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Katarzyna Żołądkiewicz[1], Paulina Brzezińska[2], Jacek Żakowiecki[3]

Heart muscle dysfunction during physical activity – biochemical response of muscle tissue

[1] Institute of Physical Education, Kazimierz Wielki University, Sportowa 2 Str, 85-091 Bydgoszcz; Poland
[2] Gdansk University of Physical Education and Sport, Department of Gymnastics and Dance, Gdansk, Poland
[3] WSB University in Toruń, Toruń, Poland

Abstract
In order to achieve the goal of completing a marathon run, athletes who make this effort are on the verge of their endurance. The marathon run is a serious challenge for many regulatory and homeostasis systems. During exercise, dehydration, hyperthermia and the synergistic effect of both stressors occur, reduce the stroke volume of the heart and thus blood flow to the muscles, skin and brain. Such intense effort releases markers of heart damage. Their presence is influenced by many factors.
The aim of this review is to summarize current knowledge regarding to effect of intensive physical activity on heart muscle functioning and biochemical response during this type of response. This type of revive could lead to better understanding of this process and propose protective methods during this type of response.

Keywords: rhabdomyolysis, physical activity, myoglobin, muscle

Introduction

Moderate intensity physical activity is recommended for every person who wants to maintain good health and basic physical fitness. The presence of any physical activity is definitely more health-promoting than leading a sedentary lifestyle. Introducing aerobic training to everyday life is conducive to maintaining proper functions, e.g. the cardiovascular system [1,2]. According to a study by Thompson et al., The risk of cardiovascular mortality was reduced by 45% in people with cardiovascular disease. Moreover, Arem et al. reported that performing physical activity 10 times the minimum recommended by official guidelines is not associated with an increased risk of mortality, but at the same time this level of PA has a higher risk of all-cause mortality compared to moderate levels of PA.

Depending on the intensity and duration of physical activity, the level of energy activation and physiological changes may generate changes in serum cytokines secretion, which may persist for up to several days [7] and indicate the occurrence of injuries during activity, such like rhabdomyolysis, inflammation process or a heart failure.

[1] Katarzyna Żołądkiewicz – corresponding author. e-mail: zoladkiewicz.k@gmail.com, ORCID: 0000-0001-5222-8419
[2] Paulina Brzezińska, ORCID: 0000-0001-5274-6208
[3] Jacek Żakowiecki, ORCID: 0000-0002-5940-6502

For the correct diagnosis of those changes it is urgent to know what type of biochemical markers may indicate deadly for the sportsman cardiovascular system failure. From long list of biochemical parameters only few of them are directly specific and indicate only heart dysfunction. They are called heart markers, and changes of them are associated with direct heart dysfunction (for example heart attack, prolonged ischemia etc) [1,2,3].

Heart dysfunction markers

As a result of myocardial hypoxia, some proteins are being produced. The amount of them is associated with the general overload or damage to the heart muscle. Most of those proteins are typical enzymes that are essential for the normal functioning of the heart muscle. Diagnosis of them can be important specially in many cardiovascular diseases, because provides information about metabolic activity of heart muscle. The concentrations of individual markers increases, reach a maximum and return to normal levels depending on the time of occurrence of the myocardial damage. By monitoring the concentration of those enzymes, it is possible to observe the course of the disease and implement appropriate medical treatment [6]. Most important proteins indicating heart dysfunction are Troponins (TnC, TnT, TnI) and keratin kinase (CK-MB) [4,5,16,17].

Troponins, are the group of proteins responsible for the proper functioning of the heart, regulating contractions of muscle fibers. This group includes troponin T (TnT), responsible for the attachment of tropiomiosin, troponin I (TnI), which binds actin and inhibits its contact with myosin, and troponin C (TnC), which binds calcium during contraction.

The troponin complex is located on the thin fiber of the striated muscle contraction apparatus and consists of troponin T (39 kD), troponin I (26 kD) and troponin C (18 kD), each encoded by a separate gene [16]. The increase in TnT and TnI levels may indicate, among others, myocardial infarction [8], but it is also noted when exposed to a stress factor, without the need for coronary disease [22]. The troponin increase occurs several hours after the myocardial damage, and this condition may persist even up to 10 days after the sytuation [6]. In clinical practice concentration of cTnI is as effective in diagnosis and prognosis as concentration of cTnT. Any differences in the results are likely due to differences in patient populations, blood collection times, and used analytical methods. In impaired renal function, cTnI is higher even to second-generation cTnT tests. In the case of muscle damage, cTnI measurement is as useful as cTnT [17].

CK-MB - creatine kinase MB - is one of the most popular markers of the heart muscle cells (myocytes) dysfunction. CK is an enzyme that converts creatine into a high-energy compound, which is phosphocreatine. This cardiac enzyme is helpful in the diagnosis and treatment of myocardial infarction (an increase in CK-MB activity in myocardial infarction is observed 4-6 hours after the onset of symptoms) coronary artery disease, heart injury, skeletal muscle disease, hypothyroidism, alcohol intoxication [6]. It is believed that the patient's age, gender, race and physical activity influence CK activity. In a study by Brewster et al., Physiologically higher CK values were found in 49% of black people, 13% of white Europeans and 23% of South Asian people [9].

In the studies by Brewster et al., it was shown that the increase in CK activity has an effect on blood pressure and is a one of the factors in the development of hypertension, especially in the presence of comorbid diseases. Of the 46 participants of the study, arterial

hypertension was diagnosed in 48% of patients and the increased CK activity in serum was associated with increased CK activity in tissues [12].

Biochemical changes after exercise

Troponin I (cTnI) and troponin T (cTnT) are highly specific "cardiac troponins", proteins (cTns) involved in the damage of myocardial cells are a key element in the diagnosis of acute coronary syndromes and myocardial necrosis. Stressful situations may lead to an increase in cTns in competitive and recreational athletes without signs of coronary artery disease, while the clinical significance of exercise-induced cTns release is still not fully understood [13]. Recent studies have shown that cTns levels increase after aerobic exercise with increasing heart rate and blood pressure [14].

This phenomenon occurs during prolonged physical exertion, during which the heart is exposed to hard work. The troponin concentration value is an individual factor, depending on on age, body weight and VO2max [23].

In 2002, Kratz, in a study conducted on marathon runners, determined the cTnl protein to check, e.g. influence of prolonged effort on the work of the heart muscle. Serum was collected immediately before the run, 4 and 24 hours after the run. The concentration of troponin I in the serum before the run was not recorded, after 4h - 0.02 ng / mL, so that after one day the value was again 0. Statistically, changes in cTnl were recorded in 3 people.

In the studies conducted in 2009 on runners covering a distance of 21 km, troponin I and T were determined. The study group was teenagers. Four blood samples were collected - before the run, 2, 4 and 24 hours after the run. The results are presented in Table 1.

Table 1. Troponin I and T 21 km running response [According to Fu et. all 2009].

	Rest	2h	4h	24h
Cardiac troponin T (cTnT), (ng/mL)				
Median (range)	**0.005 (–)**	**0.065 (0.005–0.08)**	**0.10 (0.005–1.22)**	**0.005 (0.005–0.05)**
Positive rate (%)	**0**	**66.7**	**66.7**	**8.3**
Cardiac troponin I (cTnI), (ng/mL)				
Median (range)	**0.02 (0.01–0.05)**	**0.13 (0.03–1.13)**	**0.195 (0.04–2.21)**	**0.045 (0.02–0.17)**
Positive rate (%)	**0**	**66.7**	**91.7**	**41.7**

The study found that cTnT, cTnI values increased significantly above the resting state of teenage athletes after a 21 km run. Eight out of twelve runners had cTnT above the AMI cutoff (0.05 ng / mL) after the run. Seven of them had results above 0.075 ng / ml. It was noticed that compared to trained adults, adolescents with a similar level of physical capacity released more cTnT [15]. According to Fu, the high level of tropin release in young players is due to the endogenous protective system of the heart, which is not yet fully formed.

Creatine kinase isoenzymes are studied in assessing the state of myocardial damage and is also a marker of the state of skeletal muscle damage. The popularity of the marathon resulted in the interest in measuring CK-MB activity in the serum of athletes [18]. The percentage of CK-MB (1 to 10% of total CK activity) was recorded in runners at the time of blood drawing, both during and after training for the marathon. This range indicates acute myocardial damage. Additionally, the pattern of increase and decrease in total serum CK and CK-MB after marathon (42.2 km) is similar to that after acute myocardial infarction [19].

Research shows that during long-distance runs, the greatest increase in MB creatine kinase does not occur immediately after the run. In a study by Kratz (2001) on marathon runners, no statistical significance was found in the samples obtained within 4 hours after the marathon compared to the control samples. However, a significant increase in CK-MB was observed with the subsequent consumption 24 h after the run. Creatine kinase-MB (ng / mL) results before - 2.3 (1.61), 4h after - 23.8 (25.17), 24h after - 56.2 (46.54).

When measuring CK-MB in teenage runners after 21 km competitions, a significant increase in serum CK-MB concentration was also noted. Here, however, the greatest increase was noticeable 4 hours after the competition. Pre-race results - 8.8 ± 6.0, 2h after - 13.4 ± 8.0, 4h after - 13.9 ± 8.0 and 24h after 13.4 ± 6.8 (ng / mL) [21].In training populations CK values can be up to ten times than a normal limit. Moreover, in people regularly engaged in vigorous physical activity, baseline CK values may be higher to [11].

Conclusion

The obtained results confirmed that the increase in troponins after long-distance running is associated with muscle dysfunction. Most of the values in presented research were above the norm even 24 hours after exercise. The occurrence of myocardial necrosis cannot be ruled out in these cases, the more so as the type of exercise is cyclically repeated by athletes.

It has been observed that the troponin secretion is also related to the athlete's age. In a Fortescue study, he noticed a significant increase in troponins in teenage runners. In addition, it is worth noting that the increase in heart markers is directly related to the level of training of a given player, during training, the heart tissue adjusts to work, and thus, when exposed to extreme effort, cells are not damaged to such a large extent. So the main findings from this work is that observation of changes of troponins and CK-MB is a good predicting factor on work overload and can be a useful tool the evaluation on sportsman preparation specially in the case of aerobic preparation for the activity.

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