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REVIEW ARTICLE

Rowing, the ultimate challenge to the human body – implications for physiological variables

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Summary

Clinical diagnoses depend on a variety of physiological variables but the full range of these variables is seldom known. With the load placed on the human body during competitive rowing, the physiological range for several variables is illustrated. The extreme work produced during rowing is explained by the seated position and the associated ability to increase venous return and, thus, cardiac output. This review highlights experimental work on Olympic rowing that presents a unique challenge to the human capacities, including cerebral metabolism, to unprecedented limits, and provides a unique opportunity to reveal the extreme range of many physiological variables.

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Introduction

Rowing over a 2000 m course, accomplished in about 6·5 min, requires a power of ∼590 W and represents a maximum for humans, while every step of the oxygen transport system from atmospheric air to the working muscles is challenged. In quadrupedal mammals most of the blood volume is at the level of the heart but upright humans have some 80% of the blood volume below the level of the heart, thus, constraining venous return. The extreme work produced during rowing is explained by the seated position and the associated ability to increase cardiac output during intense involvement of both arms and legs (Vogelsang *et al.*, 2006). This review highlights first the antropometric characteristics followed by the metabolic capacities of rowers with emphasis in physiological parameters of the oxygen cascade, from the moment it enters the body to when it reaches the muscle fibres.

The oarsman

The male rower is ∼195 cm and weighs about 95 kg with the female counterpart at 182 cm and 80 kg, respectively (Secher, 1993). These characteristics are comparable with the Neanderthaler (Kappelman, 1997), while the average human is ∼30% smaller. Nevertheless, with the introduction of the lightweight class in the Olympic program, rowing became a 'sport for all' rather than a 'sport for tall' (Khosla, 1983).

During rowing each crew member generates in synchrony a force of ∼450 N for more than 200 strokes. Muscle strength is larger than expected for the body size and, yet the strength of each muscle group is unrelated to the force produced in the rowing position that requires a unique capacity. For example, in most people the cumulative strength of unilateral leg extensions is lower than the strength developed during simultaneous extension of both legs. This leg strength paradox reflects the habitual unilateral use of the legs during locomotion. In contrast, rowers do not demonstrate such strength deficit as a result of the chronic simultaneous leg extension during rowing (Secher, 1975).

The rowing pace is slow compared with running and the percentage of slow twitch (ST) muscle fibres in rowers is as high as 85%, while the remainder of the fibres are dominated by fast twitch (FTa) fibres with high oxidative capacity (Roth *et al.*, 1993). The large strength of rowers reflects muscle hypertrophy in both FT and ST fibres but it is unique that in rowers the ST fibres may be larger than the FT fibres (Roth *et al.*, 1993).

Metabolism

The aerobic capacity of rowers is from the largest among endurance athletes (∼6·9 l min^{-1}). For comparison, the highest reported VO_2max is 7·48 l min^{-1} in a large, elite skier (Saltin, 1996), while in elite swimmers, who are also using both their arms and legs, is ∼5·1 l min^{-1} during swimming (Holmér,

1972). Anaerobic metabolism, indicated by blood lactate, may reach 32 mM during rowing and plasma bicarbonate is eliminated, while pH decreases to 6·74 (Nielsen, 1999). Thus, the oxygen deficit, representing the part of metabolism that is not covered by oxygen uptake is 90 ml kg^{-1}, or ~30% larger than during running (Medbø *et al.*, 1988). Lactate increases progressively with exercise intensity, a manifestation of attenuated elimination by the liver and kidneys (Nielsen *et al.*, 2002) and it serves as a substrate for tissues including muscle, and the brain (Quistorff *et al.*, 2008; Secher *et al.*, 2008).

Endocrine and metabolic responses

In response to mental stress, circulating eosinophils decrease by 80% before and are almost eliminated after a rowing race (Renold *et al.*, 1951). At the same time plasma catecholamines increase ~10-fold, while pancreatic polypeptide, a hormone under vagal control, doubles, (Holmqvist *et al.*, 1986) suggesting that vagal activity contributes to the symptoms experienced at exhaustion. Testosterone is increased by 15% after maximal rowing, and performance has been related to the resting values (Jürimäe & Jürimäe, 2001), while growth hormone is increased 10-fold (Jürimäe *et al.*, 2005). After rowing, the elevation of serum concentration of myoglobin and creatinine kinase indicates skeletal muscle cellular damage that explains eventual residual muscle soreness (Hansen *et al.*, 1982).

Ventilation

Ventilation (V_E) of 270 l min^{-1} is developed during rowing (Jensen *et al.*, 2001) despite the cramped body position during the initial phase of the stroke. Since breathing frequency does not differ between trained and untrained, and lung function does not improve with training, rowers are selected among people with an extraordinarily large vital capacity (VC). A correlation between rowing performance and VC is reported and rowers demonstrate values of 7 l compared to 5·5 l as expected for their body size, while a VC of 9·1 l has been recorded (Secher, 1993).

Haemoglobin

During rowing, the arterial oxygen tension (PaO_2) is reduced to the level seen at high altitude, and the pH reduction affects the ability of haemoglobin to transport oxygen (the Bohr-effect) and, consequently, VO_2max is restrained by ~7%, in proportion to the haemoglobin desaturation (~90%; Nielsen *et al.*, 1999). Conversely, breathing oxygen-enriched air attenuates the influence of a low pH on haemoglobin saturation and enhances performance by 2%. Arterial oxygen saturation is also restored in response to pH normalisation following bicarbonate administration. Oxygen delivery is enhanced both by the elevation of body temperature and the Bohr effect, due to the lower pH of venous compared to arterial blood as carbon dioxide is exhaled (PCO_2 reduced from 90 to 40 mmHg; venous versus arterial).

Blood volume and starling law of the heart

Despite the muscle pump and redistribution of blood volume by splanchnic vasoconstriction, the elevated blood flow demands of active muscles and skin, for thermoregulation, limit central blood volume. Yet, the central blood volume is higher during seated rowing than during running and allows for lower heart rate and larger VO_2max (Yoshiga & Higuchi, 2002). Thus, the seated position may partially explain the extreme work capacity during rowing.

The heart

With endurance training the internal diameters of the heart enlarge and the highest values for left ventricle end-diastolic diameter are 65 mm in rowers, while the ventricular shape is not altered (Pelliccia *et al.*, 1991, 2000). The demand for a large stroke volume in addition to overcoming the high blood pressure at the beginning of each stroke (Clifford *et al.*, 1994), in response to a Valsalva-like manoeuvre, is manifested in a heart size comparable with the largest hearts among elite athletes. For comparison, the largest stroke volume reported in world champion cyclist is 212 ml (Levine, 2008). The enlargement of the internal dimensions, with left ventricular mass of 330 g and left ventricular wall thickness of 16 mm, provokes inhomogeneous myocardial perfusion (Bartram *et al.*, 1998).

Exercise tachycardia develops although plasma potassium increases, e.g. to 6–7 mM (Nielsen *et al.*, 1999) in response to sympathetic activation that not only enhances cardiac function but also clears plasma potassium during and after rowing. Cardiac function is supported by a small increase in free plasma calcium released from albumin as pH decreases (Nielsen *et al.*, 1999).

Cardiac output, blood pressure and regional blood flow

During exercise, cardiac output of athletes ranges from 30 to 40 l min^{-1}. Rowing, with the associated exercise-induced hypoxaemia, stimulates erythropoietin production, and the haemoglobin concentration of rowers is larger than of normal healthy adults, with 10% of rowers having higher haematocrit than the no start limit in, e.g. cycling with 51% (Johansson *et al.*, 2009). Due to limitations on cardiac output, imposed by a restricted preload, mean arterial pressure is maintained by vasoconstriction not only to internal organs but also to working muscles and to the brain. During rowing blood flow is not allowed to increase at the expense of blood pressure, and muscle blood flow is reduced by ~30% compared to that seen during exercise involving a small muscle mass (Volianitis *et al.*, 2004). Consequently, gas diffusion is restrained despite enhanced muscle capillarisation (2·6 capillaries per fibre in trained subjects). During arm cranking oxygen diffusion in the arms may be ~50 ml min^{-1} $mmHg^{-1}$ in trained rowers (20 ml min^{-1} $mmHg^{-1}$ in the untrained), while it decreases

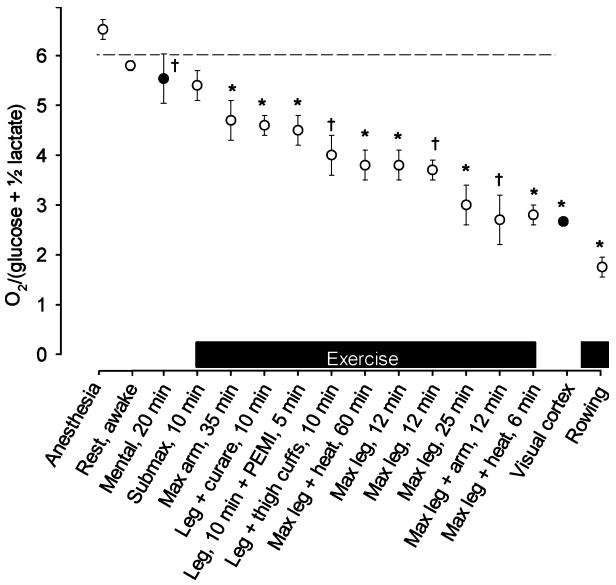

Figure 1 The cerebral metabolic ratio (brain uptake of oxygen relative to that of carbohydrate; glucose+1⁄2 lactate) during general anaesthesia, at rest and during various types of brain activation including several types of exercise with ergometer rowing demonstrating the largest deviation from the resting value of 6.

to 32 ml min^{-1} $mmHg^{-1}$ when legwork is added to arm exercise (Volianitis *et al.*, 2004).

Cerebral blood flow and oxygenation

Ultimately, it is the brain that limits performance and central fatigue describes a central limitation in recruitment of ST rather than FT fibres since the contraction maintains its rate of rise of tension while it looses its endurance (Secher *et al.*, 2008). With physical activity regional cerebral blood flow (CBF) increases in activated areas of the brain, but with the marked hyperventilation associated with rowing, the $PaCO_2$ tension decreases and that reduces CBF. Together with the hypoxemia developed during rowing, the reduction in CBF means that cerebral oxygenation decreases by ~10% (Nielsen *et al.*, 1999), a magnitude similar to that seen during fainting (Madsen & Secher, 1999), and is a factor to central fatigue.

Brain metabolism is expressed as the ratio of oxygen to carbohydrate uptake of the brain, known as the cerebral metabolic ratio (CMR) that is close to 6 at rest. The largest reduction in CMR, 1·7 during exhaustive ergometer rowing (Volianitis *et al.*, 2008), suggests that less than 30% of the carbohydrate taken up by the brain is oxidised, or that glycolysis accounts to more than ~20 mmol glucose-equivalents (Fig 1).

Conclusion

Olympic rowing presents a unique challenge as it stresses the human capacities, including the brain, to unprecedented limits. Rowing, thereby, provides a unique opportunity to reveal the extreme range of many physiological variables. These values provide for continuous clinical education that can support better informed diagnoses and decisions.

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