

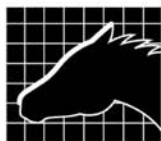
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Remarks on the Benefits of Heart Rate Recordings

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INTRODUCTION

In physiology, as well as nutrition, we use the term “energy” as a self-explaining phenomenon. But in fact, it is difficult to define energy, as it has no substrate and no status, but refers to a change in the electrochemical conformation of molecules. Adenosine triphosphate (ATP) is the ingenious concept that makes the power of those changes available to biological processes. It is a universal currency that pays for several types of cellular performance.

ATP is not the only energy-offering substance, particular at a static standard metabolic rate (animal at rest, stress-free, no digestion); uncoupled energy supply for body processes is driven by other substances besides ATP like NADH or NADPH. But an increase in metabolic rate (e.g., by muscle activity) requires more ATP. The availability of ATP depends on oxidative processes. The split of glucose, fatty acids, or even amino acids fuels ATP synthesis. Kleiber’s famous book titled *The Fire of Life* gives an impressive view on that context. As with any fire, the biological fire inside the cells requires oxygen. Roughly 90% of oxygen (O₂) consumption is used in the cellular power units, the mitochondria; 80% of that O₂ consumption is coupled to ATP production serving protein synthesis (28%), sodium (Na)-potassium (K) ATPase (19-28%), Ca₂-ATPase (4-8%), actinomyosin-ATPase (2-8%), gluconeogenesis (7-10%), and ureagenesis (3%) (Rolfe and Brown, 1997). At rest in a nondigesting condition, tissues representing roughly 50% of body weight (BW) account for 88% of O₂ consumption (Table 1) (Rolfe and Brown, 1997). A comparable distribution of tissues to O₂ consumption can be expected for horses.

Table 1: Major contributors to oxygen consumption.

Tissue	mass kg ⁻¹ BW		O ₂ consumption, %
	Human ¹	Horse ²	Human ¹
Heart	4	7.2	11
Kidney	5	3.6	6
Lung	9	11.5	4
Liver	20	13.2	17
GIT ³	20	61	10
Brain	20	2.5	20
Muscle	420	449	20
TOTAL	498	548	88

¹Rolfe and Brown, 1997

²Grace et al., 1999; Coenen, 1991

³Gastrointestinal tract

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Oxygen (O₂) consumption by muscles is the most flexible figure in healthy animals; at heavy exercise up to 90% of O₂ is directed to the muscle cells. The degree of increase in O₂ consumption reflects the demand for O₂ as well as the availability of cellular structures to drive metabolic processes via oxidation of substrates. It is interesting that the maximal oxygen consumption of animals (VO₂) over a wide range of species scales by BW^{0.872} and BW^{0.942} (Weibel et al., 2004) for 34 mammalian species and athletic species, respectively (Figure 1). In addition, the mitochondrial volume is parallel to VO₂max (Figure 2).

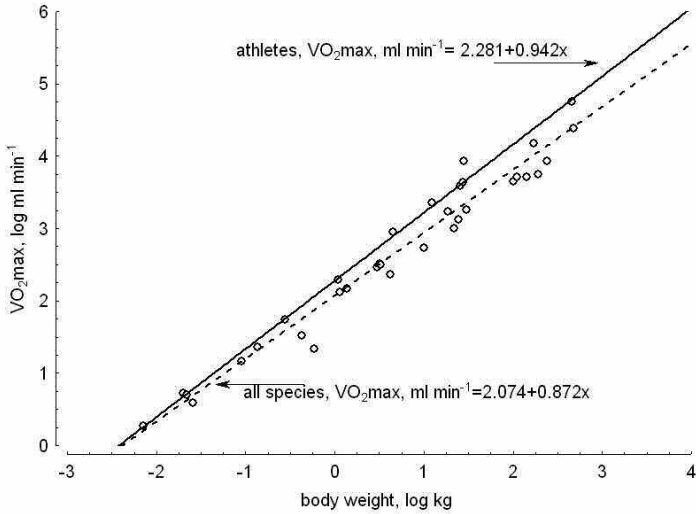


Figure 1. Maximal oxygen consumption is dependent on body weight (Weibel et al., 2004).

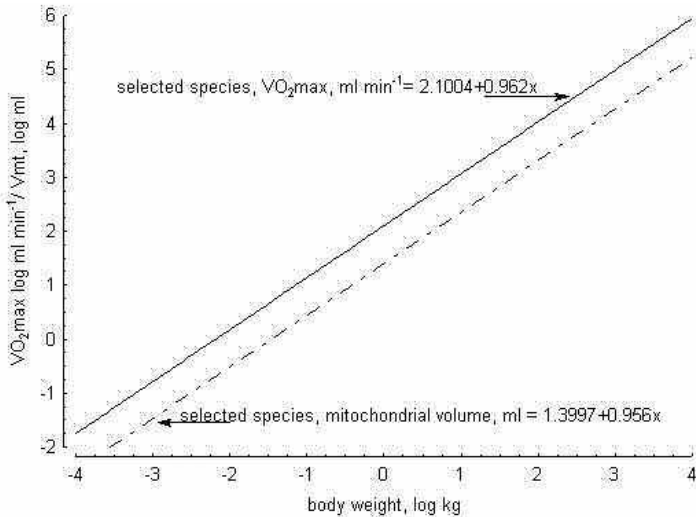


Figure 2. Maximal oxygen consumption and mitochondrial volume are dependent on body weight (woodmouse, mole rat, white rat, guinea pig, agouti, fox, goat, dog, pronghorn, horse, steer) (Weibel et al., 2004).

It can be expected that heart characteristics show a comparable constant allometric scaling. Heart size makes by $\sim 5.8 \text{ g kg}^{-1} \text{ BW}$ a rather constant fraction of BW (Prothero, 1979). An interspecies description of heart size for mammals is: heart, $\text{kg} = 0.00588 \text{ BW}^{0.984}$ (Brody, 1945). It is evident that regardless of the mentioned type of interspecies scaling, athletic species have a higher heart mass than nonathletes.

On average, mammals have $130 \text{ g hemoglobin L}^{-1}$ blood shuttling 175 ml O_2 (Schmidt-Nielsen, 1984). So far, the heart in species with similar body weights is faced by a rather uniform fluid. Therefore, the heart rate should yield a specific performance in terms of O_2 transportation per unit of time. A common expression of that is the "oxygen pulse," the rate of an animal's O_2 consumption (ml min^{-1}) per heart beat (HR, beats min^{-1}): O_2/HR is 0.047 BW^1 (Brody, 1945). This equation can be rewritten as $\text{O}_2/\text{BW} = 0.05 \text{ HR}$.

In conclusion it can be stated that the mitochondria represent the O_2 -consuming units. VO_2 , mitochondria volume and heart functions are scaled by BW. Therefore, heart rate must be linked to oxygen consumption and aerobic energy utilization by cellular functions.

Flexibility of VO_2 in Horses

The expenses for different metabolic products are quite different. The equine fetus realizes a VO_2 of $6.1 \text{ ml kg}^{-1} \text{ BW min}^{-1}$ (Silver and Comline, 1976; Fowden and Silver, 1995; Fowden et al., 2000). The consideration that only 45.6% of energy expenditure (EE) in the pregnant uterus is due to fetal metabolic rate while the rest is contributed by the adnexes and uterine tissue results in a high oxygen consumption and EE per unit of mass for gestation. Maintenance metabolism, growth, and lactation are less expensive per unit of product. The organism can easily adapt to those demands as they are developing over time.

Exercise is a different product because:

- there is no synthesis like milk
- physical energy is at the end
- the transformation of chemical energy into kinetic energy leaves as a "by-product" the highest portion of heat energy of all metabolic processes
- this transformation can immediately be activated
- the degree of kinetic energy production is extremely variable
- oxygen consumption follows strictly the demands of the muscle cell without a time delay
- kinetic energy at high-end exercise is the only metabolic energetic product that is realized by an anaerobic pathway.

Table 2 shows selected data for horses at rest and maximum exercise intensities. Impressively, the differences can be obtained within a very short time. Heart rate increases by a factor close to 10 to the maximum of $240 \text{ beats min}^{-1}$, while VO_2 consumption as well as the caloric equivalents is maximized by a factor ~ 47 .

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Table 2: Selected data for energy-related processes at rest and maximal exercise in horses (adapted from Hodgson and Rose, 1994).

	Rest	Exercise	Relative change
Speed	-	~16 m sec ⁻¹	
Heart rate, beat min ⁻¹	25	~240	~9.6
VO ₂ , ml kg ⁻¹ BW min ⁻¹	3.4	160	
Energy expenditure			
kJ/kcal kg ⁻¹ BW min ⁻¹	0.068/0.016	3.216/0.768	~47
kJ/kcal kg ^{0.75} BW min ⁻¹	0.323/0.077	15.21/3.634	

The increase in heart rate as the immediate response to O₂ consumption as well as to CO₂ liberation—being a strong trigger—in the muscle tissue is naturally accompanied with responses of the respiratory tract, particularly breathing. The breaths/min⁻¹ increases from 10–15 to 120–150 even if exercise intensity continues to increase. The tidal volume shows a linear growth from ~5 to >15 liters. In combination, this results in a nearly complete linear adaptation of minute ventilation on tissue demands (Marlin and Nankervis, 2003). The work of breathing can reach 14 kJ min⁻¹, which indicates the intensity of respiratory tract responses. Herein is one of the limitations for the equine species. There is no excess in diffusion capacity left; the total time of capillary flow is needed for total oxygenation of blood (Weibel, 1999).

A review of literature (87 papers published between 1898 and 2004; 759 horses; max speed 16.7 m sec⁻¹; max HR 238 beats min⁻¹; max VO₂ 176 ml kg⁻¹ BW min⁻¹) (Coenen, 2008) provides a summary of data on the relationship between workload and VO₂ as shown in Figure 3. In principle, up to ~8 m/sec⁻¹ we see an almost linear increase in VO₂ in response on elevated speed. At higher speed, VO₂ plateaus. This shows on one hand the flexibility of gas exchange and the linear response on tissue demands over a wide range of speed (=challenge), but on the other hand there is still a remarkable scatter, which inhibits simply creating a model to predict VO₂ via speed. One explanation for the variability is the environment and exercising condition. The slope of the treadmill will have a huge impact. A common polynomial regression analysis and treadmill incline as a second independent variable yields the following description:

$VO_2 \text{ ml kg}^{-1} \text{ BW min}^{-1} = 0.708 + 7.068x + 1.051x^2 - 0.059x^3 + 2.077z$; $x = \text{speed m sec}^{-1}$ $z = \text{incline \%}$; 1140 paired data weighted by number of horses, $N_{\text{weighted}} = 4186$; $r^2 = 0.919$.

The two physical characteristics of speed and incline as independent variables produce a suitable model for the estimation of VO₂ and may allow having a closer look at energy efficiency.

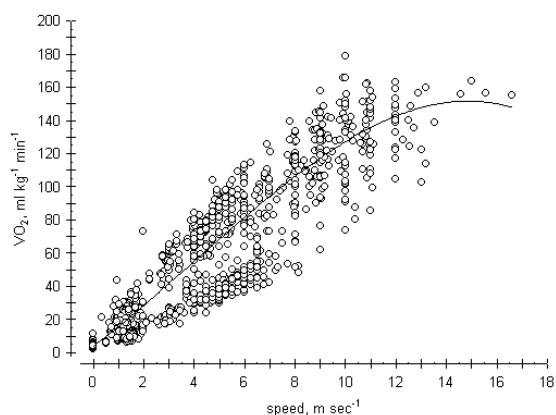


Figure 3. Plot of VO_2 against speed in 1,119 paired data representing 759 horses, mostly exercised on a treadmill.

Heart Rate as a Biological Scale for Exercise Intensity

The VO_2 -speed relationship excludes the horse which practices this relationship in an individual manner. Moreover, the given equation will lose its competence and precision to a remarkable degree if we take the horse from the treadmill to an outdoor racetrack or—even more complicated—to an undulating course with different ground quality. The mismatch between calculated energy requirement and required energy supply to an individual horse is a remarkable problem in practice. The “laboratory” method fails to estimate VO_2 and EE of the target horse in practice.

HR is the complex response on metabolic demands. In the case of muscle activity, this response includes any impact from the environment on muscle activity and metabolic performance (e.g., different track surfaces will be associated with different heart rates although speed is constant). A selection of factors making an impact on metabolic effort includes:

- breed
- body weight
- speed
- gait
- oxygen debt
- extra load by rider and saddle
- rider’s capability
- track surface
- temperature
- wind
- positive and negative incline
- shoeing
- experience
- status of training
- individual muscle fiber profile
- individual tonus of the sympathetic/parasympathicus.

It is evident that HR is the only biological signal that integrates the listed factors and that can be recorded by commercially available instruments under practical conditions during any type of exercise. However, under the standardized treadmill condition, there should be a strict link between velocity and HR like between the former and VO_2 . Figure 4 shows the HR dependence on speed.

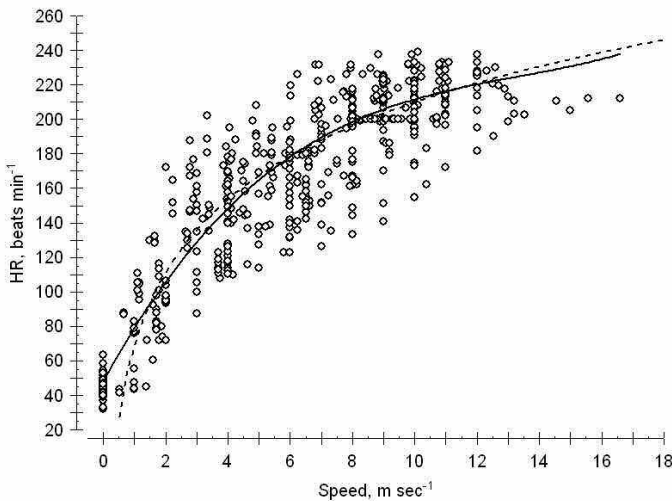


Figure 4. Plot of heart rate against speed in 501 paired data; polynomial regression closed line, logarithmic regression broken line.

Including incline, the HR predicting model is: $41.8+26x-1.274x^2+0.022x^3+2.877z$, where x =speed, m sec^{-1} and z =incline, %; data weighted by number of horses, $N^{\text{weighted}}=3328$; $r^2=0.897$.

Exclusively speed as a predictor of HF is not sufficient as it is the same for VO_2 vs. speed. The fact that HR shows a high scatter around the regression line is not limiting the value of HF as an integrated signal; moreover, the comparable scatter in both figures indicates a relation between VO_2 and HR.

Data processing in that way yields a model to estimate VO_2 by HR, which is shown in Figure 5. This is slightly different from one which is obtained after logarithmic transformation. Neglecting small differences in the model >91% of VO_2 variation is explained. So far, HR is a suitable predictor of VO_2 , and from a biological point of view, superior in comparison to speed and treadmill incline.

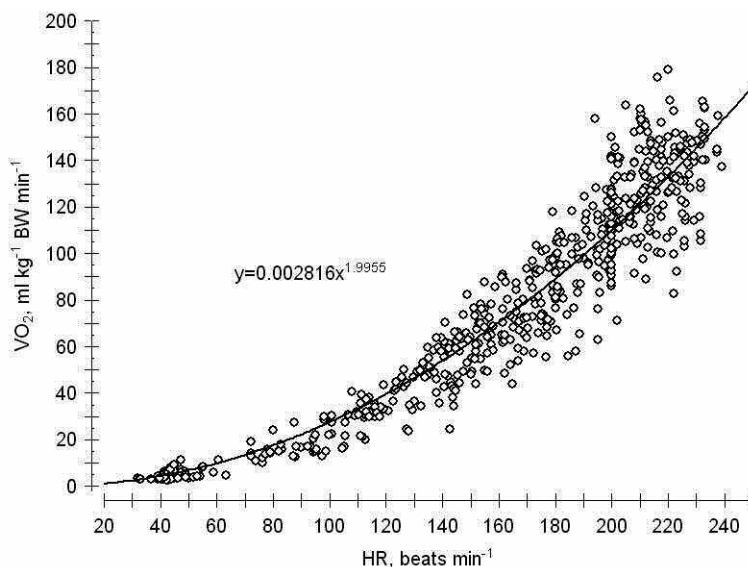


Figure 5. Plot of VO_2 against heart rate; 569 paired data weighted by number of horses, $N_{\text{weight}} 3710$; $r^2=0.911$.

The described model includes two cardiac responses during exercise: (1) increase in frequency, and (2) increase in stroke volume. The latter is the reason for the nonlinear nature of the relationship between VO_2 and HR. This is also reflected by a constant increment of oxygen pulse from $\sim 0.1 \text{ ml kg}^{-1} \text{ BW min}^{-1}$ per heart beat to 0.7 at maximal exercise intensities. Consequently, a model like that one which is substantiated in Figure 5 reflects the entire cardiac response on all VO_2 -modifying factors.

Computing Energy Expenditure by VO_2 Predicted Via Heart Rate

ATP production via substrate oxidation is linked to a defined O_2 production. Differences in dependence on type of substrate, e.g., glucose or fat, are commonly described by the respiratory quotient = the CO_2 production per unit of O_2 consumption. On average, 1 ml VO_2 can be taken as 20.1 J. This element of indirect calorimetry can easily be applied to the HR- VO_2 relationship. For that purpose, the product of the constant factor 0.002816 in the equation of Figure 5 multiplied by 20.1 is inserted. The equation is now rewritten: $EE, \text{ J kg}^{-1} \text{ BW min}^{-1} = 0.0566x^{1.9955}$, where $x=HR$. In that way, the aerobic part of EE can be predicted by HR.

Insecurities and Limitations of Energy Expenditure Derived from VO_2

Regardless if measured or predicted by HR, VO_2 during exercise $\times 20.1$ is not the complete figure about the total EE for three reasons.

(1) *Oxygen depletion.* After onset of exercise, a delay exists in balancing O_2 supply and O_2 requirement that means the recorded VO_2 during this lag phase underestimates EE. But this phase is measured in seconds and is of minor consequence.

(2) *Excessive post-exercise oxygen consumption.* After the end of an exercise session, EE cannot return to pre-exercise levels within a short time. Duration as well as degree of VO_2 and consequently EE being above resting levels is not well-defined. It should be a function of oxygen depletion, oxygen deficit, and the loss of intracellular energy stores. Actually no data are available to model an estimate of this important part of exercise-associated EE.

(3) *Anaerobic part of exercise.* It is common sense that at higher intensities, increasing blood lactate levels indicate an increasing contribution of anaerobic energy metabolism to total EE. The lactate accumulation in blood is taken as an estimate for the degree in anaerobic energy metabolism and several studies derive a caloric equivalent for accumulated lactate; a lactate accumulation of 1 mmol min^{-1} is equivalent to $1.03\text{--}1.8 \text{ J sec}^{-1}$. Modeling a lactate accumulation curve therefore enables the addition of the anaerobic part of energy metabolism to the defined aerobic part. However, lactate accumulation as well as lactate utilization is variable dependent on type of exercise and training status of the horse. Furthermore, the energetic value of accumulated lactate is not intensively investigated. Therefore, the calculation described above is more an approximation than a precise estimate. Using an average lactate curve as shown in Figure 6 ($5.8 \text{ mmol lactate at } \sim 8 \text{ m sec}^{-1}$ and 180 HR), the lactate accumulation per minute would be small per change of HR and speed, respectively.

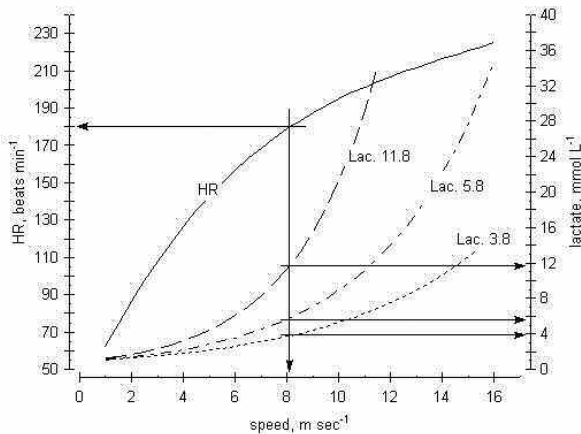


Figure 6. Examples for blood lactate curves dependent on speed and the corresponding heart rate (numbers indicate lactate concentration at a HR of 180; Models for Lac. 3.8: $y = 0.9112 \cdot \exp(0.176x)$; for Lac. 5.8: $y = 0.9112 \cdot \exp(0.228x)$; for Lac. 11.8: $y = 0.9112 \cdot \exp(0.3154x)$).

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Fit and unfit horses may show lower or higher lactate levels respectively at the same condition in terms of speed and HR. At highest speed realizing ~30-35 mmol lactate L⁻¹ (Hodgson and Rose, 1994), horses need an anaerobic contribution of 60% to total EE. As the blood lactate curve can be accepted as an indicator of the continuously occurring lactate accumulation, it mirrors the partition of anaerobic contribution to total EE. Consequently, any lactate curve can be transformed into the percentage of anaerobic contribution of EE using a maximum of 35 mmol lactate per liter of blood and the reported partition of 60% anaerobic metabolism to EE (Hodgson and Rose, 1994). Of course, the inaccuracy herein is that changes in lactate dynamics are neglected. Table 3 summarizes the estimates for the anaerobic part of EE. For the average condition of (5.8 mmol lactate at HR 180) an anaerobic contribution to total EE of 18 and 46% can be expected for HR of 200 and 220, respectively.

Table 3. Modeling the anaerobic contribution to total energy expenditure for three different lactate curves with 3.8, 5.8, and 7.8 mmol L⁻¹ at a heart rate of 180 beats min⁻¹ (basic model, % anaerobic contribution = $ax + b \cdot \exp(cx)$, where $x = \text{HR}$; the lactate levels from the curve in Figure 6 are transformed by the assumption that 35 mmol lactate equals 60% of anaerobic partition).

Blood lactate at HR 180			3.8	5.8	7.8	
Factors in the model $y = ax + b \cdot \exp(cx)$			a	0.0252	0.0338	0.0382
			b	0.000140	0.000079	0.000089
			c	0.05282	0.05952	0.06155
HR, beats min ⁻¹	VO ₂ , ml	kj	% anaerob			
	kg ⁻¹ BW min ⁻¹					
180	89	1.79	6	10	13	
200	110	2.21	10	18	27	
220	133	2.67	21	46	76	

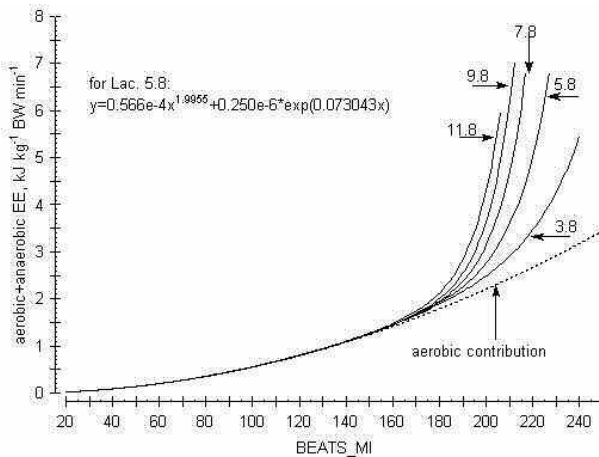


Figure 7. Total energy expenditure is dependent on heart rate and different lactate curves which are characterized by lactate concentrations at HR 180 (Lac. 180 in brackets); the ends of the lines mark the lactate-HR combination at which the anaerobic partition exceeds 60%.

After the estimation of the relative anaerobic part of EE and based on the given aerobic contribution calculated from $\dot{V}O_2$, it is possible to calculate absolute values for total EE. Figure 7 shows EE curves dependent on HR. It is evident that lactate accumulation creates a high impact on the results if horses exercise at a HR above ~ 160 beats min^{-1} . The differences are relevant in two ways: (1) the variation in anaerobic contribution to EE, and (2) the limitation in exercise. A horse that shows, for example, 9.8 mmol lactate per liter of blood at a heart rate of 180 beats min^{-1} will not continue to increase exercise intensity up to a HR of 240 beats min^{-1} without negative effects as that would require $>60\%$ of EE generated via anaerobic metabolism.

Application of Heart Rate Measurements

The calculated EE reflected the ATP production for muscle activity. Because the conversion of this chemically-organized energy into kinetic energy is associated with high heat losses, we can take the calculated values as metabolizable energy (ME) and ME requirements for exercise respectively. Table 4 gives an example for absolute ME values obtained by the described models.

Table 4. Energy expenditure in a 600-kg horse exercising at different intensities during a training session.

BW (kg)	min	Speed m sec^{-1}	HR, beats min^{-1}	$\text{kJ kg}^{-1} \text{BW min}^{-1}$	MJ per section
600	15	1.5	90	0.45	4.05
	15	3	140	1.09	9.83
	15	4	140	1.09	9.83
	10	8	200	2.76	16.58
	3	10	210	3.58	6.45
	13	3	140	1.09	8.52
sum	19	1.5	90	0.45	5.12
	90				60.4

The exercise intensity can be differentiated by gait and expressing the ME as multiple of maintenance requirement. Figure 8 shows that a HR of 160 beats min^{-1} corresponds to a ME requirement close to $1.5 \text{ kg}^{-1} \text{BW min}^{-1}$ equal to the 20-fold of maintenance requirement per kg BW min^{-1} .

As the maximal HR shows no remarkable changes during training the quality of the signal is rather independent from training status. HR measurements and utilization in the described way offers the following options:

- estimation of aerobic energy expenditure
- estimation of aerobic and anaerobic energy expenditure and requirements
- objective data on exercise intensity
- characterizing exercise by multiple of maintenance condition; this allows adjusting an individual horse based on its own resting level of HR
- estimation of sweat volume and electrolyte losses via sweat; for that purpose one can use the assumption that 70% of expended energy is transformed to heat and 30% of that is liberated by the

respiratory tract while 70% requires the cutaneous route by sweat production; 2.428 kJ of heat are exported per ml of sweat

- evaluation of the aerobic-anaerobic threshold; the combination of HR and lactate measurements yields information on actual fitness and effects of training
- evaluation of endurance performance; maintaining HR during a defined exercise bout (e.g., during a 15-min canter) indicates the quality of adaptation of muscle metabolism.

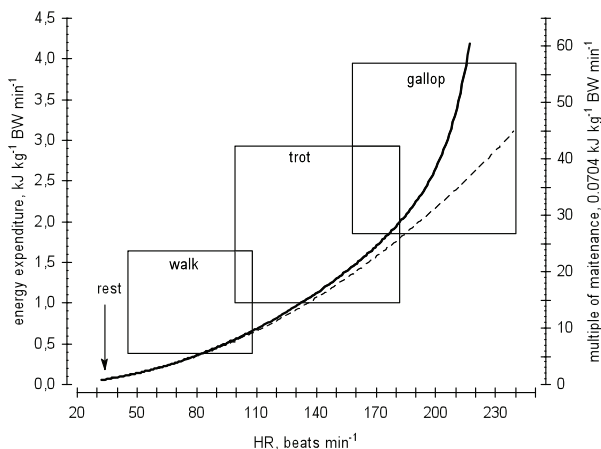


Figure 8. Energy expenditure $\text{kg}^{-1} \text{BW min}^{-1}$ in kJ (left y-axis) and as multiple of maintenance (right y-axis) in dependence on heart rate (HR); solid line=aerobic+anaerobic energy metabolism assuming 5.8 mmol lactate at a HR of 180 beats min^{-1} , broken line=aerobic ATP production only.

Conclusion

HR recording is a simple measurement to obtain data about exercise intensity. It can provide objective information about the effort for the rider. The close relationship to VO_2 can be used to estimate VO_2 and aerobic EE. The precision is as good as required for application under field conditions. The total EE requires estimation of the anaerobic energy supply to the muscle cell. Lactate is the only available parameter for that purpose outside the treadmill laboratory with direct VO_2 measurements. However, the inclusion of lactate accumulation and the corresponding caloric equivalent could be a suitable approximation, but lactate accumulation is difficult to define in a mathematical model. A simple procedure presented here is to take the lactate curve in blood as the indicator for the degree of anaerobic metabolism. The end points for well-trained high-performance horses (35 mmol lactate per liter of blood and maximum 60% anaerobic contribution to EE) enable us to estimate the anaerobic energy supply. A procedure like this is still removed from a precise measurement like in lactating mares, where the product is simply qualified. But by the inclusion of the anaerobic part, we get a valid approximation of the metabolizable energy required for exercise over the entire range of intensities.

Future research should yield a precise model for the caloric equivalent of lactate or an alternative procedure for the inclusion of the anaerobic part in EE. The problem of continuously elevated EE after finishing exercise is still unsolved. It can be speculated that after hard exercise it will remain up to 30% above resting levels, but exact measurements are lacking.

Author's Remarks

(1) The list of literature for data on HR and $\dot{V}O_2$ can be provided.

(2) HR provides a dual option. First, HR can be integrated in training management and control of training success. Second, HR serves for the estimation of energy expenditure. Regarding the latter, the value of HR recordings may be hardly elevated for high-performing horses if heart size and stroke volume measured by noninvasive methods are included. These parameters could help to "calibrate" the predictive strength of HR for an individual horse.

(3) It should be possible to integrate an individual lactate curve of a specific horse. Again, this can be of interest for high-performance horses.

REFERENCES

- Brody, S. 1945. Bioenergetics and growth. Reinhold Publishing Corporation. Reprinted 1964 by Hafner Publishing Company, Inc. New York.
- Coenen, M. 2008. The suitability of heart rate in the prediction of oxygen consumption, energy expenditure and energy requirement of the exercising horse. Wageningen Academic Publishers, Wageningen.
- Fowden, A.L., A.J. Forhead, K.L. White, and P.M. Taylor. 2000. Equine uteroplacental metabolism at mid- and late gestation. *Exp. Physiol.* 85:539-545.
- Fowden, A.L., and M. Silver. 1995. Glucose and oxygen metabolism in the fetal foal during late gestation. *Am. J. Physiol.* 269:R1455-1461.
- Hodgson, D.R., and R.J. Rose. 1994. *The Athletic Horse*. W.B. Saunders Company, Philadelphia, London.
- Marlin, D., and K. Nankervis. 2003. *Equine Exercise Physiology*. Blackwell Sciences Ltd., Oxford.
- Prothero, J. 1979. Heart weight as a function of body weight in mammals. *Growth* 43:139-150.
- Rolfe, D.S., and G.C. Brown. 1997. Cellular energy utilization and molecular origin of standard metabolic rate in mammals. *Physiol. Rev.* 77:731-758.

Schmidt-Nielsen, K. 1984. *Scaling: Why is animal size so important?* Cambridge University Press, Cambridge.

Silver, M., and R.S. Comline. 1976. Fetal and placental O₂ consumption and the uptake of different metabolites in the ruminant and horse during late gestation. *Adv. Exp. Med. Biol.* 75:731-736.

Weibel, E.R. 1999. Understanding the limitation of O₂ supply through comparative physiology. *Respiration Physiol.* 118:85-93.

Weibel, E.R., L.D. Bacigalupe, B. Schmitt, and H. Hoppeler. 2004. Allometric scaling of maximal metabolic rate in mammals: Muscle aerobic capacity as determinant factor. *Respir. Physiol. Neurobiol.* 140:115-132.