The complex relationship between effort and heart rate: a hint from dynamic analysis

Mongin, Denis; Chabert, Clovis; Uribe Caparros, Adriana; Guzmán, J F Vico; Hue, Olivier; Alvero-Cruz, Jose Ramon; Courvoisier, Delphine

How to cite

MONGIN, Denis et al. The complex relationship between effort and heart rate: a hint from dynamic analysis. In: Physiological Measurement, 2020, vol. 41, n° 10, p. 105003. doi: 10.1088/1361-6579/abbb6e

This publication URL: https://archive-ouverte.unige.ch/unige:155375

Publication DOI: 10.1088/1361-6579/abbb6e

The complex relationship between effort and heart rate: a hint from dynamical analysis

D. Mongin[*1], C. Chabert[2], A. Uribe-Caparros[1], J. F. Vico Guzmán[3], O. Hue[2], J. R. Alvero-Cruz[3], D. S. Courvoisier[1].

[1] Quality of Care Unit, University Hospitals of Geneva, Geneva, Switzerland

[2] ACTES laboratory, UPRES-EA 3596 UFR-STAPS, University of the French West Indies, Guadeloupe, France

[3] Malaga University, Andalucía Tech., Department of Human physiology, histology, pathological anatomy and physical education, Malaga, Spain

[*] Corresponding author: denis.mongin@unige.ch
26 avenue Beau Séjour
1211 Genève, Switzerland

This is the accepted version of the article published in Physiological measurements: doi: 10.1088/1361-6579/abbb6e

This is an Accepted Manuscript of an article published by IOP Science in Physiological measurements on* 4th November 2020*, available online:

https://iopscience.iop.org/article/10.1088/1361-6579/abbb6e#

Abstract

Objective: Dynamical analysis can be used to study the changes of self-regulated biological processes driven by external stimuli. Recently, heart rate during effort tests has been successfully adjusted with a simple first order differential equation with constant coefficients driven by the body power expenditure. Although producing proper estimations and yielding pertinent indices to analyze such measurements, this approach suffers from its inability to model the saturation of the heart rate increase at high power expenditure and the change of heart rate equilibrium after effort.

Approach: We propose a new analysis allowing to estimate the changes of heart rate response to effort (gain) as a function of the power expenditure value.

Main results: When applied to heart rate measured on 30 amateur athletes performing a maximum graded treadmill effort test, the proposed model was able to predict 99% of the measured heart rate change during exercise. The gains estimated decreased with power increase above the first ventilatory threshold. This trend was stronger above the second ventilatory threshold and was strongly correlated with the maximum oxygen consumption.

Significance: The proposed approach yields a highly precise modeling of heart rate dynamics during variable effort reflecting the changes of metabolic energy systems at play during exercise.

Key words—Heart monitoring, graded exercise test, dynamical analysis, Heart rate measurement curve, gain scheduling

Introduction

During physical activity, the main function of the cardiorespiratory system is to maintain an adequate oxygen and substrates input to fulfill the increased energy expenditure, while limiting the metabolic end-products accumulation in muscles cells (Rivera-Brown and Frontera, 2012). Heart rate (HR) regulation is one of the mechanisms that allows to keep the equilibrium between supply and demand during exercise. It is mainly regulated via the autonomic nervous system (ANS) (White and Raven, 2014), which responds to signals coming from various bio-sensors, such as baroreceptors, chemoreceptors, and a feed-back mechanism originating from skeletal muscle (Nobrega et al., 2014). The overall heart rate changes are mainly driven by the energetic requirements (Bernard et al., 1997). Its recording during effort tests is thus a key measurement to assess physical performance (Bellenger et al., 2016), and is everyday more accessible, thanks to the development of mobile measurement devices (Clark et al., 2017). In addition to the calculation of the heart resting rate (HRR) and the HR reserve (Bunc et al., 1988; Ludwig et al., 2018), the HR dynamics obtained during a maximal graded exercise test (HR measurement curve) has also been employed to determine the lactic accumulation threshold (Conconi et al., 1982). This method, known as the Conconi method, links the lactic accumulation threshold with the HR dynamic change at high energy expenditure, appearing as an inflection point in the HR performance curve. Widely used in sport science, it proves the interest in characterizing the dynamics changes during effort. Attractive by its simplicity, this approach suffers from several drawbacks, including a variable reliability (Cabo et al., 2011; Jones and Doust, 1995), the existence of HR performance curves having no inflection points (Hofmann et al., 1994, 1997; Kjertakov et al., 2016), and a lack of accuracy (Carey, 2002; Jones and Doust, 1997).

As an alternative method, we propose to use dynamical modeling to quantify the changes of heart rate response during effort. Recent developments of dynamical analysis based on a first order differential equation driven by power expenditure have proven to reproduce the dynamics of oxygen consumption (VO_2) and HR very accurately during any effort type (Artiga Gonzalez et al., 2017; Mongin, Chabert, et al., 2020; Mongin, Uribe Caparros, et al., 2020), and to produce indices sensible to physical performance and physical fitness (Mongin, Chabert, et al., 2020; Mongin, Uribe Caparros, et al., 2020). Among these indices, the HR gain, that is the proportionality between the effort and the HR increase, was linked with most of the performance indicators. Despite these promising results, this approach based on a differential equation with constant coefficients is unable to model HR dynamic changes occurring at high energy expenditure (Bodner and Rhodes, 2000) or to account for the fact that HR decreases back to a value higher than its resting value shortly after the effort (Lambert et al., 1998; Savin et al., 1982; Wyss et al., 1974). Such dynamic changes would require modelling the heart as a nonlinear system with power expenditure as input, resulting in more complex, harder to estimate models (Zakynthinaki, 2015). A control theory technique divides the nonlinear system in sections that could be considered linear and changes the parameters of the controller (the controller gains) in order to adapt to the operation conditions (set of parameters for the linearized system) of that section. This is called gain scheduling (Seborg et al., 2010). Although here we are not using controllers to influence the response of the system, applied to our case a first order differential equation model allowing the HR gain to have different values for different range of power spent during effort could allow to model the HR dynamics and its allostasis (i.e., return to a higher resting heart rate than before the test) more accurately during effort. It would furthermore allow to precisely quantify the HR dynamics changes occurring during the different stages of the exercise test.

In this article we will test the ability of such model to account for changes of HR response to effort increase along grading exercise tests. After describing its statistical and mathematical basis, we will apply the proposed method to heart rate measured on 30 athletes during graded exercise. We will compare its performance in predicting HR evolution with its constant coefficient counterpart. The construct validity of the measured HR dynamic changes stemming from this analysis will be provided by testing their links with the Conconi method and with standard performance indices.

Methods

Population

The population studied consisted in 30 Spanish amateur athletes (28 males, 2 females, Age = 36 ± 8 years). Participation in the study was voluntary, and prior to its initiation, written informed consent was obtained from the participants. This cross-sectional and correlational study conducted in 2012 was approved by the Research Ethics Committee of the University of Málaga, Spain (EMEFYDE UMA: 2018-02 report) and carried out according to the principles of the Declaration of Helsinki. Table 1 summarize the physiological characteristics of this population. The mean fat percentage is low, and the maximum aerobic O_2 consumption is high with low standard deviation, indicating an ensemble of well-trained persons.

Table 1: physiological characteristics and standard performance indices of the population

Number of individuals	30
Age (years)	36.50 [30.00, 41.00]
Height (cm)	174.70 [170.03, 178.75]
Weight (kg)	72.50 [64.40, 77.00]
BMI	23.30 [22.31, 24.77]
Sex (N male)	28 (93.3%)
Fat (%)	15.75 [14.67, 17.46]
VO_2 max (mL/kg/min)	50.98 [46.09, 55.79]
Maximal Speed (km/h)	18.10 [17.00, 19.10]
HR max (beat/min)	181.50 [178.00, 187.75]
VT1 (km/h)	9.00 [8.00, 10.00]
VT2 (km/h)	13.00 [12.00, 14.00]
HRR (beat/min)	36.00 [29.00, 41.75]

BMI: Body Mass Index, VO2 max: maximal aerobic capacities, VT1 and VT2: speed at the first ventilatory and second ventilatory transition, HR max: maximum HR; HRR: Heart Rate Recovery, measured as the difference between heart rate at exercise cessation and heart rate 60 s after. Values indicated are the median [Inter Quartile Range] of the corresponding variable.

Maximal effort test

The athletes performed graded exercise on a PowerJog J series treadmill connected to a CPX MedGraphics gas analyzer system (Medical Graphics, St Paul, MN, USA) with measurement of respiratory parameters of which the total volume exhausted (VE), oxygen consumption (VO_2), carbon dioxide consumption (VCO_2), and HR with a 12 lead ECG. Measurements are taken cycle to cycle. The stress test consisted of an 8-10 min warm up period of 5 km.h^{-1} followed by steps 1km.h^{-1} by minute speed increase until the maximum effort was reached. The power developed during the effort test was calculated using the formula described by the American College of Sport Medicine (ACSM) to determine an approximate VO_2 of runners (Ferguson, 2014) associated to the Hawley and Noakes equation linking oxygen consumption to mechanical power (Hawley and Noakes, 1992).

Standard performance indices

The HRR calculated is the standard HRR60, which is the difference between the HR at the onset of the recovery and the HR 60 seconds later (Cole et al., 1999). The ventilatory thresholds 1 (VT1) and 2 (VT2) are calculated using the Wasserman method using the minute ventilation (VE)/VO_2 for determining VT1 and VE/VCO_2 for VT2 (Wasserman et al., 1973). The corresponding power expenditures are noted PVT1 and PVT2.

Concerning the Conconi method, we focused on the deflection degree of the HR curve as presented in Hoffman's and coauthors work (Hofmann et al., 1997). Shortly, this method consists in fitting the HR curve with a second-degree polynomial of time for powers between the power at first ventilatory transition (PVT1) and the maximal power, in order to calculate the tangent slopes of the HR curve at these two power values (the two tangents k1 and k2 respectively at power equal to PVT1 and P max). The formula below provides the deflection degree of the HR curve:

$$k_{HR} = \frac{k1 - k2}{1 + k1 \times k2}$$

Following (Hofmann et al., 1997), $k_{HR} > 0.1$ is considered to be a normal deflection, kHR < 0 is an inverse deflection and $0 < k_{HR} < 0.1$ marks the absence of an usable deflection point.

Dynamical analysis

The basis of the dynamical analysis proposed relies on the estimation of the constant coefficients of a first order differential equation (for more details, see (Mongin, Uribe Caparros, et al., 2020)). Shortly, describing the HR dynamics by a first order differential equation consists in linking the change in time of HR with its value and the power spend during effort:

$$\dot{HR}(t) + \frac{HR(t) - HR_0}{\tau} = \frac{K}{\tau}P(t) \qquad (1)$$

Where $\dot{HR}$ is the time derivative of HR and $P(t)$ the power spent during the effort. Equation 1 describes the dynamics of a self-regulated system with an equilibrium value HR_0, a decay time τ and a gain K (see Figure 1 left panel). The equilibrium value corresponds in the case of HR to the heart rate resting value HR_0 (in beat/min) and is the HR in the absence of effort. HR described by equation 1 will respond to a constant energy expenditure P by increasing from HR_0 to $HR_0 + KP$ in an exponential manner, with the decay time τ associated being the time needed for HR to reach 63% of its total change. The gain (in beat/min/W) gives the proportionality between an effort increase ΔP (in W) and the associated total HR increase ΔHR (see Figure 1 left panel):

$$\Delta HR = K \times \Delta P$$

The determination of these 3 coefficients (resting value, decay time and gain) is performed by first evaluating the HR derivative using functional analysis regression, and then performing a multilevel linear regression between $\dot{HR}$(t), $HR(t)$ and $P(t)$(Mongin, Uribe Caparros, et al., 2020). An estimated curve can then be reconstructed from the estimated coefficients by performing a numerical integration of the differential equation.

To account for a power dependent gain, it is possible to decompose the power as a sum of successive power steps, each of them having an associated gain:

$$\dot{HR}(t) + \frac{HR(t) - HR_0}{\tau} = \frac{1}{\tau}\sum_{i=1}^{N_{steps}} K_i \times P_i(t) \quad (2)$$

Where the index i ranges from 1 to N_{steps} , the total number of power steps considered. The gain K_i is representative of the HR response for the power encompassed by $P_i(t)$ (see Figure 1 right bottom panel). An example of the HR response to an incremental effort with a decreasing gain compared to the response with a constant gain is presented in the right panel of Figure 1. In the case of a graded exercise, the natural decomposition of the power spend during the test is a sum of constant power steps. In the example depicted in the right panel of Figure 1, the 4 power steps introduced in equation 2 allow to obtain four values of gain for their respective power ranges (panel B of Figure 1).

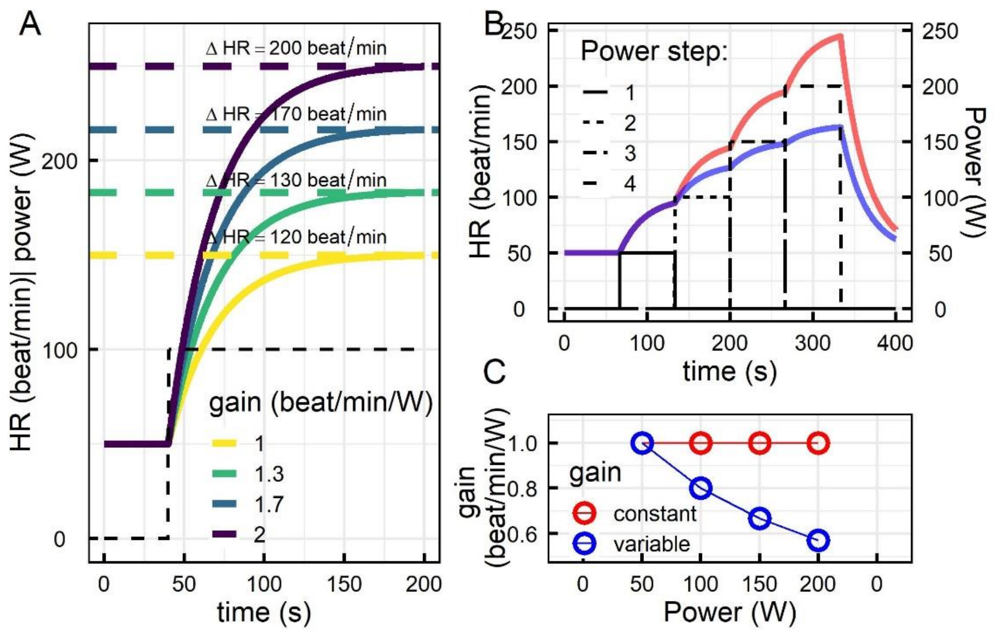


Figure 1. A: HR response following a first differential equation driven by a constant step effort of 100W. Parameters are $\tau = 30$s, $HR_0 = 50$ beats/min and four values of gain. B: HR response following a first differential equation driven by an incremental effort, with $\tau = 30$s, $HR_0 = 50$ beats/min with a constant gain of 1 beat/min/W (red curve) and a gain decreasing with power (blue curve), both represented in panel C.

This method allows at the same time to model a simple allostasis, that is to account for a change of equilibrium value. Indeed, by artificially setting the power of the recovery period to 1W before the estimation of the first order differential equation, the equilibrium value after the effort will be $HR_0 + 1 \times K_{recovery}$, different from HR_0.

Sensitivity analysis

To ensure that the variation of the HR gain with power was not dependent of the number of steps N_{steps} considered for the decomposition of the incremental effort test power, we performed the above described dynamical analysis for different values of N_{steps}. The results are described in the Results section.

Statistical analysis

Associations between continuous variables of interest were assessed using Spearman correlation. Bonferoni correction was applied for the threshold of significance (Bonferroni, 1936). Associations between gain and power were analyzed using mixed effect linear regression with a random slope and intercept on each individual. All analyses were performed using R version 3.6.2 (R Core Team, 2019), the package *doremi* (Mongin et al., 2019) for the dynamical analysis, *lmer* and *lme4* (Bates et al., 2015) for the mixed effect linear regression, and the packages *data.table*, *Hmisc* and *ggplot2* (Wickham, 2016) for the data management, statistical indicators and graphical representation.

Results

Performance of the new dynamical analysis of HR with a variable gain is illustrated for one effort test in Figure 2 (panels A and C) and compared for the same effort test with the dynamical analysis with a constant gain (panels B and D). The estimation of the HR dynamics with a constant gain yields a median R^2 of 0.91 (interquartile range IQR = [0.89-0.95]), while the new dynamical analysis considering the power as a sum of constant steps yields a median R^2 of 0.994 (IQR: [0.987- 0.996]) (bottom panels).

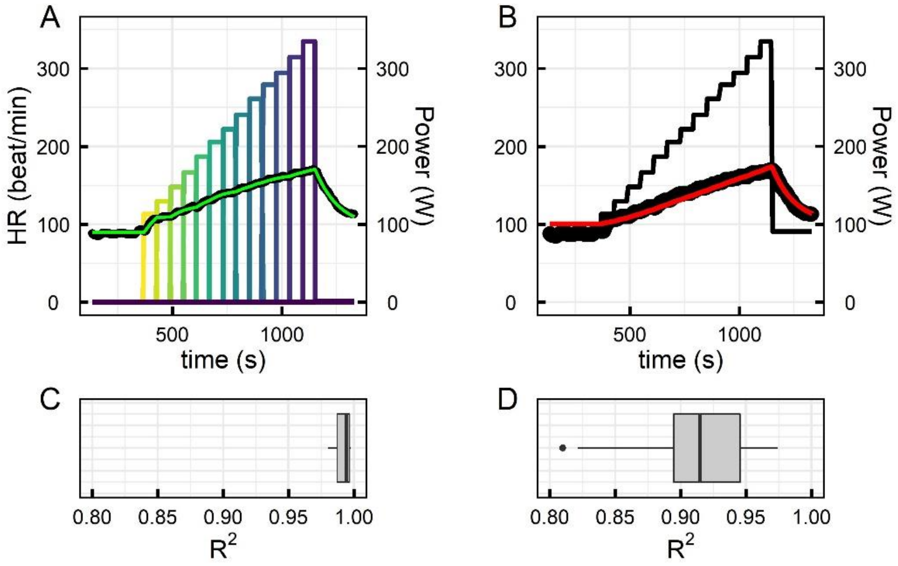


Figure 2. Example of fitted HR curve with a dynamical analysis considering the energy expenditure as a single one and a constant gain (B panel) or considering the energy expenditure as a sum of constant steps with a gain associated to each step (A). The boxplots below the graphs (C and D) represent the ensemble of the R^2 values of the estimations (A and B respectively).

The variable gain approach allows to estimate an unbiased equilibrium value HR_0, whereas as already previously seen (Mongin, Uribe Caparros, et al., 2020) the constant gain approach tends to overestimate it (see Supplementary Figure 1).

Since the dynamical change of the HR curve is linked to the ventilatory thresholds (Bodner and Rhodes, 2000), HR gains are described for power below the first threshold, between the two ventilatory thresholds and above the power corresponding to the lactate turn point. A representative measurement of the estimated HR gain in function of the power spent during exercise is displayed on Figure 3, together with the two ventilatory transitions estimated from the ventilatory measurements.

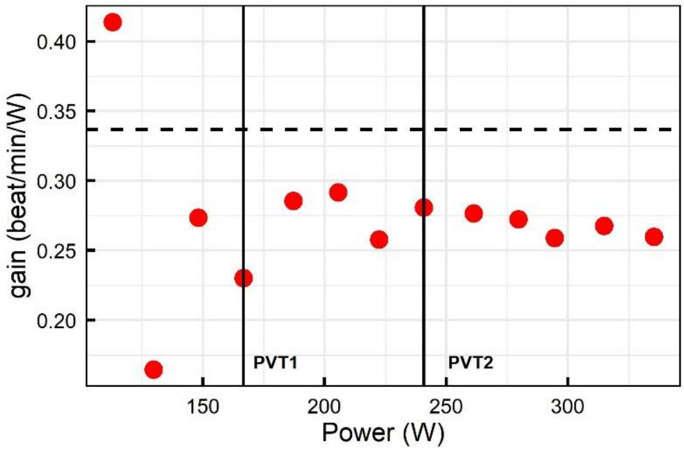


Figure 3. Example of HR gain (dots) for the different power values considered. The two vertical lines represent the two ventilatory thresholds, and the horizontal dashed line the value of the constant HR gain.

We examined the variation of HR gain with power using a linear mixed-effect model, considering a linear relation between HR gain and power with a random varying slope and intercept between the individuals. The estimated mean slopes with their corresponding standard errors are given in Table 2 for power below PVT1, between PVT1 and PVT2, or above PVT2. Before the first ventilatory transition, the gain varies greatly but without a specific trend, then decreases slightly between the two transitions and finally, it decreases strongly after the second transition (Table 2).

Table 2: Estimated linear trend of HR gain over time

Power range	Slope (beat/min/kW^2)	N	R^2	p
Power ≤ PVT1	-178 (391)	117	0.55	0.65
PVT1≤Power<PVT2	-316 (134)	130	0.80	0.02
PVT2 ≤ Power	-528 (55)	146	0.94	<0.001

Estimated fixed effects (standard error) of the linear trend of HR gain over power, for three power ranges: power below the first ventilatory threshold (Power ≤ PVT1), power between the two ventilatory thresholds (PVT1≤Power<PVT2) and power above the second ventilatory threshold (PVT2 ≤ Power). N indicates the total number of HR gain measurements considered in the mixed effect regression for the given power range. p is the p value associated with the slope estimates, and R^2 is the estimated proportion of explained variance of the regression.

The values of R^2 given in Table 2 illustrate that the gain varies considerably around its mean constant value for low powers, has a reduced variability around its decreasing trend between the two ventilatory thresholds, and stabilizes in a more pronounced decrease after the second threshold.

The mixed effect model allows to estimate the individual slopes of HR gain versus power, which can be used as an indicator of heart performance and function. The interquartile ranges of the individual slopes are [-697 ; -41] beat/min/kW2 for power between the two ventilatory thresholds and [-527 ; -486] beat/min/kW2 for power above the second ventilatory threshold. Table 3 shows how these slopes of HR gain with power correlate with other cardiovascular performance indices.

Table 3: Spearman correlation coefficients between measures of performance and heart gain decrease

	Trend in HR gain	
	PVT1<P<PVT2	P>PVT2
K_{HR}	-0.67*	-0.33
HRR	0.19	0.14
HR max	-0.38	-0.01
VO2 max	0.29	0.48*
Speed max	0.31	0.33
PVT1	--	--
PVT2	--	--
k_{HR}	-0.28	-0.57*

K_{HR}: Heart rate constant gain, VO2 max: maximal oxygen consumption, Speed max: maximal speed achieved during the effort test, PVT1 and PVT2: power at the ventilatory transitions; k_{HR}: HR curve deflection angle. Applying Bonferoni correction, significance is indicated as follows: *: p<0.004; **: p < 0.0008, ***: p < 0.00008

Between the two ventilatory thresholds, the decrease in gain is strongly associated with higher mean HR gain, meaning that athletes with a higher mean heart rate response to effort tend to have a stronger decrease of this response when power increases above the first ventilatory threshold. After the second threshold, high decrease rate of HR gain with power is correlated with lower VO_2 max. In other words, a higher maximal aerobic respiratory capacity is associated with a smaller decrease of the HR gain with power. Correlations between the constant HR gains obtained with analysis described in equation 1 and performance measurements are given in supplementary Table 2. These are similar to those obtained previously among younger athletes populations (Mongin, Chabert, et al., 2020; Mongin, Uribe Caparros, et al., 2020). We can observe that the correlation between the HR gain decrease and VO_2 max is higher and more significant than the one between the constant HR gain and VO_2 max. The decrease rate of HR gain with power above the lactic accumulation threshold is negatively correlated with the deflection angle of the HR curve measured with the Conconi technique, meaning that stronger deflections of the HR performance curve are associated with stronger decrease of the HR gain with power increase. The slopes of HR gain evolution with power for the two power zones considered are strongly correlated with each other (ρ=0.68, $p < 0.001$).

Sensitivity analysis

Decomposition of the effort test power in a fixed number of 8, 10, 12 and 14 parts did not qualitatively change the results. Specifically, the tendency of the evolution of the gain with power as presented in Table 2 stayed the same (see supplementary table 1): for each decomposition, the slope of the gain decrease is stronger, less variable and more significative above the second threshold than between the two ventilatory thresholds, whereas there is no significative change with power below the first ventilatory threshold. The correlations between the decrease of the HR gain with power and the other indices as shown in table 3 was also preserved. An example of the estimations of HR obtained by our dynamical analysis for the different N_{step} considered together with the values of gain estimated are presented in Supplementary Figure S1.

Discussion

Main findings

Considering multiple independent power steps in the dynamical analysis of heart rate allows indeed to study the heart rate dynamical changes occurring during effort tests and to account for the inherent allostasis of the heart functioning during physical exercise. This approach produced an estimation of HR accounting for more than 99% of its observed variation and allowed to estimate the HR response to each independent power steps considered. Before the first ventilatory threshold, the HR gain (i.e. the proportionality between HR increase and effort increase) was independent of energy expenditure and had an important variability. The HR gain started to decrease with power increase between the first and the second ventilatory thresholds and decreased more with power after the second ventilatory threshold. The slope of these decreases was correlated with the constant HR gain between the two ventilatory thresholds, and with VO_2 max and the HR curve deflection angle for power above the lactic accumulation threshold.

HR dynamics

The HR dynamics captured by the HR gain changes with power can be explained by the various metabolic energy systems at play during the exercise (Baker et al., 2010; Gastin, 2001).

At the onset of a physical effort, the energy needed for mechanic workload is first supplied by anaerobic pathways (including phosphagen then glycolysis systems), and progressively replaced by aerobic pathways after a few minutes of exercise (Gastin, 2001; Wells et al., 2009). In most of the graded exercise used, the fast intensity increase leads to the significant involvement of the main energetic pathways below the first ventilatory threshold. In this situation, a non-negligible part of the energy supply does not rely on exogenous oxygen nor substrates provided by blood compartment, causing the important variability of the HR gain measured by our analysis before VT1. Furthermore, heart rate adaptation at the onset of exercise is driven by both nervous and endocrine systems that have different response inertia, participating to the heart rate variability measured in this study at the beginning of exercise (Christensen and Galbo, 1983; Kjaer, 1989). Indeed, nervous and endocrine systems induce many adjustments of the cardiorespiratory system such as the muscle vasodilation and some internal organs vasoconstriction causing changes in peripheral resistance (Vatner and Pagani, 1976), progressive start of the thermoregulatory processes (Baker, 2019), and so change of heart workload.

Between VT1 and VT2, energy expenditure is mainly supplied by oxidation of substrates in mitochondria. In this range of intensity, oxidized substrates shift from lipids to glucose, the only substrate used at high intensity, and lactate starts to progressively accumulate faster than it can be eliminated (McArdle et al., 2010). Despite the better yield of glucose oxidation, a slight decrease of the HR gain with power is observed at this intensity range, which may be due to the negative effects of progressive lactate accumulation in muscle (Brooks and Mercier, 1994). Lactate may also explain the correlation observed between the mean HR gain and the HR gain decrease. The fact that athletes with higher mean HR gain, i.e. higher heart rate increase for a given energy increase, have a stronger decrease of the HR gain with power, i.e. a lower capacity to maintain their cardiac response when increasing effort, may be due to a better lactate management by athletes with a lower HR gain. This result is in line with current literature that describes the relation between performance capacity and development of lactate countermeasures (Stallknecht et al., 1998). Nonetheless, the almost exclusive participation of the aerobic pathway leads to the more stable HR gain observed, despite the physiological disturbances induced by lactate.

After VT2, aerobic then anaerobic glycolysis are the main contributors to the energy demand. At such intensity, all the physiological machinery is working to maintain mechanic workload without variations induced by metabolic energy systems inertia or lipid to glucid oxidation shift, causing the HR gain to become even more stable. However, the lactate countermeasures are overloaded, leading to a fast increase of lactate concentration and a decrease of the muscle pH that sharply affects muscle performance (Debold et al., 2008). The HR gain thus starts to decrease in a more pronounced manner with power increase, expressing the physiological limit to effort increase.

Our study shows that athletes with higher VO_2max have lower mean HR gain, and a lower decrease of HR gain with power, expressing their capacity to maintain a better cardiorespiratory response to the increasing energy demand. One possible explanation could be the higher aerobic energy supply capacity of these athletes, leading to a better ability to counteract blood lactate accumulation and so to a higher cardiac response for important efforts (Stallknecht et al., 1998). The better thermoregulation capacity of these athletes could also participate. Indeed, it can be assumed that at least 70% of all the energy expended from ATP hydrolysis is wasted as heat production (Ettema and Lorås, 2009), placing thermolysis as a key process of performance. Training increases the thermoregulation efficiency by enhancing the sweat glands, sensitivity, distribution, and output (Taylor, 1986) allowing to maintain a better muscle cell temperature. The regulation of muscle temperature is known to allow a lower heart rate for a given effort (Mostardi et al., 1974) because hyperthermia decreases the mechanic efficiency due to loss of enzymatic activity (Oliver et al., 2008) and neuromuscular fibers recruitment (Racinais and Oksa, 2010). This lower load on the heart rate is in line with the observed lower mean HR gain for trained athletes and could allow them to maintain a higher cardiac response to effort, thus explaining the lower decrease of the HR gain we observe.

Comparison with Conconi

The main objective of Conconi test is to determine VT2. Our analysis focuses more in the quantification of the HR dynamics changes. Our analysis provides a measurement similar to the deflection degree measured directly on the HR curve, as can be seen with the correlation between the HR gain decrease after the second ventilatory threshold and the deflection degree of the HR performance curve. But on the contrary to this measurement, (Bodner and Rhodes, 2000; Pokan et al., 1999) our approach is not dependent on the protocol (Artiga Gonzalez et al., 2017; Mongin, Chabert, et al., 2020; Mongin, Uribe Caparros, et al., 2020). The fact that the HR gain starts decreasing between the first ventilatory and the second ventilatory threshold could explain the difficulties encountered to systematically observe the deflection of the HR performance curve with the Conconi approach.

Comparison with dynamical approach with constant gain

While the dynamical analysis considering a constant gain gives an average measurement of the HR response to effort and is representative of the overall athlete performance, the dynamical analysis proposed in this study provides better HR estimates than the previous one, mainly due to its ability to model allostasis. It allows a deeper insight into HR dynamics with a noninvasive way to quantify the HR dynamic change during lactate production and accumulation, which constitutes a new and pertinent heart characterization index.

Strengths and limitations

The main strength of the study is the high estimation performance that the model is capable of, as well as the consistence with known physiological behaviors. The dynamical analysis is applicable to any kind of protocol. In addition, well-known indices were also calculated, to allow head-to-head comparison with the new method used.

There are two main limitations of this study. First, the sample size is relatively small, and the sample is only composed of amateur athletes. Thus, the sample is not very heterogeneous. Further studies should consider various effort tests, and diverse populations. The second limitation is the lack of lactate measurements, which would have allowed a direct testing of the association between HR gain and lactate management.

Conclusion

Dynamical analysis of heart rate with multiple independent power steps is a promising method to analyze effort tests. It provides a highly precise modeling of heart rate dynamics ($R^2 > 0.99$) with only a few parameters. HR gain and change in HR gain in particular yield good insights into the physiological underpinnings of athlete's performance during effort test.

Funding detail

This work was supported by the Swiss national science foundation (SNSF) under grant scientific exchange grant IZSEZ0_183540 and ICARUS SNSF fund 100019_166010.

Disclosure statement

The authors have no conflict of interest to report for the present work.

References

Artiga Gonzalez A, Bertschinger R, Brosda F, et al. (2017) Kinetic analysis of oxygen dynamics under a variable work rate. *Human Movement Science* In press. DOI: 10.1016/j.humov.2017.08.020.

Baker JS, McCormick MC and Robergs RA (2010) Interaction among Skeletal Muscle Metabolic Energy Systems during Intense Exercise. DOI: 10.1155/2010/905612.

Baker LB (2019) Physiology of sweat gland function: The roles of sweating and sweat composition in human health. *Temperature* 6(3). Taylor & Francis: 211–259. DOI: 10.1080/23328940.2019.1632145.

Bates D, Mächler M, Bolker B, et al. (2015) Fitting Linear Mixed-Effects Models Using **lme4**. *Journal of Statistical Software* 67(1). DOI: 10.18637/jss.v067.i01.

Bellenger CR, Fuller JT, Thomson RL, et al. (2016) Monitoring Athletic Training Status Through Autonomic Heart Rate Regulation: A Systematic Review and Meta-Analysis. *Sports Medicine (Auckland, N.Z.)* 46(10): 1461–1486. DOI: 10.1007/s40279-016-0484-2.

Bernard T, Gavarry O, Bermon S, et al. (1997) Relationships between oxygen consumption and heart rate in transitory and steady states of exercise and during recovery: Influence of type of exercise. *European journal of applied physiology and occupational physiology* 75: 170–6. DOI: 10.1007/s004210050143.

Bodner ME and Rhodes EC (2000) A review of the concept of the heart rate deflection point. *Sports Medicine (Auckland, N.Z.)* 30(1): 31–46. DOI: 10.2165/00007256-200030010-00004.

Bonferroni CE (1936) *Teoria statistica delle classi e calcolo delle probabilità*. Libreria internazionale Seeber.

Brooks GA and Mercier J (1994) Balance of carbohydrate and lipid utilization during exercise: the 'crossover' concept. *Journal of Applied Physiology (Bethesda, Md.: 1985)* 76(6): 2253–2261. DOI: 10.1152/jappl.1994.76.6.2253.

Bunc V, Heller J and Leso J (1988) Kinetics of heart rate responses to exercise. *Journal of Sports Sciences* 6(1): 39–48. DOI: 10.1080/02640418808729792.

Cabo JV, Martinez-Camblor P and del Valle M (2011) Validity of the Modified Conconi Test for Determining Ventilatory Threshold During On-Water Rowing. *Journal of Sports Science & Medicine* 10(4): 616–623.

Carey D (2002) Assessment of the accuracy of the Conconi test in determining gas analysis anaerobic threshold. *Journal of Strength and Conditioning Research* 16(4): 641–644.

Christensen NJ and Galbo H (1983) Sympathetic Nervous Activity During Exercise. *Annual Review of Physiology* 45(1): 139–153. DOI: 10.1146/annurev.ph.45.030183.001035.

Clark CCT, Barnes CM, Stratton G, et al. (2017) A Review of Emerging Analytical Techniques for Objective Physical Activity Measurement in Humans. *Sports Medicine (Auckland, N.Z.)* 47(3): 439–447. DOI: 10.1007/s40279-016-0585-y.

Cole CR, Blackstone EH, Pashkow FJ, et al. (1999) Heart-Rate Recovery Immediately after Exercise as a Predictor of Mortality. *New England Journal of Medicine* 341(18): 1351–1357. DOI: 10.1056/NEJM199910283411804.

Conconi F, Ferrari M, Ziglio PG, et al. (1982) Determination of the anaerobic threshold by a noninvasive field test in runners. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 52(4): 869–873. DOI: 10.1152/jappl.1982.52.4.869.

Debold EP, Beck SE and Warshaw DM (2008) Effect of low pH on single skeletal muscle myosin mechanics and kinetics. *American Journal of Physiology. Cell Physiology* 295(1): C173-179. DOI: 10.1152/ajpcell.00172.2008.

Ettema G and Lorås HW (2009) Efficiency in cycling: a review. *European Journal of Applied Physiology* 106(1): 1–14. DOI: 10.1007/s00421-009-1008-7.

Ferguson B (2014) ACSM's Guidelines for Exercise Testing and Prescription 9th Ed. 2014. *The Journal of the Canadian Chiropractic Association* 58(3): 328.

Gastin PB (2001) Energy system interaction and relative contribution during maximal exercise. *Sports Medicine (Auckland, N.Z.)* 31(10): 725–741. DOI: 10.2165/00007256-200131100-00003.

Hawley JA and Noakes TD (1992) Peak power output predicts maximal oxygen uptake and performance time in trained cyclists. *European Journal of Applied Physiology and Occupational Physiology* 65(1): 79–83. DOI: 10.1007/bf01466278.

Hofmann P, Pokan R, Preidler K, et al. (1994) Relationship between heart rate threshold, lactate turn point and myocardial function. *International Journal of Sports Medicine* 15(5): 232–237. DOI: 10.1055/s-2007-1021052.

Hofmann P, Pokan R, Von Duvillard SP, et al. (1997) Heart rate performance curve during incremental cycle ergometer exercise in healthy young male subjects. *Medicine & Science in Sports & Exercise* 29(6): 762.

Jones AM and Doust JH (1995) Lack of reliability in Conconi's heart rate deflection point. *International Journal of Sports Medicine* 16(8): 541–544. DOI: 10.1055/s-2007-973051.

Jones AM and Doust JH (1997) The Conconi test in not valid for estimation of the lactate turnpoint in runners. *Journal of Sports Sciences* 15(4): 385–394. DOI: 10.1080/026404197367173.

Kjaer M (1989) Epinephrine and some other hormonal responses to exercise in man: with special reference to physical training. *International Journal of Sports Medicine* 10(1): 2–15.

Kjertakov M, Dalip M, Hristovski R, et al. (2016) Prediction of lactate threshold using the modified Conconi test in distance runners. *Physiology International* 103(2): 262–270. DOI: 10.1556/036.103.2016.2.12.

Lambert MI, Mbambo ZH and Gibson ASC (1998) Heart rate during training and competition for longdistance running. *Journal of Sports Sciences* 16(sup1): 85–90. DOI: 10.1080/026404198366713.

Ludwig M, Hoffmann K, Endler S, et al. (2018) Measurement, Prediction, and Control of Individual Heart Rate Responses to Exercise-Basics and Options for Wearable Devices. *Frontiers in Physiology* 9: 778. DOI: 10.3389/fphys.2018.00778.

McArdle WD, Katch FI and Katch VL (2010) *Exercise Physiology: Nutrition, Energy, and Human Performance*. Lippincott Williams & Wilkins.

Mongin D, Uribe A and Courvoisier D (2019) doremi: Dynamics of Return to Equilibrium During Multiple Inputs. Available at: https://CRAN.R-project.org/package=doremi.

Mongin D, Uribe Caparros A, Gateau J, et al. (2020) Dynamical System Modeling of Self-Regulated Systems Undergoing Multiple Excitations: First Order Differential Equation Approach. *Multivariate Behavioral Research* 0(0). Routledge: 1–20. DOI: 10.1080/00273171.2020.1754155.

Mongin D, Chabert C, Caparros AU, et al. (2020) Validity of dynamical analysis to characterize heart rate and oxygen consumption during effort tests. *Scientific Reports*. DOI: 10.1038/s41598-020-69218-1.

Mostardi R, Kubica R, Veicsteinas A, et al. (1974) The effect of increased body temperature due to exercise on the heart rate and on the maximal aerobic power. *European Journal of Applied Physiology and Occupational Physiology* 33(3): 237–245. DOI: 10.1007/BF00421151.

Nobrega ACL, O'Leary D, Silva BM, et al. (2014) Neural Regulation of Cardiovascular Response to Exercise: Role of Central Command and Peripheral Afferents. DOI: https://doi.org/10.1155/2014/478965.

Oliver SR, Wright VP, Parinandi N, et al. (2008) Thermal tolerance of contractile function in oxidative skeletal muscle: no protection by antioxidants and reduced tolerance with eicosanoid enzyme inhibition. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology* 295(5). American Physiological Society: R1695–R1705. DOI: 10.1152/ajpregu.90429.2008.

Pokan R, Hofmann P, von Duvillard SP, et al. (1999) The heart rate turn point reliability and methodological aspects. *Medicine and Science in Sports and Exercise* 31(6): 903–907.

R Core Team (2019) *R: A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing. Available at: https://www.R-project.org.

Racinais S and Oksa J (2010) Temperature and neuromuscular function. *Scandinavian Journal of Medicine & Science in Sports* 20(s3): 1–18. DOI: 10.1111/j.1600-0838.2010.01204.x.

Rivera-Brown AM and Frontera WR (2012) Principles of Exercise Physiology: Responses to Acute Exercise and Long-term Adaptations to Training. *PM&R* 4(11): 797–804. DOI: 10.1016/j.pmrj.2012.10.007.

Savin WM, Davidson DM and Haskell WL (1982) Autonomic contribution to heart rate recovery from exercise in humans. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 53(6): 1572–1575. DOI: 10.1152/jappl.1982.53.6.1572.

Seborg DE, Mellichamp DA, Edgar TF, et al. (2010) *Process Dynamics and Control*. John Wiley & Sons.

Stallknecht B, Vissing J and Galbo H (1998) Lactate production and clearance in exercise. Effects of training. A mini-review. *Scandinavian Journal of Medicine & Science in Sports* 8(3): 127–131.

Taylor NAS (1986) Eccrine Sweat Glands. *Sports Medicine* 3(6): 387–397. DOI: 10.2165/00007256-198603060-00001.

Vatner SF and Pagani M (1976) Cardiovascular adjustments to exercise: Hemodynamics and mechanisms. *Progress in Cardiovascular Diseases* 19(2): 91–108. DOI: 10.1016/0033-0620(76)90018-9.

Wasserman K, Whipp BJ, Koyl SN, et al. (1973) Anaerobic threshold and respiratory gas exchange during exercise. *Journal of Applied Physiology* 35(2): 236–243. DOI: 10.1152/jappl.1973.35.2.236.

Wells GD, Selvadurai H and Tein I (2009) Bioenergetic provision of energy for muscular activity. *Paediatric Respiratory Reviews* 10(3): 83–90. DOI: 10.1016/j.prrv.2009.04.005.

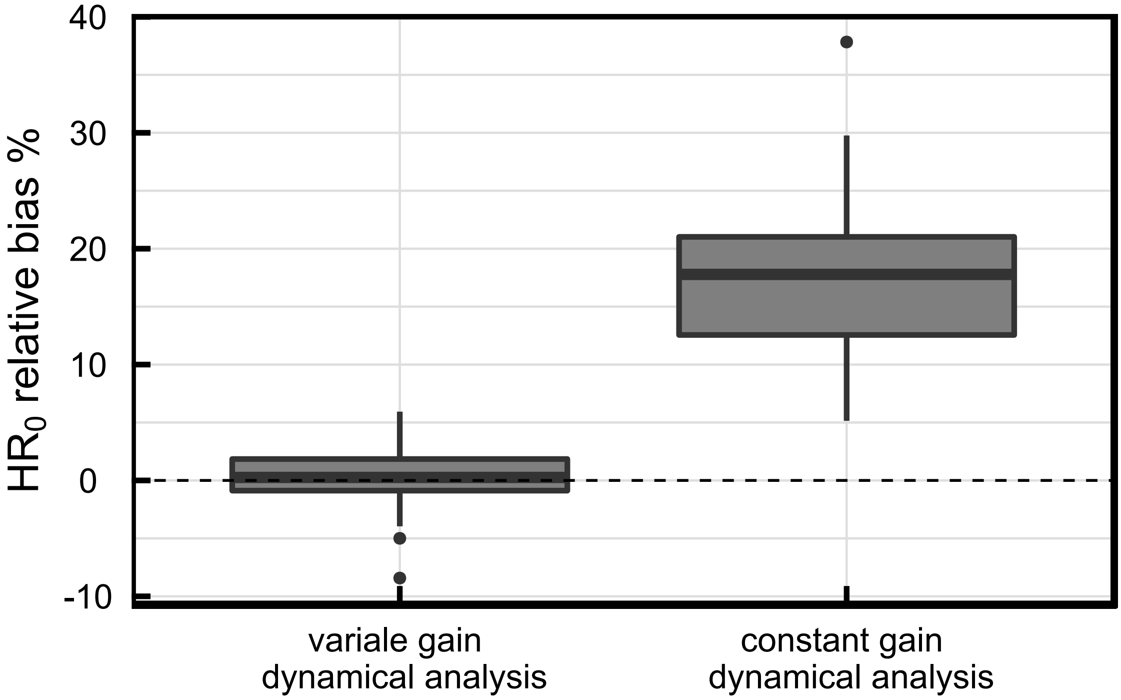
White DW and Raven PB (2014) Autonomic neural control of heart rate during dynamic exercise: revisited. *The Journal of Physiology* 592(12): 2491–2500. DOI: 10.1113/jphysiol.2014.271858.

Wickham H (2016) *Ggplot2: Elegant Graphics for Data Analysis*. Springer-Verlag New York. Available at: https://ggplot2.tidyverse.org.

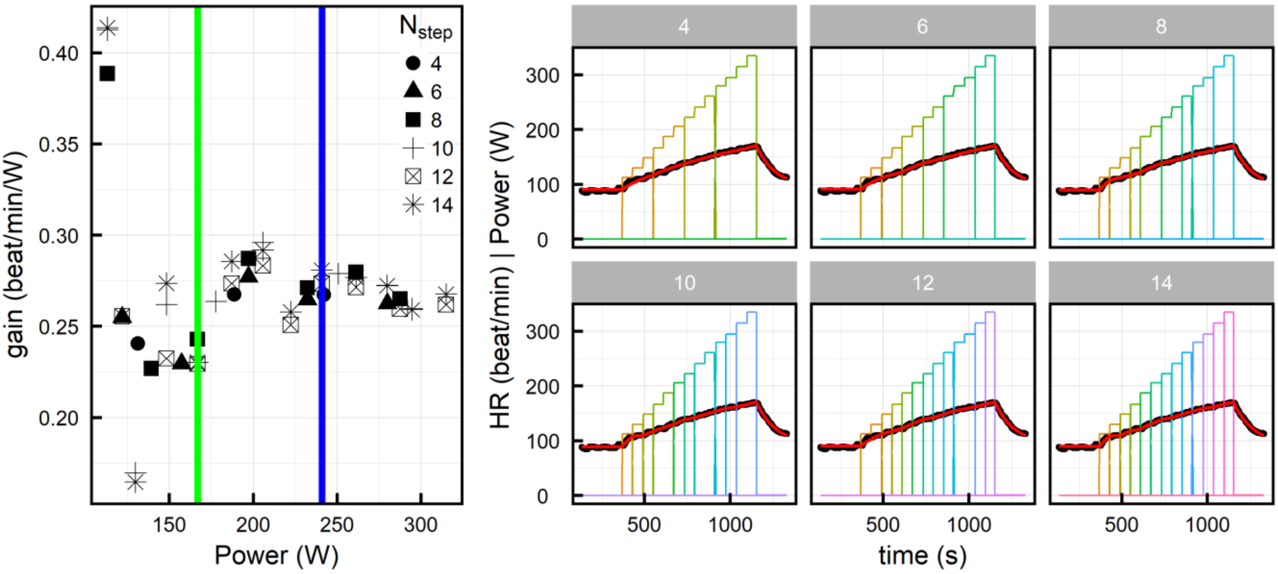
Wyss CR, Brengelmann GL, Johnson JM, et al. (1974) Control of skin blood flow, sweating, and heart rate: role of skin vs. core temperature. *Journal of Applied Physiology* 36(6): 726–733. DOI: 10.1152/jappl.1974.36.6.726.

Zakynthinaki MS (2015) Modelling Heart Rate Kinetics. *PLOS ONE* 10(4): e0118263. DOI: 10.1371/journal.pone.0118263.

Supplementary Figure S1: Relative bias of the estimated equilibrium value compared to the actual mean HR value before the beginning of the incremental effort test, for the two analysis.



Supplementary Figure S2: right: HR estimation (red line) compared to the experimental HR (black dots) produced by our dynamical analysis for 6 different energy expenditure decomposition (coloured solid line, for N_{step} = 4, 6, 8, 10, 12, 14). Left: HR gain estimated from the different analysis presented in the right panel.



Slope (beat/min/kW^2)	N	R2	Power range	pval	N power steps
-559 (66)	84	0.94	PVT2 ≤ Power	<0.001	8
-567 (62)	109	0.91	PVT2 ≤ Power	<0.001	10
-533 (61)	127	0.91	PVT2 ≤ Power	<0.001	12
-528 (59)	137	0.91	PVT2 ≤ Power	<0.001	14
-529 (175)	81	0.78	PVT1≤Power<PVT2	0.0038	8
-533 (168)	101	0.81	PVT1≤Power<PVT2	0.0026	10
-500 (161)	115	0.82	PVT1≤Power<PVT2	0.0035	12
-365 (129)	120	0.79	PVT1≤Power<PVT2	0.0056	14
-71 (484)	83	0.50	Power ≤ PVT1	0.88	8
-18 (469)	99	0.50	Power ≤ PVT1	0.97	10
-4773 (3127)	109	0.59	Power ≤ PVT1	0.14	12
-6222 (2902)	122	0.56	Power ≤ PVT1	0.046	14

Supplementary table 1: Estimated fixed effects (standard error) of the linear trend of HR gain over power, for three power ranges (power below the first ventilatory threshold (Power ≤ PVT1), power between the two ventilatory thresholds (PVT1≤Power<PVT2) and power above the second ventilatory threshold (PVT2 ≤ Power)) and 4 different decompositions of the power exerted during effort (power decomposed in 8, 10, 12 or 14 different power steps). N indicates the total number of HR gain measurements considered in the mixed effect regression for the given power range. p is the p value associated with the slope estimates, and R^2 is the R square of the regression.

	K_{HR}
K_{HR}	
HRR	0.13
HR max	0.15
VO2 max	-0.41
Speed max	-0.50*
PVT1	-0.37*
PVT2	-0.70**
k_{HR}	0.32

Supplementary Table 2: Spearman correlation coefficients between measures of performance and constant heart gain. K_{HR}: Heart rate constant gain, VO2 max: maximal oxygen consumption, Speed max: maximal speed achieved during the effort test, PVT1 and PVT2: power at the ventilatory transitions; k_{HR}: HR curve deflection angle. Applying Bonferoni correction, significance is indicated as follows: *: $p<0.007$; **: $p < 0.001$, ***: $p < 0.0001$