Saint Sophia”; Heatdistribution “Brunata”; Supreme Judicial Council; Ministry of Health; Sophia’s District Court of Justice; GSM Operator - Mobikom; Higher Transport School “Todor Kableshkov”; Children’s Social Medical Care Home “Saint Paraskeva”; State Agency for Refugees, and etc.)

Results of research investigations enable to be performed following conclusions about the pattern of cardiovascular responses at muscular static (isometric) load and contraction:

**CIRCULATORY CHANGES TO ISOMETRIC CONTRACTIONS**

At the onset of isometric contraction heart rate and arterial pressure both increase immediately [11, 16, 21, 32, 36]. The results of these studies reveal that the
maximal contraction of upper arm flexors for a period less than 1 sec in response to an acoustic signal the cardiac cycle following the one in which the contraction started was shortened in 95% of cases. Research studies show also that a latency of about 550 msec exists between the onset of the contraction and the first detectable significant shortening in cardiac cycle length [21]. In subsequent study is concluded that anticipation or perception of the acoustic or visual signal might be responsible for the instantaneous rise in heart rate [6].

Following short-lasting (< 1 sec) maximal isometric contractions a peak cycle length shortening was found at approximately 2 sec after the onset of contraction and the amount of cycle length shortening varied between individuals from 5 to 20% [6, 21, 28]. With longer-lasting isometric contractions the increase in heart rate and arterial pressure depends on the intensity and duration of the muscular effort [23]. There is still no unanimity regarding the relation between the magnitude of the circulatory responses and the force of muscular contraction. Research studies reveal an independency of the cardiovascular responses to the amount of muscle mass involved at handgrip contractions, thigh muscle contractions or adduction of a single finger all performed at 20% of their maximal effort [29]. Another result is that the effects of simultaneously contracting muscle groups was not additive – persisting and short-lasting (< 1 sec) bilateral and unilateral handgrip and ankle contractions were related to identical cardiovascular responses [6].

In contrast to the preceding results recent studies show that the increase in heart rate and arterial pressure depend on the absolute force developed [33, 37]. Isometric finger contractions, handgrip contractions, knee extensions, and combined handgrip plus knee extensions all performed at 40% of Maximal Voluntary
Contraction (MVC) resulted in increasing of the hemodynamic changes. A clear linear relation between the responses and the force developed is not found.

Summarized research studies investigate the cardiovascular responses to short-lasting isometric contractions. The extent of circulatory changes depends on the duration of the effort. With non-fatiguing contractions (less than 10-25 % MVC) the heart rate increases rapidly, reach a plateau within the first minute and remain on this level throughout the duration of the contraction. With stronger contractions of long duration the heart rate and arterial pressure continue to rise during the contraction, reaching their peak values at the moment that fatigue occurs or when the contractions ends earlier at the moment of release. This implies that the sizes of the cardiovascular responses also depend on the type of muscles used, fatiguable (white musculature) or fatigue-resistant (red musculature). When a contraction is performed using a fatigue-resistant muscle at a given percentage of its maximal effort, the effect on heart rate and arterial pressure will be less than the effect of contraction using a fatiguable muscle at the same percentage MVC and of equal duration.

REFLEX MECHANISMS CONTROLLING CARDIOVASCULAR FUNCTION DURING MUSCULAR STATIC CONTRACTION

Reflex mechanism mediating contraction-induced cardiovascular responses

At an investigation of reflex mechanism mediating the phasic changes in heart rate response at the onset of brief isometric handgrip contraction (0.5 sec lasting) (voluntary and electrically induced at 70 % MVC) is observed acceleration of heart
rate response [21]. The increase of duration extent of voluntary and electrically induced mm. quadriceps contractions at 20 \% MVC to 5 min is associated with identical responses in heart rate and arterial pressure [22]. Research analysis indicates that the reflex mechanism of central command or co-activation of the cardiovascular regulatory centers in the brainstem from impulses radiating from higher motor centers for motion control could not be excluded for the occurrence of circulatory changes during isometric contraction. Results of research studies reveal a reflex co-activated mechanism which co-ordinates cardiovascular control at the onset and during muscular contraction.

Stimuli which initiates the reflex mechanism and nerve structures which compose the reflex arc

Stimuli which initiate the reflex mechanism

The precise character of the stimulus necessary for the initiation of reflex mechanism mediating cardiovascular changes during isometric contraction is still debating. Research studies suggest the functional role of accumulation of some metabolite substances emanating from the contracting muscle, changes in pH, O\(_2\), and lactate, and the efflux of potassium for the changes in arterial pressure during isometric contraction [8, 9]. Results of these and other studies demonstrate the influence of intra-arterial injections of isotonic potassium solutions for stimulation of the activity of small myelinated (group III), and unmyelinated (group IV) muscle afferents [10, 15, 19]. This influence is related to increase in heart rate, arterial pressure, and contractility of the left ventricle.
Although potassium seems to be a likely stimulus for the mediation of the reflex responses to muscle contraction the effect of other stimuli could not be excluded [16, 17, 25]. Intramuscular pressure has been shown to vary linearly with the force developed [38], and that the light pressure applied locally to the exposed belly of a muscle activates about 40% of myelinated muscle afferents [35]. Muscle stretch activates half of all group III afferents, and only 10% of group IV fibers [35]. 40% of group III muscle afferents are activated by tetanic muscle contractions in the range of 20-100% MVC, and about 30% of group IV fibers activate non-proportionally upon contraction. Thus the research studies assume that more than one of the stimuli influence the reflex activation of the cardiovascular changes upon muscular contraction.

Nerve structures which compose the reflex arc

1. Muscle afferents involved in the reflex mechanism

There are research evidences showing that large myelinated fibers from muscle (group I and II) are not involved in the reflex mechanism mediating cardiovascular changes during isometric contraction. Research evidences reveal the functional role of autonomic activity in cardiovascular changes during isometric contraction. The fast vagally mediated cardiac acceleration at the onset of short-lasting muscle contractions might only be mediated by group III muscle afferents. [21]. These results and the results presented in relation to the activation of muscle afferents by chemical and mechanical stimuli indicate the involvement of group III, and possibly of group IV afferents in the reflex mechanism [19, 36].
2. Central pathways and connections

Research studies examine the influence of the ascending pathways in the spinal cord that might mediate somato-cardiovascular reflexes [10, 12, 15, 39]. Kalia et al. (1981) [24] indicate that groups III and IV muscle afferents have significant role for the cardiovascular effects in muscular contraction. Researchers demonstrate, by using standard histological techniques, that some of those afferent fibers relayed directly to the nucleus tractus solitarius in the brainstem. Fibers originating in this area are shown to have direct access to the nucleus ambiguus in the lower brainstem where cardiac vagal inhibitory fibers take their origin. Furthermore other fibers in the muscle nerve activated by muscular contraction are shown to terminate on ascending spino-thalamic tract neurons. Collaterals of these neurons may terminate in cardiovascular control center of the brainstem.

Inputs from group II and III somatic afferents crossing in the spinocerebellar tracts have been shown to elicit reflex responses in the inferior cardiac nerve. These afferents are known to project to the lateral reticularis nucleus in the brainstem. Electrical stimulation of this nucleus elicits arterial hypertension, tachycardia and increased activity in the inferior cardiac nerve through preganglionic sympathetic neurons emanating from this nucleus.

3. Efferent pathway of the reflex

The heart rate is controlled by the inhibitory effect of parasympathetic activity and the stimulating effect of sympathetic activity. Research topic of investigation is
whether the increase of heart rate during isometric contraction is a result of withdrawal of vagal activity, enhancement of sympathetic activity or to a dual autonomic effect. The study of this research issue is significant to predict the time course of the cardiac acceleration upon the onset of muscular activity or alternatively following a change in the intensity of a sustained effort.

Investigations suggest contradictory results. Some researchers assume that the heart rate response to isometric contraction is vagally mediated while other indicates the significance of sympathetic activity [7, 26, 28, 34]. Hollander H Bouman (1975) [21] observed an inhibition of cardiac acceleration to short-lasting (< 1 sec) voluntary contractions after the injection of atropine.

In conclusion it can be emphasize that the following three mechanisms control the response of heart rate during isometric contraction – immediately, i.e. within 0.5 sec after the onset of contraction heart rate increases as a result of the withdrawal of vagal restraint; after a delay of at least 2-5 sec following the onset of muscular contraction the effect of an increase of cardiac sympathetic activity may become apparent which also causes an acceleration of cardiac activity [20, 27]; predominantly during long-lasting contractions heart rate might be controlled by an increase in circulating catecholamines excreted by activation of the adrenal medulla.

4. Central command (cortical irradiation)

The efferent pathways are not activated only by a peripheral reflex mechanism but also by descending activity originating in higher motor structures in the brain passing over through the brainstem to its target – the spinal motoneurons. In passing by the
circulatory brainstem centers it would radiate collateral impulses that activate those centers.

The mechanism of central command or co-activation of the cardiovascular centers by impulses irradiating from higher motor centers of motion control could not be excluded for the occurrence of circulatory changes during isometric contraction. This concept is sustained of the following research evidences:

Freyschuss 1970 [14] compared the heart rate and arterial pressure responses to light handgrip contractions with the responses to intended contractions of the same muscles and the same intended force after local paralysis of the muscles and concluded that the cardiovascular changes during paralysis are elicited by central command.

Goodwin et al. (1972) [18] investigating the cardiovascular responses to isometric contractions vary the central command necessary to achieve a given force of contraction by means of tendon vibration. This vibration techniques predominantly activates the primary afferents of muscle spindles. Activity in these afferents excites the spinal motoneurons of the homonymous and agonist muscles, and inhibits the activity in motoneurons of antagonist muscles. Consequently when during isometric contraction the tendon of the contracting muscle is vibrated less central command is necessary to achieve a certain level of tension because the vibration-stimulated primary afferents promote to achieve this level by exciting homonymous motoneurons. When spindle afferents are activated in the antagonist of a contracting muscle a greater central command is required to achieve a given level of force development. It was shown in these studies that when the effect of vibration promotes to build a given force in a contracting muscle the heart rate and arterial pressure are less than without vibration, and conversely, when the effect of vibration counteracts
the achievement of a given level of tension of an antagonist, the cardiovascular responses are more than in contractions at the same level of force but without vibration. These observations suggest strongly that the cardiovascular responses are related to the required amount of central command.

Freund et al. (1979) [13] investigate the cardiovascular responses to a maximal effort of the quadriceps muscles during complete motor loss and lack of sensory information of the leg muscles during peridural anesthesia. No pressor responses are shown during the anesthesia but during the return of strength after anesthesia increasing pressor responses are observed at contractions of increasing force, the relation being linear.

The pressor responses are result of mechanism of central command. The existence of central command is indicated in other studies [12, 15, 18, 25, 31, 39, 40].

In conclusion in this review article evidences are indicated and analysed showing the existence of a reflex mechanism that controls the cardiovascular function from the onset of and throughout isometric contraction. The analysis of some results necessitates a second neural mechanism to be operative and inferential evidence in favour of central command is indicated. Both neural mechanisms might mediate cardiovascular responses during isometric contraction in normal circumstances. When one of the mechanisms is turned off the other will take over. This co-ordination necessitates convergence of neurones on cardiovascular brainstem centers. Responses of heart rate and arterial pressure vary with the intensity of isometric contraction during static load and are among the processes developed to adapt the circulation to the muscles' need of blood.
Besides the research issue of cardiovascular responses and regulating reflex mechanisms during static load in recent physiological studies and occupational field medicine investigations which are implementing, an issue of current research is the assessment of health risk during muscular static load. Risk of static load should not be considered only in the context of inducing musculoskeletal disorders and complaints. The simultaneous process of human-environment and human-computer interaction is associated with exposure of every individual to the effect of occupational psychological and social stress that contributes to inducing of complaints of muscle strain and musculoskeletal disorders. Occupational exposure to psychological and social stress at the work place leads to an increased risk of Cardiovascular Diseases (CVD). The summarized results of occupational medicine studies in our review synopsis are aimed at determination of the pattern of cardiovascular responses during static load. Next phase of our studies might be the change of the pattern of cardiovascular responses during static load at different occupations. In view of indicated risk exposures the process of work activity is associated with necessity of analysis and assessment of occupational health risk – risk of musculoskeletal disorders, cardiovascular risk, and determination of CVD as a result of the effect of work-related psycho-social stress. Next phase and theme of our studies might be an investigation and analysis of physiological mechanisms that mediate occupational psycho-social stress and musculoskeletal disorders.


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