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Estimation of Aerobic and Anaerobic Metabolism in Isometric Forearm Exercise

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ABSTRACT

The aim was to evaluate the energy turnover in the forearm during isometric submaximal exercise. Eight subjects performed isometric handgrip contractions until exhaustion at 10%, 25% and 40% of maximal voluntary contraction (MVC). Blood samples were drawn frequently from the cubital vein of the exercising arm during exercise and a 60 minutes recovery period, and forearm blood flow (plethysmography) was monitored during the same periods. Oxygen uptake and lactate release were used to quantify aerobic and anaerobic energy expenditure. Electromyography (frequency analysis) was monitored during exercise. Anaerobic glycolysis contributed 4%, 31% and 37% of the ATP production in the three experiments, respectively, but total ATP production from anaerobic glycolysis was the same, suggesting a maximal anaerobic glycolytic capacity (MAGC). MAGC may be a limiting factor for muscle performance at 25 and 40% MVC, but not at 10% MVC. Judging from the match between oxygen debt or estimated oxygen deficit vs the anaerobic energy production, the muscles recovered during 60 min rest after 10% MVC and 40% MVC. However, recovery after 25% MVC may have been incomplete.

INTRODUCTION

The local muscle metabolism in high intensity contractions depends to a large extent on the recruitment of fast glycolytic fibers and has been thoroughly investigated. Thus it has been proposed that the development of fatigue is associated with accumulation of metabolites (15). Also, depletion

of substrates (14) may be responsible for loss of force at intense exercise, as well as an interaction between substrate depletion and metabolite accumulation (31).

The situation in low intensity exercise is rather different due to recruitment of predominately slow oxidative fibers, which requires a sufficient oxygen supply. It has been suggested that the oxygen supply is always sufficient since venous oxygen pressure has not been observed below 10 mm Hg (24), a level high enough for oxygen extraction. Conversely, oxygen supply may be limiting for muscle performance since the mitochondrial respiratory capacity exceeds that which could be achieved with maximal oxygen delivery (11). Veerkamp and Paulussen (28) claimed that availability of free fatty acids may be limiting. In work situations with even lower contraction intensities other factors such as electrolyte homeostasis (7), muscle pain (13) and motivation (6) may influence the generation of force.

The mechanisms responsible for loss of force at low intensity contractions may vary depending on the type of exercise and muscles involved. Many jobs today include prolonged sequences of isometric, submaximal forearm contractions but little is known about this from the energy-turnover point of view, including the recovery period. This study was designed to evaluate the energy expenditure during and after different types of prolonged isometric, submaximal forearm exercise.

METHODS

Subjects

Eight healthy subjects volunteered in the study (4F + 4M, age 23-34 years) which was part of a project approved by the Ethical Committe of the Karolinska Institute, Stockholm.

Protocol

Three types of experiments were performed: isometric handgrip until exhaustion at 10% MVC (Maximal Voluntary Contraction), 25% MVC and 40% MVC. Exhaustion was defined as when the subject was unable to continue at the target force. In the 10% MVC experiment, contractions were held until exhaustion or 60 min, whichever came first. Recovery was followed for 60 min.

Procedures

In a sitting position the subject performed isometric handgrip on an adjustable strain-gauge dynamometer. The forearm was horizontal and firmly attached to supports at the elbow and wrist. Elbow angle was 115 degr. After insertion of the intravascular catheter, blood samples were collected and resting bloodflow and MVC were measured. After a subsequent 15 min rest, electromyographic (EMG) signals were recorded during four 10 s testcontractions at the same

intensity as the subsequent exercise. EMG-signals were recorded continuously during exercise. Bloodflow was measured and blood samples drawn frequently during exercise and recovery at intervals shown in figure 1. Room temperature was air-conditioned to 22° C.

An ethylene catheter (Venflon 1.2) was inserted into a deep cubital vein of the exercising arm and advanced 3-5 cm in the upstream direction. Samples of blood withdrawn from a catheter so placed have been shown to come from the muscles (8). The catheter was kept patent by repeated injections of 2 ml heparinized saline solution.

Forearm blood flow (BF) was measured by strain-gauge plethysmography (32) with a cuff pressure of 52 mm Hg (Elektromedicin HB, Göteborg, Sweden 1987). The pneumatic cuff was placed distally on the upper arm. Arterial occlusion of the wrist was not employed since it has been shown not to affect forearm blood flow neither during sustained nor intermittent isometric contractions (33). BF was measured during contraction, which gives reliable measurements provided there is no fatigue tremor (4). Skin BF was not measured since forearm BF consists of about equal contributions from skin and muscle at rest (10), and skin BF has been shown not to increase more than about 24% from resting values during exercise at normothermic conditions (25).

Venous blood oxygen saturation (O_{2sat}), hemoglobin concentration [Hb] and pH were determined spectrophotometrically (ABL2, Acid-Base Laboratory Radiometer, Copenhagen). Forearm oxygen uptake (VO_2) was calculated as [Hb] x ΔO_{2sat} x 1.34 x BF. Arterial O_{2sat} was assumed to keep steady at a set value of 97%, since the forearm comprises a minor part of the total muscle volume. $\Delta O_{2sat}$was derived from 97% - venous O_{2sat}. The oxygen content of myoglobin was neglected since its contribution to total forearm oxygen uptake was calculated to constitute a maximum of 1% in the 10% MVC experiment and 2.5% in the 40% MVC experiment, based on 5mg myoglobin x g^{-1} w.w. muscle at rest. Adenosinetriphosphate (ATP) production from oxidative processes was calculated from 1 mmol $O_2 \approx$ 6.34 mmol ATP (27).

Venous blood lactate concentration $[La^-]_v$ was determined by a fluorometric assay (17). Lactate release (LR) was calculated as BF x $\Delta[La^-]_v$ x time. $\Delta[La^-]_v$ was calculated as the difference between $[La^-]_v$ at rest and the values obtained during exercise and recovery (21). It was assumed that $[La^-]_v$ at rest equaled arterial $[La^-]$ and that no significant change in arterial $[La^-]$ occurred during exercise (22) since the forearm comprises a minor part of the total muscle volume. ATP production from anaerobic glycolysis was calculated as 1 mmol $La^- \approx$ 1.5 mmol ATP (1).

The estimated oxygen deficit can be obtained from the calculated oxygen demand during exercise minus the actual (measured) oxygen uptake. The problem is to determine the oxygen demand during

isometric exercise. In situations with restricted blood flow (isometric contractions), the oxygen demand may therefore be determined by measuring the oxygen uptake during dynamic exercise with similar endurance time. Hartling et al (13) measured the oxygen uptake during dynamic forearm contractions at 40-90% of maximal work load. The time to exhaustion was about 10 min. Considering the higher bridge cycling rates (up to twice the metabolic rate depending on fibre type recruitment) in dynamic exercise (3), 10 min endurance time for dynamic exercise corresponds to the isometric endurance times in this study of about 8 min at 25% MVC and 3 min at 40% MVC. The peak oxygen uptake is in the order of 0.2 mmol x min^{-1} x $100g^{-1}$ (13). This figure may therefore be used, and the estimated oxygen deficit for the experiments could be calculated.

The oxygen debt was calculated from the measured oxygen uptake during recovery minus resting uptake.

EMG signals were collected from the belly of the extensor digitorum muscle by two 5 mm-electrodes (Medicotest E-05-VS) separated by 15 mm in the muscle fibre direction. Before the electrodes were placed on the forearm the skin was shaved and carefully cleansed with 70% alcohol. Analysis was done by frequency-analysis (zero-crossing technique). The zero-crossing technique has been shown to have similar properties concerning muscle fatigue as other more frequently used techniques (16). Before each exercise bout, the subject performed 4 test-contractions at the same contraction intensity as the subsequent exercise, and values obtained during exercise were normalized against these.

Statistical analysis was performed by a one-way analysis of variance. $p < 0.05$ was considered statistically significant.

RESULTS

Endurance times were 53.6 (SE 3.4), 7.6 (SE 0.6) and 2.7 (SE 0.2) min at 10, 25 and 40% MVC, respectively. At 10% MVC, only three subjects were able to continue exercise until 60 min.

BF increased five-fold to about 20 ml x min^{-1} x $100g^{-1}$ in all experiments and increased further to about 30 ml x min^{-1} x $100g^{-1}$ at 25% MVC (figure 1). Flows up to 35-40 ml x min^{-1} x $100g^{-1}$ were registered in some subjects. Resting BFs were obtained after about 20 min.

VO_2 increased continuously and seemed to follow forearm BF in all experiments, failing to show a steady state, except for possibly in the later part of the 10% MVC experiment (figure 1). Recovery was quick (2 min) after the 10% MVC experiment. After 25 and 40% MVC recovery was complete in 20-30 min.

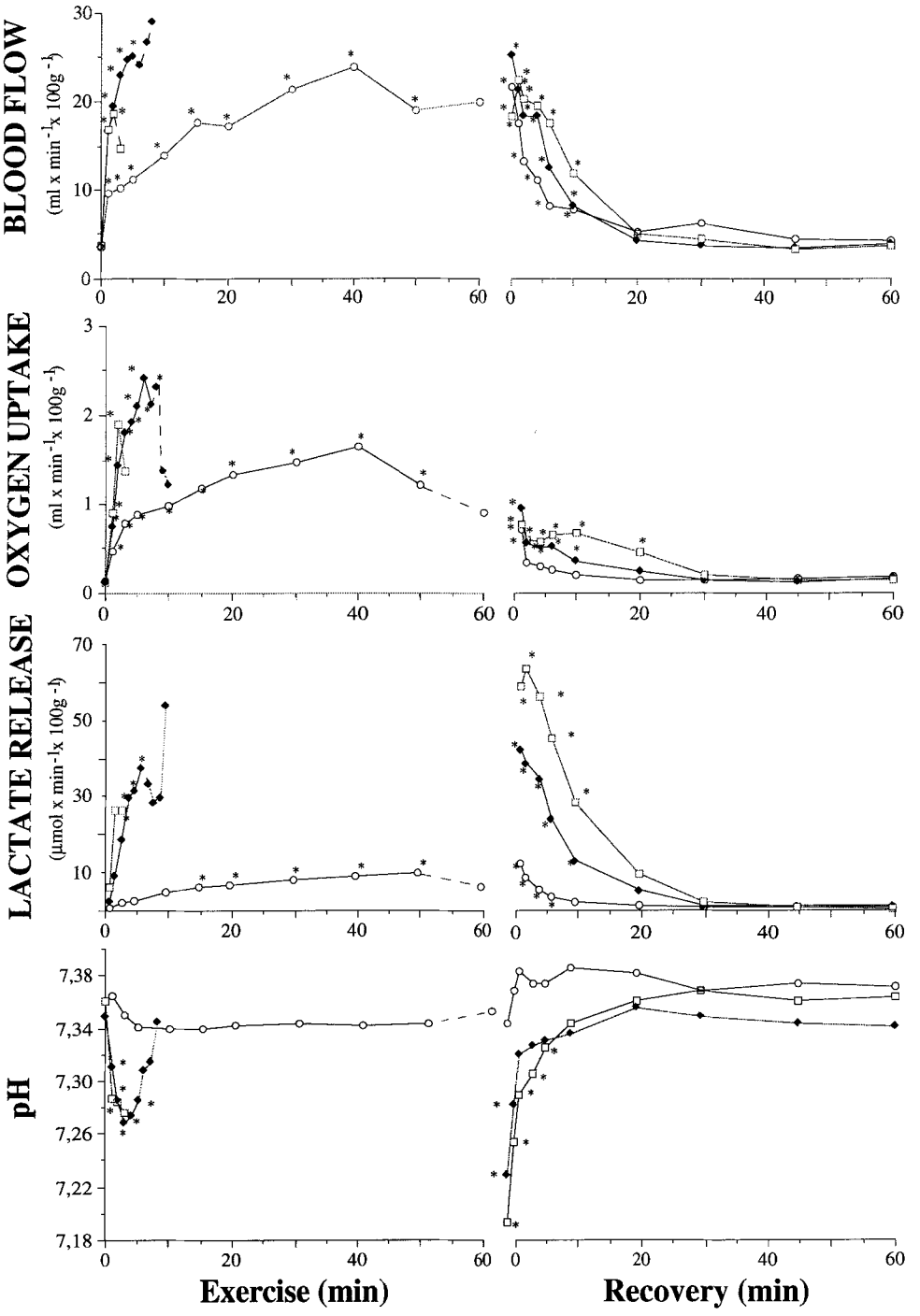

Fig 1. Results from local forearm blood flow and analysis of the venous blood from v.cubiti during exercise and recovery. Full lines: ≥4 subjects. Dotted lines: ≤3 subjects. denotes values significantly different from resting value. Top: Blood flow in the forearm. There was a continous increase even at10% MVC. Resting values were obtained after 20 min of recovery in all experiments. Second from top: Oxygen uptake as determined by changes in O2sat of the forearm venous effluent and forearm blood flow. Third from top: Lactate release as determined by changes in [La-]v of the forearm venous effluent and forearm blood flow. Bottom: [pH] in the forearm venous effluent. Values from the10% MVC experiment did not differ from resting values.*

*Table 1. Summary of the aerobic and anaerobic energy yield (±SE) in the forearm during the exhaustive handgrip contractions. For calculations, see Methods and Results. * Value adapted from data by Spriet et al 1987 and Vøllestad et al 1988.*

	10% MVC	25% MVC	40% MVC
Oxygen uptake (exercise)			
mmol x $100g^{-1}$	2.7 (0.22)	0.8 (0.20)	0.6 (0.17)
mmol ATP x $100g^{-1}$ (equvivalents)	17.1 (1.41)	4.9 (1.25)	3.9 (1.06)
Anaerobic energy yield (lactate release)			
mmol x $100g^{-1}$	0.5 (0.15)	0.6 (0.10)	0.7 (0.10)
mmol ATP x $100g^{-1}$ (equvivalents)	0.7 (0.23)	1.0 (0.17)	1.1 (0.15)
Anaerobic energy yield (ATP and PCr)			
mmol ATP x $100g^{-1}$ (equvivalents)	---	1.2 *	1.2 *
Total anaerobic energy yield			
mmol ATP x $100g^{-1}$ (equvivalents)	0.7 (0.23)	2.2 (0.17)	2.3 (0.15)
Oxygen debt			
mmol ATP x $100g^{-1}$ (equvivalents)	0.7 (0.52)	1.7 (0.84)	3.1 (0.99)
Estimated oxygen deficit			
mmol ATP x $100g^{-1}$ (equvivalents)	---	6.2 (0.93)	2.5 (0.36)

$[La^-]_V$ increased slightly from a resting value of 0.8 mM to a steady state of about 1.2 mM in the 10% MVC experiment. In the other two experiments $[La^-]_V$ increased progressively until exhaustion with peak values of 3.2 and 2.9 mM at 25 and 40% MVC, respectively. $[La^-]_V$ returned to resting level within 15-30 min in all experiments. LR showed a similar path (figure 1). Surprisingly, the total amount of lactate released during exercise and recovery did not differ significantly between the various types of exercises (table 1).

The change in pH_V at 10% MVC was negligible. At 25 and 40% MVC there was a steep decrease which recovered quickly (≤10 min) after exercise (figure 1).

Energy utilized during the experiments including the energy equivalents of the calculated oxygen debt and oxygen deficit are shown in table 1 and figure 2.

The degree of fatigue development during exercise, as reflected by the number of zero-crossings (EMG), are shown in figure 3.

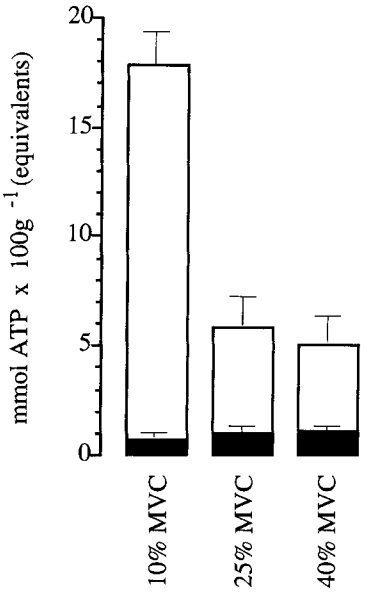

Fig 2. Relation between energy derived by aerobic (white) and anaerobic (black) processes. The absolute valueas are shown +SE. There was no difference between the three experiments in the estimated amount of energy derived by anaerobic glycolysis.

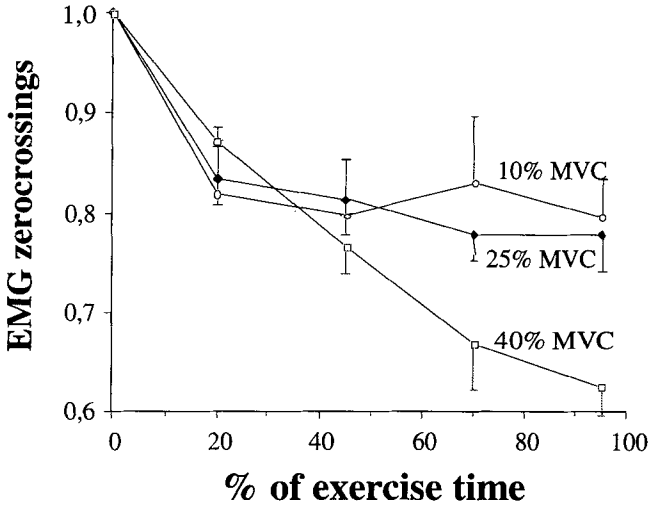

Fig 3. Number of zero-crossings (EMG) during the handgrip contractions, which may be used as an indirect measure of the fatigue state of the forearm muscles. All values normalized to three 10 s pre-exercise test-contractions. The decrease was continous only at 40% MVC, indicating progressive fatigue.

DISCUSSION

Forearm oxygen uptake showed values of the same order (1-2 ml x min^{-1} x $100g^{-1}$) as has been demonstrated before during similar isometric (2) and dynamic (13) exercise. In the 10% MVC experiment, no plateau but a slight and continuous increase in VO_2 could be identified although not

significant after 5 min (figure 1). Judging from other studies this was not expected. An increase in skin oxygen uptake during contraction does probably not contribute since no increase in skin metabolic demands is likely. Also, the skin veins drain proximal to the catheter tip, and no significant blood flow increase from the hand could be expected. Therefore, the increase in oxygen uptake is due to processes located within the forearm muscles and may reflect a possible increased cost of contraction, as recently suggested for intermittent isometric contractions (29).

There was a significant release of lactate throughout all experiments (figure 1). This was the case also at 10% MVC, indicating the forearm not to be content with its oxygen supply. Alternatively, the oxidative processes were insufficient. This is in line with previous results showing that BF in the forearm at 10% MVC isometric contraction is insufficient (5).

Estimation of the total lactate release is based on the assumption that lactate release during exercise and recovery equals lactate formed in the muscles. Because $[La^-]_V$ was followed frequently until it had returned to resting values, all lactate released from the muscles could be traced as it passed out in the veins. However, some lactate was probably oxidized in the muscles before it was ever released into the blood. This must result in a slight underestimation of the lactate release. Bangsbo et al (1), using muscle biopsies and venous blood samples, found that 70% of the lactate accumulated by the end of intense exercise with m.quadriceps (similar fiber-composition as the forearm) was washed out in the post-exercise period. The remaining 30% represents an estimation of the amount of lactate oxidized in the muscles. Therefore, lactate release during recovery in this study has been roughly corrected according to this figure. It could also be argued that since arterial $[La^-]$ was not measured, no confident v-a difference could be obtained. However, the forearm is too small to cause an increase in arterial $[La^-]$, except in situations of severe exercise. This is supported by results obtained by Reddy et al (22), who did not find elevated $[La^-]_V$ in the non-exercising forearm during 6 min of 25% MVC isometric arm exercise, and similar results have been obtained previously (6). Based on the above, total lactate release during exercise and recovery (corrected for the estimated amount oxidized in the muscles during exercise) can be converted to ATP equivalents and function as a measure of the anaerobic glycolysis.

The anaerobic metabolism must also include contributions from ATP and phosphocreatine (PCr) stored in the muscle cells. Since no great amount of lactate was accumulated during 10% MVC it is not likely that stored ATP and PCr were ever used to a significant degree (12). Even though ATP and PCr stored in the cells have a very limited capacity, they may be significant contributors to energy production in short and intense exercise such as in the 25 and 40% MVC experiments. During 30% MVC intermittent isometric exercise until exhaustion with the quadriceps, Vøllestad et

al (30) found that ATP decreased little and that PCr was depleted by some 70-80% at exhaustion. Similar figures have been obtained during isometric exercise by others (23). Using the results from these studies, energy derived from stored high energy phosphates can be calculated to correspond to 1.2 mmol ATP x $100g^{-1}$ (equivalents) in both the 25 and 40% MVC experiments (table 1), corresponding to a depletion of 4 mmol x kg^{-1} w.w. of ATP and 10-12 mmol x kg^{-1} w.w. of PCr at the point of exhaustion.

Energy derived by anaerobic glycolysis until exhaustion amounted to about 4, 31 and 37% of the total ATP turnover and was approximately the same (0.7-1.1 mmol ATP · $100g^{-1}$), in the 10, 25 and 40% MVC experiments, respectively (figure 2). The lack of statistical difference could possibly be an effect of the small number of observations. It may however be hypothesized that the forearm muscles have a maximal anaerobic glycolytic capacity (MAGC) in the order of 1 mmol ATP· $100g^{-1}$, which is used during the course to exhaustion. However, this figure could probably vary since there are considerable differences in the cost of contraction depending on the recruitment pattern for different types of contraction (3), as well as differences in fiber composition of various muscles. When MAGC is reached, contraction has to cease because the capacity of the high energy phosphates is very limited. Depletion of glycogen is unlikely as a cause to the drop in force at exhaustion since increasing evidence show that no more than 25-40% of the muscle glycogen can be depleted during single contractions (20). On the other hand, Danforth (9) showed that low muscle glycogen results in low glycolytic enzyme activity. Also, an inhibition of phosphofructokinase (26) is likely, or a combination of the two latter mechanisms (18). The result will be a severe shortage of ATP. Other mechanisms related to the accumulation of metabolites may be responsible for exhaustion as well (18). These mechanisms would to some extent require a decreased pH, and this was not observed at 10% MVC (figure 1). Thus, MAGC does not seem to be a limiting factor for the force-generating capacity at 10% MVC, but could indeed be so at 25 and 40% MVC. Further invasive studies are needed to verify this hypothesis.

An interesting question is whether metabolic homeostasis was restored or not in the recovery period of the exhaustive contractions. This may be answered by studying how the oxygen debt and/or the oxygen deficit match the anaerobic energy production (mainly through changes in lactate, ATP and PCr). The oxygen debt is known to produce erroneously high estimates of the anaerobic energy liberation due to restrictions in blood supply (19). Accordingly, at 40% MVC the energy equivalent of the oxygen debt was higher than the anaerobic energy production; however the procedure may be justified at 10% MVC. The energy equivalent of the oxygen debt corresponded well to the anaerobic energy production at 10% MVC (table 1), indicating full recovery.

The estimated oxygen deficit is known to be in better accord with the anaerobic energy production at intense contractions with a small muscle group (1). This may be explained by the fact that at intense contractions, blood supply is highly insufficient and the measured oxygen uptake during exercise therefore will be much below the actual need of the muscles. Hence, the estimated oxygen deficit seems to be better match the anaerobic energy production than the oxygen debt at intense contractions. At 40% MVC, the energy equivalent of the oxygen deficit corresponded rather well to the anaerobic energy production (table 1), suggesting full recovery.

Thus, metabolic homeostasis seemed to be restored in the recovery period after the 10 and 40% MVC contractions. This may not be the case after 25% MVC, since the estimated oxygen deficit was rather high due to the relatively long endurance time and insufficient blood flow. The conclusion that metabolic homeostasis was restored in the recovery period after 25% MVC is tempting, since the oxygen debt roughly corresponded to the anaerobic energy production. This is probably incorrect because blood supply is insufficient at this contraction intensity (5) and the oxygen debt will therefore be too small. Since the methods used in this study do not have the accuracy to measure small changes in oxygen uptake, it could well be that there was a small and prolonged oxygen uptake in the recovery after 25% MVC eventually restoring metabolic homeostasis.

At 10% MVC, exercise could be performed on a steady-state basis with regard to lactate release and pH_v. Also, fatigue development as reflected by the EMG (number of zero-crossings) was negligble and post-exercise oxygen consumption was minimal. Therefore, the development of fatigue at 10% MVC must rely on other mechanisms than at 25 and 40% MVC. This could include a redistribution of potassium released from the exercising muscles during low intensity contractions as recently suggested (7), especially during prolonged contractions as in this protocol.

In conclusion, the results of this study show that the forearm muscles may have a maximal anaerobic glycolytic capacity during isometric contractions, and this may limit muscle performance for contractions ≥25% MVC. The oxygen debt corresponds to anaerobic energy production at 10% MVC, but the estimated oxygen deficit is better suited for situations with restricted blood supply (intense isometric exercise). Metabolic homeostasis seemed to be restored in the recovery period after 10 and 40% MVC, but a longer recovery period may be needed after 25% MVC since the estimated oxygen deficit in this experiment was large. Further, only contractions at 10% MVC could be performed on a steady state basis with regard to the parameters employed in this study.

REFERENCES

1. Bangsbo, J., Gollnick, P.D., Graham, T.E., Juel, C., Kiens, B., Mizuno, P. & Saltin, B.: Anaerobic energy production and O2 deficit-debt relationship during exhaustive exercise in humans. J Physiol 422: 539-559, 1990.
2. Barcroft, H., Greenwood, B. & Whelan, R.F.: Blood flow and venous oxygen saturation during sustained contraction of the forearm. J Physiol 168: 848-856, 1963.
3. Bolstad, G. & Ersland, A.: Energy metabolism in different human skeletal muscles during voluntary isometric contractions. Eur J Appl Physiol 38: 171-179, 1978.
4. Byström, S.: Physiological response and acceptability of isometric intermittent handgrip contractions. Thesis, Karolinska Institute, Stockholm, 1991.
5. Byström, S. & Kilbom, Å.: Physiological response in the forearm during and after isometric intermittent handgrip. Eur J Appl Physiol 60: 457-466, 1990.
6. Byström, S., Mathiassen, S.E.M. & Fransson-Hall, C.: Physiological effects of micropauses in isometric handgrip exercise. Eur J Appl Physiol 63: 405-411, 1991.
7. Byström, S. & Sjøgaard, G.: Potassium homeostasis during and following exhaustive submaximal static handgrip contractions. Acta Physiol Scand 142: 59-66, 1991.
8. Coles, D.R., Cooper, K.E., Mottram, R.F. & Occleshaw, J.V.: The source of blood samples withdrawn from deep forearm veins via catheters passed upstream from the median cubital vein. J Physiol 142: 323-328, 1958.
9. Danforth, W.H.: Activation of the glycolytic pathway in muscle. *In* B. Chance & R.W. Estabrook (ed.). Control of energy metabolism. New York, Academic Press, 1965, pp. 287-298.
10. Detry, J.M., Brengelman, G.L., Rowell, L.B. & Wyss, C.: Skin and muscle components of forearm blood flow in directly heated resting man. J Appl Physiol 32: 506-511, 1972.
11. Gollnick, P.D., Armstrong, R.B., Saubert, C.W., Piehl, K. & Saltin, B.: Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. J Appl Physiol 33(3): 312-319, 1972.
12. Harris, R.C., Sahlin, K. & Hultman, E.: Phosphagen and lactate contents of m. quadriceps femoris of man after exercise. J Appl Physiol 43(5): 852-857, 1977.
13. Hartling, O.J., Kelbaeck, H., Gjørup, T., Schibye, B., Klausen, K. & Trap-Jensen, J.: Forearm oxygen uptake during maximal forearm dynamic exercise. Eur J Appl Physiol 58: 466-470, 1989.
14. Hermansen, L., Hultman, E. & Saltin, B.: Muscle glycogen during prolonged severe exercise. Acta Physiol Scand 71: 129-139, 1967.
15. Hibberd, M.G., Dantzig, J.A., Trentham, D.R. & Goldman, Y.E.: Phosphate release and force generation in skeletal muscle fibers. Science 228: 1317-1319, 1985.
16. Hägg, G.M.: Zero crossing rate as an index of electromyographic spectral alterations and its applications to ergonomics. Thesis, Chalmers University of Technology, 1991.
17. Lowry, O.H. & Passonneau, J.V.: A flexible system of enzymatic analysis. New York, Academic, 1972.
18. MacLaren, D.P.M., Gibson, H., Parry-Billings, M. & Edwards, R.H.T.: A Review of metabolic and physiological factors in fatigue. Ex Sport Sci Rev 17: 29-67, 1989.
19. Margaria, R., Cerretelli, P., Di Prampero, P.E., Massari, C. & Torelli, G.: Kinetics and mechanism of oxygen debt contraction in man. J Appl Physiol 18: 371-377, 1963.
20. Maughan, R.J. & Poole, D.C.: The effects of glycogen-loading regimen on the capacity to perform anaerobic exercise. Eur J Appl Physiol 46: 211-219, 1981.
21. Pendergast, D., Cerretelli, P. & Rennie, W.: Aerobic and glycolytic metabolism in arm exercise. J Appl Physiol 47(4): 754-760, 1979.
22. Reddy, H.K., Weber, K., Janicki, J. & Mc Elroy, P.: Hemodynamic, ventilatory and metabolic effects of light isometric exercise in patients with chronic heart failure. J Am Coll Cardiol 12: 353-358, 1988.

23. Spriet, L.L., Söderlund, K., Bergström, M. & Hultman, E.: Anaerobic energy release in skeletal muscle during electrical stimulation in men. J Appl Physiol 62(2): 611-615, 1987.
24. Stainsby, W.N. & Otis, A.B.: Blood flow, blood oxygen tension, oxygen uptake and oxygen transport in skeletal muscle. Am J Physiol 206: 858-866, 1964.
25. Taylor, W.F., Johnson, J.M., Kosiba, W.A. & Kwan, C.M.: Cutaneous vascular responses to isometric handgrip exercise. J Appl Physiol 66(4): 1586-92, 1989.
26. Ui, M.: Arole of phosphofructokinase in pH dependent regulation of glycolysis. Biocheim Biophys Acta 124: 310-322, 1966.
27. Vander, A.J., Sherman, J.H. & Luciano, D.S.: Human Physiology: The mechanisms of body function. 4 ed. New York, McGraw-Hill, 1986.
28. Veerkamp, J.H. & Paulussen, R.J.A.: Fatty acid transport in muscle: the role of fatty acid binding proteins. Biochem Soc Trans 15: 331-336, 1987.
29. Vøllestad, N., Wesche, J. & Sejersted, O.: Gradual increase in leg oxygen uptake during repeated submaximal contractions in humans. Eur J Appl Physiol 68(4): 1150-1156, 1990.
30. Vøllestad, N.K., Sejersted, O.M., Bahr, R., Woods, J.J. & Bigland, R.B.: Motor drive and metabolic responses during repeated submaximal contractions in humans. J Appl Physiol 64(4): 1421-7, 1988.
31. Wenger, H.A. & Reed, A.T.: Metabolic factors associated with muscular fatigue during aerobic and anaerobic work. Can J Appl Sports Sci 1: 43-48, 1976.
32. Whitney, R.J.: The measurement of volume changes in human limbs. J Physiol (Lond) 121: 1-27, 1953.
33. Williams, C.A. & Lind, A.R.: Measurement of forearm blood flow by venous occlusion plethysmography: influence of hand blood flow during sustained and intermittent isometric exercise. Eur J Appl Physiol 42(3): 141-9, 1979.

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