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Editorial

Are the lungs built for rowing?

For a biological tissue, including the brain, to develop and to remain alert, it requires challenges, and, sadly, early retirement reduces cerebral blood flow. In Western style societies, the brain may be stimulated adequately, but the physical activity needed to maintain the locomotor and cardiovascular systems depends on leisure time activity. Interest in sports allows for the evaluation of health benefits – beginning with the finding of a 2-year prolonged life of former English rowers already in 1873 and now explained by the excellent physiological profile of elderly oarsman in several papers by Yoshiga and Higuchi (e.g. Yoshiga et al., 2002). Sport activities demand specific skills, and with excellence requiring years of training, adaptations can be evaluated in detail. As stated by A. V. Hill, "*the most accurate data are found not in textbooks on physiology, not even in textbooks on medicine, but in the World records (of runners)*".

Oarsmen are familiar with ergometer rowing including competitions over "2000 m", and the physiology of maximal exercise is studied in the laboratory. This issue addresses the O_2 transport system. Yoshiga and Higuchi (2003a) establish a relationship between maximal O_2 uptake ($VO_{2\,max}$) and body size among a large number of rowers to explain the difference in the work performed by females and males. Although the majority of the variation in power is explained by body dimensions, a 5% difference remains when sexes are compared at similar lean body mass. They (Yoshiga & Higuchi, 2003b) also evaluate the heart rate (HR) response to rowing and running. Surprisingly, the relationship between HR and VO_2 does not always prevail when different types of exercise are compared. Despite a larger $VO_{2\,max}$ during rowing than during running, the HR response is attenuated. The authors argue that the central blood volume is reduced during (upright) running compared with (seated) rowing. Hereby they address a variable that has been overlooked in the cardiovascular adaptation to exercise, and its integration could provide insight not only to the regulation of HR but also to the regulation of flow and, in turn, metabolism.

Yoshiga and Higuchi (2003b) pose the idea that the larger ventilation during rowing than during running indicates that it does not limit $VO_{2\,max}$, but the situation may be more complicated than that. Skeletal muscles extract almost all the O_2 provided by blood, and whole-body exercise in particular challenges the O_2 transport system. The traditional view is that the circulation limits $VO_{2\,max}$; however, the lungs may fail to secure oxygenation of arterial blood. A reduction in the arterial O_2 tension (PaO_2) is reported for some intensively trained runners and cyclists, but for all oarsmen during (ergometer) rowing (Hanel et al., 1994). At the same time, pH decreases to an impressive 6.74 reflecting blood lactate levels of 30 mM (Nielsen, 2003). The Bohr effect on the oxyhaemoglobin dissociation curve is considered to promote O_2 delivery to the tissue, but with the combined lowering of PaO_2 and pH the arterial saturation (SaO_2) is affected. During rowing, SaO_2 decreases to $\sim$90% corresponding to what is manifest at an altitude of more than 3000 m above sea level. In support, an increase in the inspired O_2 fraction, or the administration of bicarbonate, restores SaO_2, and the percent increase in $VO_{2\,max}$ corresponds to the enhanced SaO_2 (Nielsen, 2003).

It may be questioned as to when it pays off to perform work provided for by anaerobic metabolism in situations where PaO_2 is low. Clearly there is a delicate balance between ATP production by anaerobic metabolism and the lowering of aerobic metabolism caused by a reduced SaO_2. Some patients and also normal subjects exposed to high altitude tend to produce very little lactate ("the lactate paradox") (Lundby et al., 2000). The implication of the findings by Nielsen (2003) is that for endurance-type exercise, people and patients are wise not to lower their arterial pH so as to preserve SaO_2, and thereby VO_2, when PaO_2 is low. Conversely, hyperventilation secures an increase in alveolar O_2 tension to overcome the diffusion limitation of the lungs. Could it be that oarsmen cannot increase their alveolar O_2 tension to the same extent as the often smaller runners and cyclists?

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