Cerebral metabolism during upper and lower body exercise

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IN THE AWAKE AND RESTING CONDITION, the energy demand for the brain is met almost completely by oxidation of glucose. However, with enhanced neuronal activity, the cerebral uptake of O2 increases less than that of glucose; i.e., the cerebral metabolic ratio of O2 to glucose decreases (15, 27, 28). A similar reduction in the cerebral metabolic ratio is provoked by intense exercise, where lactate also is taken up by the brain (6, 20). Inasmuch as lactate does not accumulate within the brain (9), it must be metabolized (43), and it is therefore included in the equation as O2/(glucose + 1/2 lactate). Although the fate of this surplus cerebral uptake of carbohydrate relative to that of O2 remains unexplained, it is, to some extent, related to replenishment of brain glycogen deposits (12, 27, 48). Intense neuronal activity may depend specifically on breakdown of astrocyte glycogen (5), and, in regard to exercise, depletion of cerebral glycogen deposits may signify “central fatigue” (6, 20).

The cerebral metabolic ratio has been evaluated during leg exercise, and at exhaustion it is reduced to its lowest value (6, 9, 17, 20, 33). The cerebral metabolic ratio is an integral of metabolism in several brain areas, and its reduction points to an influence of the mental effort associated with exercise. The importance of central integration for the reduction in the cerebral metabolic ratio is illustrated with enhanced command to exercise or sensory input from the working muscles using neuromuscular blockade (6) or thigh cuffs (7): in both cases, the reduction of the cerebral metabolic ratio is smaller than during control maximal exercise, which requires a maximal mental effort to accomplish the task and results in intense sensory stimulation, such as muscle pain (20). Furthermore, when arm cranking is superimposed on leg exercise, the cerebral metabolic ratio decreases more than when exercise is conducted with the legs alone (9).

Although it seems to be established that exhaustive exercise reduces the cerebral metabolic ratio, it is unknown to what extent distinct brain regions contribute, and this quantitative aspect was addressed in the present study by comparing the cerebral metabolic response of arm cranking with that of leg exercise. Exercise with the arms seems to stimulate the brain more than leg exercise and would be expected to induce a greater reduction in the cerebral metabolic ratio. The reasons for this are as follows: 1) The cortical representation of the upper extremity is greater than that of the lower extremity (37, 46, 56), and during exercise with muscle groups of the upper extremity, the increase in regional cerebral blood flow is greater than for comparable exercise with the lower extremity (25, 34, 46, 49). 2) Arm exercise is associated with a larger “central command,” as judged by the greater increase in cardiorespiratory variables (45, 52). 3) Movement of the upper limb is intentional or directed by a “will” and, therefore, controlled by supraspinal centers, whereas the legs in gait and locomotion are controlled by the spinal cord as modified by influence from supraspinal centers (18). 4) The potentially smaller muscle mass of the upper than of the lower body elicits a comparable, or even higher, blood lactate level (1), which appears to be of consequence for the reduction in the cerebral metabolic ratio (9, 20). Thus we hypothesized that exhaustive arm cranking would reduce the cerebral metabolic ratio more than exhaustive leg exercise.

METHODS

Sixteen healthy young men gave written informed consent to participate in the study as approved by the Copenhagen Ethical Committee (KF 01-36997). Half of the subjects cycled on a modified Krogh ergometer in a semisupine position (means ± SE: 22 ± 1 yr of age, 181 ± 4 cm, 71 ± 3 kg body wt), while the other subjects performed arm cranking and were slightly older (26 ± 5 yr of age, 181 ± 3 cm, 71 ± 3 kg body wt). The group that performed arm cranking attended the laboratory 3–5 days before the main study for determination of their exercise capacity (149 ± 21 W) and familiarization with the protocol. Both groups of subjects were instructed to refrain from exercise 24 h before

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the main study, and they fasted overnight, although drinking of water was permitted. A rate of 60 rpm was dictated by a metronome, and the workload was chosen with the aim of eliciting exhaustion after ~30 min, i.e., ~60% of the work capacity for the arms (91 ± 10 W), corresponding to a heart rate (HR) of ~150 beats/min. To control for individual differences between HR and exercise capacity, the subjects rated their perceived exertion (RPE) on the Borg scale (4).

Brain metabolism was evaluated by arterial-venous differences obtained from a catheter in an internal jugular vein (2.2 mm, 14 gauge) with the tip positioned below the base of the skull and another catheter in the brachial artery of the nondominant arm (1.1 mm, 20 gauge). One subject from the leg-exercise group was excluded from the study because of difficulties with blood sampling. HR was calculated from a three-lead electrocardiogram (Dialogue 2000, Danica, Copenhagen, Denmark), and blood samples were obtained simultaneously three times at rest, every 5th min during exercise, and in the recovery period at minutes 1, 2, 3, 5, 7, and 10. The syringes were preheparinized, rotated, and placed in ice water until analysis for glucose, lactate, and blood gas variables (model ABL 625, Radiometer, Copenhagen, Denmark).

Values are means ± SE unless stated otherwise. Within-group changes over time were detected by Friedman’s test and subsequently located using Wilcoxon’s matched-pairs test by rank. Between-group differences were identified using Mann-Whitney’s test. P < 0.05 was considered to be statistically significant.

RESULTS

Leg exercise. Leg exercise was sustained for 25 ± 6 min, during which RPE increased to a maximal level (i.e., 20), while the arterial lactate concentration increased to 12.6 ± 2.0 mM (Fig. 1). Conversely, arterial PCO2 (PaCO2) decreased during exercise and remained low in the recovery period. The arterial-venous difference for O2 increased at exhaustion and remained high during the recovery period, and the arterial-venous difference for lactate increased from −0.1 ± 0.0 mM at rest to a peak of 1.1 ± 0.3 mM (P < 0.05), whereas that for glucose did not change significantly except in the immediate recovery (Fig. 2). Consequently, the cerebral metabolic ratio decreased from 5.6 ± 0.2 at rest to 3.5 ± 0.2 after 10 min of exercise and further to 3.3 ± 0.3 at exhaustion (P < 0.05).

Arm cranking. Arm cranking lasted longer (35 ± 4 min) than exhaustive leg exercise but likewise elicited a gradual increase in RPE to 20, although it was lower during the first 15 min (P < 0.05), and the arterial lactate concentration also tended to be lower (9.7 ± 1.2 mM; Fig. 1). As during leg exercise, arm cranking reduced PaCO2, but in the recovery period it increased faster toward the resting value to become higher than during recovery from the leg trial. Furthermore, with arm cranking the arterial-venous difference for O2, glucose, and lactate increased, as it did during leg exercise, but for glucose the arterial-venous difference decreased to lower levels in the recovery period (Fig. 2). In contrast, the arterial-venous difference for lactate was lower during arm cranking and in recovery as it increased from −0.1 ± 0.0 mM to the highest value of 0.3 ± 0.1 mM (P < 0.05). As with leg exercise, the cerebral metabolic ratio decreased from 6.6 ± 0.3 at rest to 5.0 ± 0.3 after 10 min of arm cranking, and it continued to decrease to a nadir of 4.7 ± 0.4 at exhaustion; i.e., it remained higher than during leg exercise (P < 0.05).

DISCUSSION

The main finding of this study is that exhaustive arm cranking reduced the cerebral metabolic ratio less than exhaustive leg cycling (4.7 vs. 3.3). A cerebral metabolic ratio of 4.7 at exhaustion is higher than that observed in other protocols of maximal exercise (Fig. 3). Also, the resting value before arm cranking (6.7) was larger than the commonly reported value of ~5.6 found in the group of subjects performing leg exercise, and it is explained by a lower arterial-venous difference for glucose before arm cranking. However, the cerebral metabolic ratio would be expected to decrease to an absolute value, rather than to one relative to the baseline, under the assumption that it corresponds to a specific level of neuronal activity. Judged by the nadir of the cerebral metabolic ratio, arm cranking seemingly stimulates the brain more than mental activity [Wisconsin card-sorting test (28)] and more than submaximal exercise (6, 7), which does not reduce the ratio significantly. However, a reduction in the cerebral metabolic ratio in the range 4.0–4.6 is noted during exercise that only partially stimulates the brain, e.g., as enhanced will to exercise with
little work carried out (6) or by the sensation of aggravated pain in ischemic muscles (7).

A low cerebral metabolic ratio incorporates metabolism in several brain regions, including those engaged not only with sensorimotor function during exercise, but also vision and maintenance of balance (10), control of ventilation (50), central regulation of cardiac function (53, 55), perception of effort (54), imagery of movement (30, 39), and even the intent to exercise (13). Because arm exercise would be expected to stimulate the brain more than leg exercise, as deduced from the larger cortical representation (37, 46, 56), increase in regional blood flow (25, 34, 46, 49), and enhanced central command (45, 52), metabolism could be attenuated in other brain regions. The notion of downregulation in nonactivated brain regions is advanced to account for a stable global cerebral blood flow and O\(_2\) consumption, while local values are elevated, e.g., during mental activity (21, 44) and in exercise (22, 29), although a global increase is observed during imagery of movement (39). Alternatively, some centrally controlled or integrated aspects of exercise were lower during arm cranking. For instance, the duration of exercise may have been so long that motivation became affected (2) before the subjects reached a “true” physical exhaustion, with the associated pain and requirements for a maximal mental effort. Thus it appears to be important that the subject is accustomed to the modality of exercise (35) and familiarized with the protocol (41) for performance to be “optimal” (47). On the other hand, familiarization with the experimental setting and/or an arithmetic test may abolish a previously observed increase in regional cerebral blood flow (21). Nevertheless, despite familiarization before the main study, the less familiar task of arm cranking, with its inherent demand for attention, may have hindered exhaustion from becoming as severe as during the (in Denmark) more practiced task of cycling.

Such differentiation of exhaustion elucidates not only why the cerebral metabolic response during arm cranking diverged from that during leg exercise but also why previous protocols reduce the ratio to a varying degree (Fig. 3). Incremental leg exercise (~12 min) in the somewhat awkward semisupine position reduces the cerebral metabolic ratio to 3.7–3.8 when subjects cannot overcome the resistance (6, 20). When termination of exercise becomes a matter of endurance, rather than the ability to overcome the resistance at a constant work rate in the same semirecumbent position, the decrease is more pronounced, and this is reproduced with a shorter duration of exercise with β-adrenergic receptor blockade (8). Accordingly, the brain may become maximally stimulated when athletes, who are accustomed to exercise at the verge of exhaustion, reach a limit beyond what can normally be achieved in non-athletes, and the ratio drops to ~3 (17).

It appears that the reduction of the cerebral metabolic ratio approaches a value close to 3 with the most intense exercise (Fig. 3). This is of interest because a value of 3 is predicted by

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**Fig. 2.** Arterial-venous differences (a-v diff) across the brain, cerebral metabolic ratio of O\(_2\) to (glucose + ½lactate), and perceived exertion (RPE) in response to exhaustive arm and leg exercise. RPE values are medians with 25th and 75th percentiles; other values are means ± SE. *Different from rest (for RPE, after 5 min of exercise), \(P < 0.05\). Different from leg exercise: †\(P < 0.05\); ‡\(P < 0.01\).
the “glycogen shunt hypothesis” for a brain area wherein the majority of the glucose consumed shuttles through astrocyte glycogen stores (42). Observations that conform to this hypothesis include the finding that neuronal activity lowers the glycogen level (26), especially when neuronal activity is intense (5), whereas the local metabolic ratio of O$_2$ to glucose concomitantly is reduced to ~3 as calculated from the visual cortex (14). Furthermore, the hypothesis is compatible with the observation that, during neuronal activity, ~75% of glucose is metabolized in the glial compartment (51), even though, in the brain, neuronal signaling accounts for the majority of the energy production (3). However, the glycogen shutt also infers a net degradation of glycogen, which adds to the quantity of the surplus carbohydrate taken up by the brain relative to that of O$_2$ that needs to be accounted for. Additionally, the premise for the calculation of the lowest cerebral metabolic ratio of 3 is that lactate is released from the astrocytes, perhaps to be taken up and oxidized in neurons (36). Such trafficking is likely to result in intracerebral lactate accumulation, as confirmed during the first ~5–10 min after physiological stimulation of the brain, which maintains a low arterial lactate concentration (19, 38, 40), and, under these conditions, lactate may efflux from the brain (27, 28). Conversely, exercise raises arterial lactate and leads to a substantial cerebral uptake of lactate (6, 17, 20) that is most likely metabolized (43), inasmuch as lactate does not accumulate in the cerebrospinal fluid or within the brain, as determined by magnetic resonance spectroscopy of the relevant sensorimotor area (9). As in humans, sensory stimulation of the rat may induce a cerebral lactate uptake secondary to a slight increase in arterial lactate [due to movement (11)], although, at least in the anesthetized rat, it is not certain whether the lactate taken up is metabolized (24).

During arm cranking the arterial-venous difference for lactate was only ~65% of that observed during leg exercise, even though the arterial lactate concentration tended to be lower by only ~20%. The lower arterial-venous difference for lactate during arm cranking seems not to be explained by enhanced global cerebral blood flow, because P$_{ACO_2}$ values were equal in the two conditions. Consequently, such divergence in the arterial-venous difference for lactate between trials, despite comparable arterial concentration (Fig. 4), may be explained by the level of cerebral activity. If exercise-induced brain activity is reflected by the magnitude of the reduction in the cerebral metabolic ratio, the combination of a large reduction in the cerebral metabolic ratio and a larger arterial-venous difference for lactate during leg exercise compared with arm cranking supports the notion that lactate is taken up by the brain when the “lactate shuttle” between astrocytes and neurons is operative (5), i.e., during neuronal activation (23, 36). Such a coupling between brain activity and lactate uptake is indicated also within trials. The arterial-venous difference for lactate was lower during the recovery period (where cerebral activity would be expected to be low) than during exercise (where cerebral activity would be expected to be high) in a comparison at equal arterial lactate concentrations (Fig. 4). However, discrete regions within the brain, e.g., those involved with arm vs. leg exercise, may express a different metabolic capacity for lactate. As an incidental observation, establishment of a coupling between the arterial concentra-

Fig. 3. Reduction of cerebral metabolic ratio in response to exercise- and non-exercise-induced brain activation. Protocols of cerebral activation are ranked according to the lowest value for the cerebral metabolic ratio of O$_2$(glucose + ½ lactate) (bottom) and presented with the corresponding ratio of O$_2$ to glucose (top). Dashed horizontal line denotes cerebral metabolic ratio of 6, where glucose taken up by the brain is completely oxidized. Rest, awake, mean across the presented protocols; visual cortex, metabolic changes in visual cortex upon visual stimulation (15); max leg + heat, 6 min, maximal leg exercise for ~6 min in hyperthermia (17); max leg + arm, 12 min, incremental exercise with legs and arms (9); max leg, 25 min, maximal leg exercise for ~25 min (8); max leg, 12 min, incremental leg exercise in semisupine position for ~12 min (6, 20); max leg + heat, 60 min, maximal cycling for 1 h in hyperthermia on a regular ergometer (33); leg + thigh cuffs, 10 min, exercise at a light workload made difficult by ~100-mmHg thigh cuffs to increase sensory input to the brain and central command to exercise; leg, 10 min + PEMI, 5 min, exercise at a light workload followed by 5 min of postexercise muscle ischemia to selectively increase sensory stimulation (7); leg + curare, 10 min, exercise at a light workload made difficult by neuromuscular blockade to selectively enhance central command (6); max arm, 35 min, present study; submax, 10 min, average value from 4 bouts of 10-min exercise at light-to-moderate intensity (6, 7); mental, 20 min, mental and visual stimulation (28). Values are mean ± SE (SD for mental, 20 min). Different from respective resting values: *P < 0.05; †P < 0.01.
tion of lactate and its arterial-venous difference poses the risk of a spurious correlation only if the venous concentration of lactate remains constant, despite changes in the arterial content, whereby the arterial-venous difference for lactate would increase simply because of increasing arterial lactate. Importantly, however, this is not the case for the arterial-venous difference for lactate, inasmuch as, indeed, the concentration in the internal jugular vein changes as a function of the combined effect of the arterial lactate concentration, brain metabolism, and blood flow. Clearly, this is demonstrated for glucose, where widely different arterial concentrations yield an identical arterial-venous difference; i.e., a coupling is not substantiated.

As opposed to considering brain lactate uptake as a function of cerebral activity, it may be relevant to consider whether the reduction in the cerebral metabolic ratio is influenced by the lactate uptake per se. Not only in the present study but also in other protocols, the cerebral metabolic ratio of O2 to glucose has been reported to remain stable, whereas the ratio of O2 to (glucose + 1/2lactate) decreases (7). Yet the uptake ratio of O2 to glucose for the brain as a whole decreases during pure mental activation (28) and during prolonged exercise, where plasma lactate stays low (33). Thus the cerebral metabolic ratio decreases when brain activity increases, and, when available, lactate appears to substitute for glucose as an energy source (43). In contrast to glucose, lactate cannot maintain glycogen levels in cultures of astrocytes (5). Moreover, lactate is readily converted to pyruvate and is probably oxidized, and both processes produce metabolites and energy-rich compounds that inhibit glycolytic breakdown of glucose (11). Thus the larger the lactate uptake by the brain, the larger is the fraction of the concomitant glucose that may be spared for other metabolic purposes, e.g., storage as glycogen.

Cerebral metabolism may change over time during sustained cerebral stimulation. Although glycogenolysis (12) and glycolysis (19, 40) seem to support energy production, especially in the initial phases of neuronal activity, oxidative metabolism increases at later stages (15, 16, 31). A slowly responding oxidative metabolism may explain why the cerebral metabolic ratio decreases to only 5.3 in well-trained subjects who exercise for 1 h under highly specified conditions, although making the task difficult by addition of hyperthermia exacerbates the reduction to 3.8 (33). If exercise is continued even longer, i.e., for 3 h, the cerebral metabolic ratio remains stable for as long as blood glucose is maintained by supplementation, but when blood glucose and, in turn, its arterial-venous difference over the brain decreases, the ratio tends to increase (32).

The cerebral metabolic ratio is regularly reduced close to the time of exhaustion, whereas it was already decreased after 10 min of exercise with the arms or the legs. We used a heavier workload than those used in protocols of submaximal exercise demonstrating a stable cerebral metabolic ratio (6, 7), and the early reduction in the cerebral metabolic ratio reinforces the notion that it is reflective of a determined effort. Expressed in terms of RPE, the cerebral metabolic ratio decreased at an intensity that was graded as “moderately hard” for arm cranking and “very hard” for leg exercise (Fig. 2).

Arm exercise demonstrated that the cerebral metabolic ratio integrates metabolism in distinct brain areas and that its reduction as such is intrinsic to the cerebral metabolism may change over time during sustained cerebral stimulation. Although glycogenolysis (12) and glycolysis (19, 40) seem to support energy production, especially in the initial phases of neuronal activity, oxidative metabolism increases at later stages (15, 16, 31). A slowly responding oxidative metabolism may explain why the cerebral metabolic ratio decreases to only 5.3 in well-trained subjects who exercise for 1 h under highly specified conditions, although making the task difficult by addition of hyperthermia exacerbates the reduction to 3.8 (33). If exercise is continued even longer, i.e., for 3 h, the cerebral metabolic ratio remains stable for as long as blood glucose is maintained by supplementation, but when blood glucose and, in turn, its arterial-venous difference over the brain decreases, the ratio tends to increase (32).

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