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Opinion

Heat Stress and Psychomotor Performance - Neglected Mechanisms of Cardiopulmonary Capacity in Health and Heart Failure

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The reliable assessment of exercise performance and the precise definition of all factors determining aerobic capacity in health and chronic heart failure (CHF) are still a matter of debate. It is known from previous investigations that the aerobic capacity is modulated by many determinants, i.e. ergoreceptors and chemoreceptors, skeletal muscles, neurohumoral, hormonal and immunological factors, autonomic nervous system [1,2]. There is an increasing number of evidences that confirm the contribution of thermoregulation and psychomotor performance to the exercise tolerance [3-5]. It was previously suggested that blood's kinetic energy derived from thermal conditions of the tissues may affect circulatory flow and subsequently determine cardiopulmonary capacity [6,7]. The relevant discrepancy between the cardiac function parameters and functional capacity during cardiopulmonary exercise testing (CPX) in patients with systolic heart failure or athletes obviously indicates the importance of extracardiac factors for exercise tolerance [8,9]. The central nervous system (CNS) processing that determines psychomotor performance and controls human heat balance can be altered both in patients with heart failure and healthy persons with reduced exercise capacity [9,10]. The hypothalamus – the superior regulatory center of core body temperature in humans remains in an anatomical and functional relationship with cerebral cortex, brain stem autonomic cardiovascular and respiratory centers, hormonal system that control cardiopulmonary capacity. It is suggested that this specific brain structure as well as brain cortex participate in the regulation of hyperventilation during physical effort (locomotor region in hypothalamus – HTLR) in the physiologic conditions [11]. It was previously demonstrated that brain structural injuries in CHF patients emerge in areas involved in the physiologic control of core body temperature (hypothalamus) and breathlessness sensations in healthy humans and patients with idiopathic hyperventilation [12,13].

An increase in core body temperature during exercise is a physiological reaction that is related to the acceleration of metabolism necessary for the maintenance of proper energy delivery to working skeletal muscles and the enhanced production of heat by contracting skeletal muscles [14]. Previous reports confirmed the strong link between core body temperature and aerobic capacity in the heat stress conditions (high ambient temperature) [15]. The core body temperature during exercise is determined by the efficacy of central and peripheral thermoregulatory mechanisms, autonomic nervous and hormonal system and ambient temperatures. The magnitude of increase in core body temperature during exercise is mainly a resultant of the intensity of metabolic processes within human organs (especially in the skeletal muscles) and the efficacy of mechanisms of heat expulsion, i.e. ventilation, vascular system (skin vessels reactivity), heart function, water balance, baroreflex that are controlled by hypothalamus and the autonomic cardiopulmonary centers [16]. The previous reports validate importance of baroreflex to the pathophysiology of chronic heart failure and regulation of blood pressure [17]. The experimental studies demonstrated that gigantocellular nucleus responsible for brain controlling of cardiovascular functions has specific neuronal projections to hypothalamus and is very sensitive to the thermal stimulation [18]. There are strong functional and

anatomical links between thermoregulation (heat stress), central nervous system (brain cortex activity, psychomotor functions), skeletal muscle physiology and cardiopulmonary system. Skalik., *et al*. demonstrated that heat stress during physical exercise, psychomotor performance and oxygen consumption at anaerobic threshold (VO_2AT), i.e. one of the main determinants of skeletal muscle (respiratory and limb muscles) contraction efficiency during exercise, build one functional module that determines maximal exercise performance [19-21]. The experimental studies confirm the relevant contribution of central thermoregulatory mechanisms (hypothalamus) to carbon dioxide central chemosensitivity and the intensity of ventilation [22].

Nybo and Nielsen showed that the increase in core body temperature was strongly related to the significant changes in electroencephalography (EEG) reflecting brain cortex activity [23]. The high psychomotor performance (brain processing) can contribute to less fatigue of respiratory and limb muscles during exercise and facilitate the attainment of higher minute ventilation, higher oxygen consumption at the maximal exercise and more effective expulsion of heat through the lungs. Chmura and Nazar demonstrated that the improvement of exercise performance following regular training sessions in athletes is related to the metabolic changes and amelioration of psychomotor functions [4]. It was previously demonstrated that minute ventilation is a very important mechanism to expel excess of heat during extreme exercise in humans [24]. Thomas., *et al*. demonstrated that the significant increase in core body temperature and following diminished cortical activity in healthy humans are mainly responsible for the decrease in contraction force of the working skeletal muscles (limb and respiratory muscles) which is partially responsible for breathlessness sensations [25]. The heat stress does not impair the ability of the muscles to generate force, but sustained force production is lowered as a consequence of a reduced neural drive from the CNS. The respiratory muscle fatigue can play a significant role in limiting the human performance at the extremes of exercise [26].

The significant increase in core body temperature can impair exercise capacity, and the primary pathway of the impairment may be the neuromuscular system [27]. The functional capacity of the neuromuscular system, from the central activation of the motor unit (brain cortex), through neural transmission along the peripheral nervous system, and at the individual muscle fibre is reflected by psychomotor performance in clinical conditions and can be directly altered by elevations in local muscle and core temperature.

Herman., *et al*. argue that heat stress can suppress activity of muscle pyruvate kinase - the main enzyme participating in energy generation for working skeletal muscles [28]. Hence, heat stress can impair VO_2AT that leads to muscle fatigue and subsequent reduction in maximal oxygen consumption (probably both in healthy people, athletes and patients with chronic heart failure). VO_2AT is one of the main metabolic determinants of skeletal muscle contraction power during exercise. VO_2AT is more dependent on the oxidative activity of skeletal muscles, type of muscle fibers engaged in exercise and mitochondrial volume than cardiovascular adaptation (cardiac output, stroke volume).

The importance of the above-presented physiologic mechanisms of exercise performance can be confirmed by some clinical observations in CHF patients and athletes. Miyamoto., *et al*. demonstrated the relevant positive effect of the controlled heat exposure (sauna, steam bathing) in CHF patients [29]. The cyclically repeated heat exposure during training period in healthy volunteers or CHF patients may cause habituation of cerebral cortex to the enhanced thermal load during exercise, resetting of the hypothalamic responsiveness to heat stress, amelioration of cardiopulmonary capacity and exercise performance [30]. There are also some preliminary reports confirming the positive impact of body heat extraction by means of the innovative techniques on exercise performance, muscle fatigue in athletes and patients with neurological disabilities [31].

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