

Letter to the Editor

Does The Second Wind Blow From The Muscle, Heart or Brain?

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In a recent article, Vissing *et al.*, reported about a study of 12 patients with McArdle's disease who profited from intake of 75g sucrose before exercise with regards to exercise-tolerance and well-being (Vissing J., & Haller, R.G. 2003). The study raised the raised the following comments and concerns.

In addition to skeletal muscle, cerebrum and heart may be affected by the myophosphorylase-gene-mutation, manifesting as seizures (Leproive, T. *et al.*, 2002) or dilated cardiomyopathy (Walker, A.R. *et al.*, 2003). Since cardiac involvement was not assessed, it remains uncertain if improvement of exercise-tolerance can be exclusively attributed to improved fuel availability for the skeletal muscle. Glucose may reduce catecholamine levels and consequently heart-rate or improve myocardial contractility by increasing the Na/K-pump content (Haller, R.G. *et al.*, 1998). Reduction of heart-rate may also result from insufficient ADP-phosphorylation (Sahlin, K. *et al.*, 1990) or increased vagal tone from cerebral involvement. Glucose-administration may also economize heart-work, and influence blood-pressure, which was not registered, as well as expiratory exchange-ratio. Increase of heart-rate at the beginning of exercise may be the response of an impaired myocardium to maintain an adequate cardiac output. Adjusting exercise-workload to perceived exertion and heart-rate is subjective and requires prior exclusion of heart-disease.

To explain reduction of heart-rate and second-wind-phenomenon following sucrose administration in McArdle disease, cerebral or cardiac mechanisms should be additionally considered and investigated.

REFERENCES

1. Vissing J., & Haller, R.G. (2003). The effect of oral sucrose on exercise tolerance in patients with McArdle's disease. *N Engl J Med*, 349, 2503-9.
2. Leproive, T., Legendre, E., & Pinaud, M. (2002). Anesthesia for cesarean section in a patient with McArdle disease and hereditary dilated cardiomyopathy. *Ann Fr Anesth Reanim*, 21, 517-20.
3. Walker, A.R., Tschetter, K., Matsuo, F., & Flanigan, K.M. (2003). McArdle's disease presenting as recurrent cryptogenic renal failure due to occult seizures. *Muscle Nerve*, 28, 640-3.
4. Haller, R.G., Clausen, T., & Vissing, J. (1998). Reduced levels of skeletal muscle Na⁺K⁺-ATPase in McArdle disease. *Neurology*, 50, 37-40.
5. Sahlin, K., Areskog, N.H., Haller, R.G., Henriksson, K.G., Jorfeldt, L., & Lewis, S.F. (1990). Impaired oxidative metabolism increases adenine nucleotide breakdown in McArdle's disease. *J Appl Physiol*, 69, 1231-5.

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