MedicalXpress

Exercise nerve response in type 1 diabetes worsens over time

October 19 2017

A new study finds that late-stage type 1 diabetes mellitus (T1DM) weakens the autonomic reflex that regulates blood pressure during exercise, impairing circulation, nerve function and exercise tolerance. The study is published in the *American Journal of Physiology—Heart and Circulatory Physiology*.

T1DM is a lifelong autoimmune disease of the endocrine system in which the body does not produce insulin. Insulin is a hormone necessary for transporting glucose throughout the body. T1DM may also damage the nerves and heart. Statistics show that approximately 70 percent of people with diabetes develop nerve damage (neuropathy) to some degree and 17 percent of people with T1DM suffer from cardiovascular disease within the first 10 years of diagnosis. The combination of neuropathy and cardiovascular risk factors significantly increases comorbidity in this population.

A research team from the University of Texas at Austin studied the exercise pressor reflex in three groups of rats in the early, middle and late stages of an animal model of T1DM. When muscles contract, the exercise pressor reflex is responsible for increasing the amount of blood pumped out from the heart and causes small blood vessels to constrict, which together increase blood pressure. This response helps "the cardiovascular system to better match oxygen delivery with oxygen demand," the researchers explained.

An abnormal increase (exaggerated response) in the exercise pressor

reflex could be especially dangerous to people with T1DM who are already at higher risk for cardiovascular abnormalities and could increase their risk of heart attack and stroke during physical activity. This is concerning because medical professionals typically prescribe exercise as a treatment modality.

The researchers measured the exercise pressor reflex in the animals they studied. They found the blood pressure response to be exaggerated in the early and middle stage groups, indicating increased demands on the heart during exercise. At this stage of the disease, the researchers believe that hyperactive nerves that relay information from the contracting muscle are responsible for the abnormal increase in blood pressure, similar to what happens in painful diabetic neuropathy. Conversely, in the late stage of the disease, the male rats, but not the females, had a noticeably weaker exercise pressor reflex, which was demonstrated by a very small increase in blood pressure. This suggests that neuropathy had developed to the point where nerves were not able to sufficiently relay the information from the muscles. Taken together, the response seen in the early and late stages of the disease would both impair exercise capacity significantly and could impair blood flow to the heart and the brain, potentially leading to a heart attack or stroke.

"These novel findings provide new insights on the effects of T1DM on the autonomic control of circulation during exercise over the course of diabetes and highlight another severe implication that T1DM has on the cardiovascular system," the research team wrote. Due to these potentially harmful changes in the reflex response, people with T1DM are encouraged to take precautions that limit exercise intensity, they noted.

More information: Ann-Katrin Grotle et al. Temporal changes in the exercise pressor reflex in type 1 diabetic rats, *American Journal of Physiology - Heart and Circulatory Physiology* (2017). DOI: 10.1152/ajpheart.00399.2017