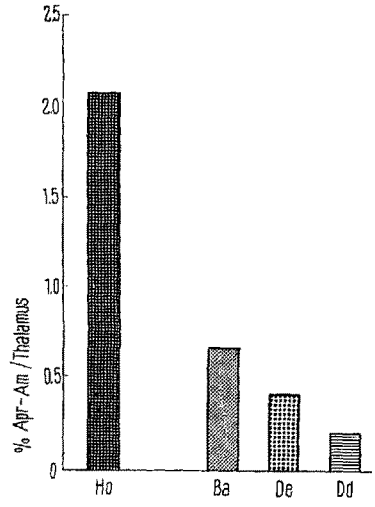

Fig. 1. Relative portion of volume of the Nucleus anterior principalis and medialis in the total thalamus. Ba, *Balaenoptera physalus*, Dd, *Delphinus delphis*, De, *Delphinapterus leucas*, Ho, Homo.

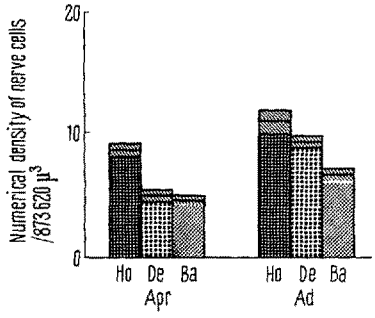

Fig. 2. Numerical density of nerve cells of the Nucleus anterior principalis (Apr) and anterodorsalis (Ad).

mately $^1/_5$ of the percentage value of the human brain (2.15%) (Table I). The same is true of the main nucleus (Apr-Am) although it forms the bulk of the Nucleus anterior thalami (Figure 1). The Nucleus anterodorsalis (Ad) is not very well developed. *Delphinapterus* has the greatest volume, 0.086%, which compares approximately with the percentage of the Ad of the human brain. The other 2 Cetacea have only 0.014 and 0.0004%.

The numerical density of both the anterior nuclei (Apr and Ad) are less dense than in the human brain (Figure 2). The reduction in the number of cells is, however, somewhat compensated by the size of the individual cells. This fact can also be seen in the voluminal percentages, which are approximately the same as those of the human brain (Table II).

The glial cells of both anterior nuclei are more dense than in the human brain. HAWKINS and OLSZEWSKI[7] also found that the glia/neuron index is higher in the cortex of *Balaenoptera physalus* than in man. They believe that this is caused by the size of the brain.

Zusammenfassung. Der Thalamus – insbesondere der Nucleus anterior thalami – einiger Cetacea wird architektonisch und volumetrisch untersucht. Die Ergebnisse werden mit den Befunden beim Menschen verglichen. Der Nucleus anterior thalami ist bei den untersuchten Cetacea stark reduziert. Die geringere numerische Nervenzelldichte der Cetacea wird durch das grössere Volumen ihrer Nervenzellen kompensiert. Die Gliazelldichte ist bei den Cetacea höher als beim Menschen.

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Brain Anatomy Institute of the Psychiatric Clinic, University of Berne, 3072 Waldau-Berne (Switzerland), 29th June 1967.

[7] A. HAWKINS and J. OLSZEWSKI, Science *126*, 76 (1957).

STUDIORUM PROGRESSUS

The Substrate Supply of the Human Skeletal Muscle at Rest, during and after Work[1]

The capacity of the human body for work is dependent on energy-producing metabolic processes in the muscle cell. A continuing production of energy in the mucle cell is tied to the supply of oxygen and energy-carrying substrates, of which the most important are glucose, lactate, pyruvate, free fatty acids and amino acids. The following is a report of the substrate uptake and discharge of the human skeletal muscle, established by the determination of arterio femoral venous differences during varied work load and at rest.

Material and methods. Subject matter: fourteen normal male persons aged 20–26 years ($\bar{x} = 24$) were examined. Course of the examination: the ergometric work test was carried out with the bicycle ergometer of the Elema Schönander company, Stockholm. It was preceded by a detailed clinical examination. Pulse frequency was determined auscultatorily. Work was done according to the principle of the relative steady state[2]. Starting with 50 W, the work load was increased by 50 W for each specific work level until the limit of capacity was reached. Arterial blood samples were taken from the arteria brachialis with a Cournand canula, femoral venous samples from the deep vena femoralis with a catheter. The samples

[1] This investigation was supported by the Deutsche Forschungsgemeinschaft and Kuratorium für sportmedizinische Forschung.

[2] H. MELLEROWICZ, *Ergometrie, Grundriss der medizinischen Leistungsmessung für Innere Medizin, Arbeitsmedizin, Sportmedizin, Versorgungsmedizin und Versicherungsmedizin* (Verlag Urban und Schwarzenberg, München und Berlin 1962).

were withdrawn during the 6th min of each specific work level and in the 3rd, 15th and 30th min following cessation of work. The rest value was determined immediately preceding the beginning of work.

Preparation of the blood samples. Immediately (i.e. 30–50 sec) after removal, the blood samples were deproteinized with ice-cold perchloric acid (7%) in the ratio 1:2. They were centrifuged at about 2300 *g* and the remainder decanted. *Glucose* was determined by the enzymatic colorimetric test[3]. *Lactate* was determined by lactate dehydrogenase and DPN[4]. *Pyruvate* was determined by lactate dehydrogenase and DPNH[5]. *Free fatty acids* in the blood plasma determined according to [6] with modifications[7,8]. The *O_2-pressure* in the anaerobically drawn blood was measured polarographically with a platinum electrode[9].

Statistics. The mean value ($\bar{x}$) and standard deviation (S.D.) were calculated. The comparison of 2 means, and the probability of significance (P) of their difference were calculated according to the *t*-test. The sequential analyses were based on couple-differences[10].

Results. Glucose (Table I). The mean arterial glucose concentration at rest is 4.40 µmols/ml. During work the concentration decreases, and at 200 W has sunk to 80% of the rest value ($P < 0.01$). Three min after work stop, there is a significant increase of 0.58 µmols/ml ($P < 0.01$). The arterio venous differences are significant ($P < 0.01$) at rest, as well as during and after work. At rest, the glucose extraction measures + 0.30 µmols/ml; during light work it reaches 2 or 3 times rest value. At 200 W, a reduction of the venous value to 55% of the original measurement is seen. Three min after exercise the arterio venous difference (AVD) is significantly reduced to 0.48 µmols/ml.

Lactate (Table II, Figure 1). The arterial concentration of lactate increases with the work load. It reaches 10.24 µmols/ml during maximal steady state exercise, and 10.71 µmols/ml following work stop. At submaximal work levels a slight release of lactate into the venous outflow and at the maximal level lactate discharge is significant ($P < 0.01$). In the 3rd min following work, the discharge continues to be significant ($P < 0.01$).

Pyruvate (Table III, Figure 1). The arterial pyruvate concentration at rest measures 0.103 µmols/ml. The highest value appears during the 3rd min of recovery (0.230 µmols/ml). At rest and at 50 W there is a significant pyruvate extraction. Under increased work load there follows a pyruvate discharge (200 W, $P < 0.01$). During the 3rd recovery min, the arterio-venous difference remains as high as during maximal exercise.

The lactate/pyruvate ratio (Table IV). The lactate/pyruvate ratio increases under work load and reaches the highest mean value (49.6) at 200 W. In the femoral venous blood, the lactate/pyruvate ratio is at rest and at 50 W

[3] H. U. Bergmeyer und E. Bernt, in *Methoden der enzymatischen Analyse* (Ed. H. U. Bergmeyer; Verlag Chemie, Weinheim 1962), p. 123.
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Table I. The concentration of glucose in the arterial and femoral venous blood of 14 normal persons at rest, during and after exercise

	Glucose µmol/ml	Rest	Exercise				Recovery		
			50 W	100 W	150 W	200 W	3 min	15 min	30 min
Arterial	$\bar{x}$	4.40	4.39	4.16	3.98	3.58	4.16	4.01	4.23
	S.D.	± 0.76	± 0.97	± 0.88	± 0.81	± 0.51	± 1.18	± 0.83	± 0.70
Femoral venous	$\bar{x}$	4.10	3.69	3.36	2.94	2.27	3.68	3.56	3.62
	S.D.	± 0.82	± 0.88	± 0.94	± 0.82	± 0.33	± 0.65	± 0.78	± 0.59
AVD	$\bar{x}$	+ 0.30	+ 0.70	+ 0.80	+ 1.04	+ 1.31	+ 0.48	+ 0.45	+ 0.61
	P	××	××	××	××	××	××	××	×

AVD, arterio venous difference.

Table II. The concentration of lactate in the arterial and femoral venous blood of 14 normal persons at rest, during and after exercise

	Lactate µmol/ml	Rest	Exercise				Recovery		
			50 W	100 W	150 W	200 W	3 min	15 min	30 min
Arterial	$\bar{x}$	1.20	1.30	1.95	4.39	10.24	10.71	6.45	3.34
	S.D.	± 0.58	± 0.50	± 1.02	± 2.54	± 2.47	± 2.64	± 2.17	± 1.57
Femoral venous	$\bar{x}$	1.17	1.34	2.02	4.70	10.97	11.70	6.70	3.42
	S.D.	± 0.39	± 0.51	± 1.07	± 2.40	± 2.66	± 2.95	± 2.30	± 1.56
AVD	$\bar{x}$	+ 0.03	− 0.04	− 0.07	− 0.31	− 0.73	− 0.99	− 0.25	− 0.08
	P	∅	∅	∅	++	++	+++	+	∅

higher than in the arterial blood. During heavy exercise, it is lower in femoral venous than in arterial blood.

Free fatty acids (Table V). The concentration of free fatty acids in the blood plasma decreases slightly at 50 W. With increasing work, it rises and reaches a maximum mean value of 1.119 µmols/ml at 200 W. Following work, it rises even higher to 1.200 µmols/ml. At rest, the AVD of the free fatty acids is practically zero. However, it increases with increased work, and the greatest difference is recorded at 200 W. Following work stop, AVD falls abruptly.

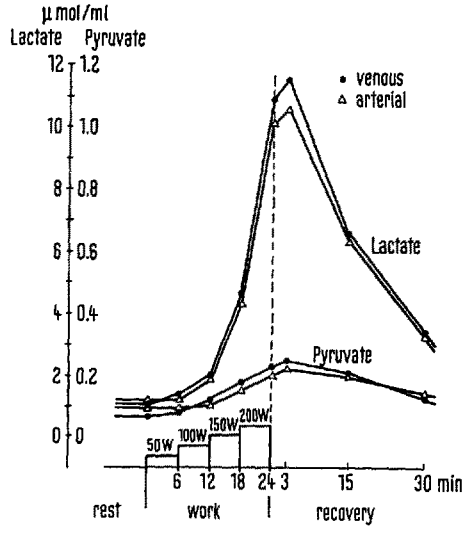

Fig. 1. The arterial and femoral venous concentrations of lactate and pyruvate at rest, during and after exercise (normal male persons $n = 14$).

Oxygen pressure (Figure 2). Starting with an average P_{O_2} of 45.6 ± 6.05 mm Hg, obtained at rest, the femoral venous oxygen pressure already shows a significant ($P < 0.025$) decline to 27.6 ± 3.48 mm Hg during light work (50 W). At 100 W it sinks further to 25.8 ± 3.39 mm Hg, at 150 W to 23.0 ± 1.85 mm Hg, and under maximal stress (200 W) to 21.7 ± 3.18 mm Hg ($P < 0.05$). As in the arterial blood, but much more pronouncedly, the cessation of physical work is followed by a significant ($P < 0.025$) O_2-pressure increase, so that in the 3rd min of recovery the $P_{O_2\bar{v}}$ measures 59.7 ± 6.80 mm Hg.

Discussion. In a summary paper, HERXHEIMER[11] reports a reduction of the arterial glucose level during, and an increase following physical exercise[12,13]. JAKOVLJEV, on the other hand, describes a different behaviour[14]. During physical work, there is a distinct decrease in glucose concentration (Table I), which could be caused by a supply insufficient for high consumption. The extraction of glucose increases with the increases in work. This is understandable, for under increased work load there is a rise, not only in oxygen extraction (as an expression of intensified oxidation processes, Figure 2), but also in the amount of lactate and pyruvate discharged by the skeletal muscles. Following work, there is an abrupt decline in glucose extraction (Table I). If we examine the extent to which the extracted glucose is entirely oxidized, and the extent to which it is merely metabolized into lactate and pyruvate (then discharged from the cell), we find that at the level of submaximal work, the glucose is

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[13] S. ROBINSON and P. M. HARMON, Am. J. physiol. *132*, 757 (1941).
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Table III. The concentration of pyruvate in the arterial and femoral venous blood of 14 normal persons at rest, during and after exercise

	Pyruvate µmol/ml	Rest	Exercise				Recovery		
			50 W	100 W	150 W	200 W	3 min	15 min	30 min
Arterial	$\bar{x}$	0.103	0.098	0.112	0.158	0.206	0.230	0.204	0.143
	S.D.	± 0.040	± 0.052	± 0.052	± 0.058	± 0.036	± 0.055	± 0.046	± 0.058
Femoral venous	$\bar{x}$	0.077	0.089	0.121	0.181	0.232	0.256	0.212	0.135
	S.D.	± 0.024	± 0.034	± 0.052	± 0.063	± 0.036	± 0.055	± 0.051	± 0.053
AVD	$\bar{x}$	+ 0.026	+ 0.009	— 0.009	— 0.023	— 0.026	— 0.026	— 0.008	+ 0.008
	P	++	+	∅	+	++	++	∅	∅

Table IV. The lactate/pyruvate ratio in the arterial and femoral venous blood of 14 normal persons at rest, during and after exercise

	Lactate/pyruvate ratio	Rest	Exercise				Recovery		
			50 W	100 W	150 W	200 W	3 min	15 min	30 min
Arterial	$\bar{x}$	11.9	13.6	17.4	25.5	49.6	46.6	30.2	23.9
	S.D.	± 2.8	± 3.7	± 4.3	± 7.6	± 7.7	± 8.9	± 5.7	± 8.4
Femoral venous	$\bar{x}$	16.7	16.1	16.4	24.9	45.3	46.7	30.7	25.7
	S.D.	± 9.0	± 5.8	± 3.0	± 9.9	± 5.7	± 11.4	± 8.6	± 7.0
AVD	$\bar{x}$	— 4.8	— 2.5	+ 1.0	+ 0.6	— 4.3	— 0.1	— 0.5	— 1.8
	P	++	+	∅	∅	++	∅	∅	∅

almost entirely oxidized (Figure 3). During increased work, however, more and more lactate and pyruvate are given off by the working muscles, until during maximal work almost $^1/_3$ of the glucose extracted is discharged as lactate and pyruvate. In the 3rd recovery min, the amount of glucose extracted corresponds exactly to the amount of lactate and pyruvate discharged.

It is well known that during heavy exercise lactate and pyruvate concentration increase in the blood. At 50 W, the arterial pyruvate level sinks below the rest value ($P < 0.05$). The highest lactate and pyruvate concentration was observed 3 min following work stoppage. The determination of the arterio femoral venous differences showed that during rest and light work the working muscles extract lactate and pyruvate ($P. < 0.01$ and $P < 0.05$) from the arterial blood (Tables II, III, Figure 1). During this stage, more lactate and pyruvate are oxidized than are formed in the muscle cells by glycolysis. Increased work load is accompanied by increasing discharge of lactate and pyruvate from the working muscles. The highest lactate discharge is found 3 min following work stop.

Shifts in the relationship of lactate and pyruvate are reflected in the lactate/pyruvate ratio. It is an accepted fact that the intracellular metabolite contents permit conclusions as to the relationship of free DPNH/DPN[15–17]; further, that there exists a permeation balance of lactate and pyruvate between the cytoplasmatic and extracellular spaces[18,19]. With certain limitations[20], it is possible from the lactate/pyruvate ratio in the blood to draw conclusions as to the relationship of free DPNH/DPN in the cytoplasm. During strenuous exercise, the arterial lactate/pyruvate ratio rises. From 100 W on, the lactate/pyruvate ratio is lower in the femoral venous than in the arterial blood. This is especially noticeable in athletes[21]. The question arises: what are the reasons for the increase in lactate and pyruvate, as well as of the lactate/pyruvate ratio, during and after exercise?

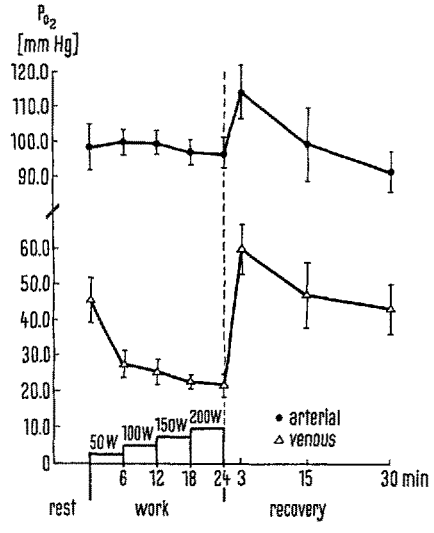

Fig. 2. The arterial and femoral venous P_{O_2} at rest, during and after exercise (normal male persons $n = 14$).

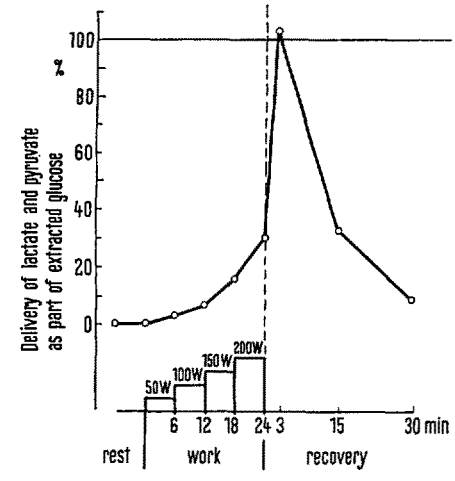

Fig. 3. During rest and light work (50 W), the extracted glucose is almost completely oxydized and only a little lactate and pyruvate is delivered by the working muscle. At the level of maximal activity (200 W), almost $^1/_3$ of the glucose is set free as lactatate and pyruvate. In the 3rd min after work stop, the extracted glucose is entirely eliminated as lactate and pyruvate into the V. femoralis. As recovery continues, the amount of extracted glucose discharged from the muscle cell as lactate and pyruvate decreases ($n = 10$).

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Table V. The concentration of free fatty acids in the arterial and femoral venous blood of 14 normal persons at rest, during and after exercise

	Free fatty acids µmol/ml	Rest	Exercise				Recovery		
			50 W	100 W	150 W	200 W	3 min	15 min	30 min
Arterial	$\bar{x}$	0.569	0.552	0.792	0.912	1.119	1.200	0.958	0.793
	S.D.	± 0.268	± 0.291	± 0.424	± 0.415	± 0.630	± 0.607	± 0.603	± 0.391
Femoral venous	$\bar{x}$	0.576	0.542	0.752	0.802	0.958	1.210	0.912	0.793
	S.D.	± 0.224	± 0.218	± 0.310	± 0.391	± 0.494	± 0.634	± 0.501	± 0.417
AVD	$\bar{x}$	— 0.007	— 0.010	+ 0.040	+ 0.110	+ 0.161	+ 0.010	+ 0.046	+ 0
	P	∅	∅	+	++	++	∅	+	∅

Previously, this increase was interpreted as an expression of insufficient oxygen supply to the working muscles, since, it was argued, the muscles were forced to extract energy anaerobically. This conception grew chiefly out of research dealing with isolated muscles, which can contract under anaerobic conditions, extracting the necessary energy mainly from metabolism of glucose to lactate[22–24]. The rise of the lactate/pyruvate ratio, as of 'excess lactate'[25], was explained by the argument that due to insufficient oxygen supply the hydrogen appearing in glycolysis and in the citrate-cycle could not be adequately oxidized. Growing oxygen deficiency resulted in an increase in the lactate/pyruvate ratio.

The conception that the rise in lactate, pyruvate, and the lactate/pyruvate ratio during exercise is the expression of insufficient O_2-supply to the working muscles cannot be reconciled with the following facts:

(1) During maximal work, the femoral venous O_2-pressure (Figure 2) sinks to 21.7 mm Hg, at which point the 'critical O_2-pressure' has not nearly been reached[26–28].

(2) In coronary sinus blood of the human heart, an O_2-pressure of 22.7 mm Hg (athletes 23.8 mm Hg) was measured during maximal work. The highest lactate level was also found at this level[29,30]. Since the transport of oxygen to the cell is achieved by diffusion[9], it is impossible to see why in 1 organ (the heart muscle) the decreased O_2-pressure should demonstrate the high oxidative metabolism which is seen in lactate extraction from the arterial blood whereas in another muscle (the skeletal muscle) it demonstrates an O_2-deficiency, which is visable in a discharge of lactate into the venous blood.

(3) Furthermore, the greatest lactate delivery by the working muscles occurs following work. At this point, the femoral venous O_2-pressure is very significantly higher than the rest value (Figure 2), so that the possibility of oxygen insufficiency can be disregarded. Nor can lactate storage in the cell during work be responsible for this outflow of lactate during recovery, for it is not possible for the muscle tissue to pile up so much lactate during work that in the 3rd min of recovery the arterio femoral venous difference would be considerably higher than during exercise.

(4) Following CARLSON and PERNOW[31], we measured lactate concentration and O_2-pressure of the femoral venous blood of both legs, while only 1 leg worked at the ergometer (Figure 4). Although the femoral venous O_2-pressure was nearly identical, the inactive leg extracted lactate, while the working leg discharged it. These facts prove that, during maximal work, the femoral venous O_2-pressure is too high to require lactate discharge due to oxygen sufficiency.

(5) The femoral venous lactate/pyruvate ratio during work is equal to or lower than the arterial lactate/pyruvate ratio (Table IV). As a result it is also questionable whether an increase in the redox-ratio of arterial blood during work can be interpreted as an expression of O_2-deficiency in the skeletal muscle, since, if this were the case, there would have to be an increase in the femoral venous lactate/pyruvate ratio in comparison to the arterial ratio. A further contradiction of the oxygen deficiency theory lies in the fact that following work there is a further increase of the lactate/pyruvate ratio in the femoral venous blood (Table IV), although in the femoral venous blood the O_2-pressure climbs significantly above rest value (Figures 2, 4).

These results are corroborated by the research of HOHORST[32] and KIRSTEN et al.[33], who report a decline of the lactate/pyruvate ratio in the abdominal and flight muscles of grasshoppers during exercise. This decrease probably results in particular from a glycerol-L-phosphate-pyruvate dismutation in the muscle tissue. They also found the highest lactate/pyruvate ratio in the abdominal muscles of rats following tetanic irritation[32,33].

(6) During increased physical activity, the skeletal muscle oxidizes increased amounts of free fatty acids (Table V).

If oxygen deficiency does not seem acceptable as a cause of the increase in lactate, pyruvate, and the lactate/pyruvate ratio during exercise, then we must ask the question: what factors are responsible for these changes? While glycolysis occurs within the cytoplasm, the oxidation of lactate and pyruvate takes place in the mitochondria. It is possible that glycolysis builds more pyruvate that can be metabolized by the intramitochondrial enzyme systems, so that during and after work there results a disproportion between glycolytic substrate

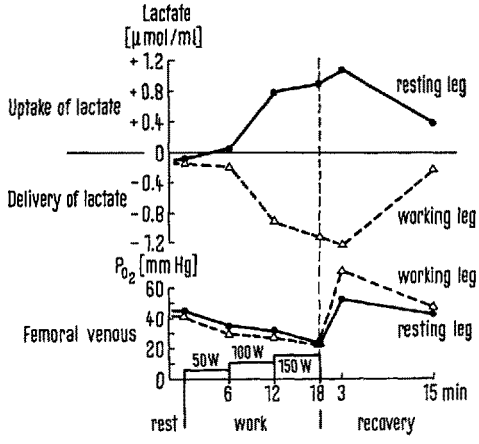

Fig. 4. While only 1 leg worked at the ergometer, lactate concentration and oxygen pressure of the femoral venous blood were measured in both legs. During exercise, the decrease of O_2-pressure in the inactive leg parallels that of the working leg. While the femoral venous O_2-pressure thus remains nearly the same, the working leg discharges lactate and the resting leg extracts lactate. Following work, the O_2-pressure in the working leg is higher than in the resting leg: at this point the greatest discharge of lactate is seen ($n = 3$).

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[33] E. KIRSTEN, R. KIRSTEN and P. ARESE, Biochem. Z. *337*, 167 (1963)

production and the further breakdown of the pyruvate. The following facts support this hypothesis:

(1) The heart muscle, which does not discharge lactate even during intensive work, has a high density of mitochondria in proportion to cytoplasm; the skeletal muscle, on the other hand, has a lower density. Muscle cells which perform a function intermediate to heart and skeletal muscles, the diaphragm, for example, also have an intermediate density of mitochondria proportionate to their cytoplasm[34]. It is possible that in the skeletal muscle the glycolytic capacity surpasses the respiratory capacity of the mitochondria, and that in the heart muscle, conversely, the respiratory capacity of the mitochondria surpasses the glycolitic capacity of the muscle.

(2) BÜCHER and RÜSSMAN[16] have shown that with sufficient O_2-supply, the lactate production in skeletal muscles and in the liver of rats is greater than can be oxidized even during maximal respiration.

(3) HOLZER and FREYTAG-HILE[35] were able to prove with yeast that glycolytic pyruvate production can be greater than maximal ability to oxidize the pyruvate, and that the bottleneck in the pyruvate oxydation lies in the pyruvate decarboxylase. Our own studies of pyruvate oxidase in the heart and skeletal muscle mitochondria of rats yielded similar results[36]. The increasing role of the free fatty acids in oxidative metabolism of the skeletal muscle during increased work load may be interpreted similarly. Lack of sufficient pyruvate oxidation would permit increased oxidation of fatty acids, as long as the capacity of the citric acid cycle or the respiration chain has not yet been reached.

(4) Three min following work, the highest lactate and pyruvate discharge is seen, indicating that the greatest disproportion between glycolytic production and the further oxidation of the glycolytic products by the mitochondria is reached at this point (Table II, Figure 1). Since O_2-insufficiency cannot be responsible (Figure 2), the reason for this disproportion is seen to lie in the varying reaction of glycolysis and respiration to the cessation of work.

Under increasing work load, a distinct extraction of free fatty acids is seen (Table V). Since the skeletal muscle also oxidizes free fatty acids[37-39], it may be assumed that increased extraction during strenuous exercise results from increased oxidation of free fatty acids. During maximal stead-state exercise, the amount of free fatty acids extracted is approximately equal to the amount of oxygen extracted and not transformed to H_2O and CO_2 by the oxidation of glucose. The respiratory quotient of 0.9–0.94 obtained through the arterio femoral venous O_2- and CO_2-differences proves that during work the free fatty acids play an increasingly large role in oxidative metabolism. This accords with the claims of HAVEL et al.[40] that during physical exertion the free fatty acids take over up to 40% of human oxidative metabolism.

It is surprising that energy production by glycolysis, if we take the amount of glucose extracted as an indication, does not amount to more than 5–6% of all energy production of the skeletal muscles at submaximal or maximal work levels. The energy gain achieved by breaking down glucose only as far as lactate, which is then discharged from the muscle cell, is approximately 1% at 100 W. At the level of maximal work, the energy produced by this process is not more than 2% of total energy production. This conclusion is the more astounding, as the lactate buildup in the blood during strenuous physical exercise had led to the assumption that the part played would be much larger[23].

Zusammenfassung. Die arterio femoral venösen Differenzen für Glucose, Lactat, Pyruvat, freie Fettsäuren und den Sauerstoffdruck wurden bei gesunden Erwachsenen in Ruhe, während leichter und schwerer körperlicher Arbeit unter Steady-state-Bedingungen und in der Erholungsphase gemessen.

Die allgemeine Vorstellung, dass der Anstieg des Lactats und des Lactat-Pyruvat-Quotienten im Blut während körperlicher Arbeit als Folge eines Sauerstoffmangels aufzufassen ist, ist mit folgenden Befunden nicht in Einklang zu bringen: (1) 3 min nach körperlicher Arbeit, wenn im femoral venösen Blut der Sauerstoffdruck signifikant über den Ruhewert angestiegen ist, wurde die grösste Lactat- und Pyruvatabgabe beobachtet. (2) Der kritische Sauerstoffdruck, bei welchem die oxydative Energiebereitstellung nicht mehr gewährleistet ist, wurde in dem Blut, welches der Arbeitsmuskulatur bei schwerer körperlicher Arbeit entströmt, nicht erreicht. (3) Der Lactat-Pyruvat-Quotient ist während körperlicher Arbeit im femoral venösen Blut niedriger als im arteriellen Blut, sodass eine unzureichende Sauerstoffversorgung des Muskels als Grund für den Anstieg des Lactat-Pyruvat-Quotienten nicht angenommen werden kann. (4) In der Erholungsphase steigt der Lactat-Pyruvat-Quotient im femoral venösen Blut über den arteriellen Wert an, obgleich der femoral venöse Sauerstoffdruck den Ruhewert übersteigt.

Während in Ruhe die extrahierte Glucose von dem Skelettmuskel fast vollständig oxydiert wird, werden bei schwerer körperlicher Arbeit $^2/_3$ der extrahierten Glucose vollständig oxydiert und $^1/_3$ zu Lactat und Pyruvat abgebaut, welches in die Blutbahn abgegeben wird. In der 3. Erholungsminute ist die Menge der extrahierten Glucose entsprechend der Menge an Lactat und Pyruvat, das vom Gewebe abgegeben wird.

Während schwerer körperlicher Arbeit beträgt der Anteil der Glycolyse der gesamten Energieproduktion durch den Skelettmuskel annähernd 4–6%.

Bei körperlicher Arbeit nimmt der Anteil der freien Fettsäuren an der Energiebereitstellung durch das Muskelgewebe zu.

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