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Combined Effects of Carbon Monoxide Inhalation and Heat Exposure during Mild to Moderate Exercise

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Carbon monoxide (CO) is an odorless, colorless gas that can cause sudden illness including myocardial ischaemia and failure even in the normal heart. CO has a very high affinity for hemoglobin and firmly forming carboxyhemoglobin (COHb). If CO is abundant in the air, CO blocks oxygen from getting into the body limiting tissue respiration by hypoxia. When blood concentration of rises above 10%, it can cause CO intoxication. However, even at a low COHb concentration of 2-6%, patients with coronary heart disease can experience ischaemic changes and ventricular arrhythmia. This level of concentration can be often seen in residents of industrial areas and in smokers. High concentration of COHb results in marked reduction of oxygen carry capacity and leftward shift of the oxyhemoglobin dissociation curve. At the cellular level, CO binds with cytochrome oxidase, inhibiting cellular respiration resulting anaerobic metabolism. CO also binds with other hemoproteins such as myoglobin, which abounds in skeletal muscles and the myocardium, causing dystunction by impairing their oxygen carrying capacity and the transportation of oxygen from the blood to the mitochondria. Many previous study showed a significant decrease of exercise performance in various concentration range of COHb. And heart rate reponses was high in CO inhalation.

The cardiovascular and thermoregulatory responses during exercise are elevated metabolic heat production resulting in increased body temperature, perfused sweating, dehydration, and cardiovascular strain. In particular, during dynamic exercise in the heat, primary cardiovascular problem is to provide sufficient blood flow to exercising skeletal muscle for adequate metabolic support and to peripheral regions for maintaining optimum body temperature. Thus, exercise in the heat creates diverse physiological stresses.

Several studies demonstrated that environmental heat stress increased intramuscular glycogen utilization, although not always evident. Much of studies support the concept of the increased anaerobic metabolism during submaximal exercise in the heat since plasma lactate accumulation is greater in a hot than in a comfortable environment. Studies on fat and protein metabolism during exercise in the heat are rare and these substances were not changed much. Collectively, the exercise and heat stress results in an increase in intramuscular glycogen use via both oxidative and nonoxidative energy pathways. Few studies examined the effect of exercise and heat stress on muscle energy metabolism. While intramuscular ATP content and phosphocreatine decreased, ADP and AMP accumulation increased in dogs. The mechanism responsible for the muscular energy metabolism has been proposed and includes blood flow reduction in the contracting skeletal muscle, alterations in neuromuscular recruitment pattern, direct temperature effect on metabolic processes, and sympathetic neural influences.

Combined exercise and heat stress while inhaling CO gas has not been investigated yet. Changes of body temperature and body fluid volume during exercise in the heat will cause energy metabolism and inhalation of CO will also cause muscular respiration. However, it is not clear whether those effects will exaggerated or abolished by these two factors.