Model-Based Heart Rate Prediction during Lokomat Walking

Alexander C. Koenig, *IEEE Student Member*, Luca Somaini, Michael Pulfer, Thomas Holenstein,
Ximena Omlin, Martin Wieser, Robert Riener, *IEEE Member*

Abstract— We implemented a model for prediction of heart rate during Lokomat walking. Using this model, we can predict potential overstressing of the patient and adapt the physical load accordingly. Current models for treadmill based heart rate control neglect the fact that the interaction torques between Lokomat and human can have a significant effect on heart rate. Tests with five healthy subjects lead to a model of sixth order with walking speed and power expenditure as inputs and heart rate prediction as output. Recordings with five different subjects were used for model validation. Future work includes model identification and predictive heart rate control with spinal cord injured and stroke patients.

I. INTRODUCTION

Treadmill training is part of the rehabilitation administered to patients after stroke, spinal cord injury or traumatic brain injury. The length and intensity of training was found to have significant impact on the rehabilitation success [13]. As manual treadmill training is exhausting for patients and therapists alike, the driven gait orthosis Lokomat was developed to automate treadmill training and allow longer training durations [6].

Monitoring and controlling heart rate during exercise can be crucial for preventing overtraining and in providing an efficient training protocol to subjects [1]. We expect control of heart rate during Lokomat exercise to be beneficial to neurological patients for two reasons. On the one hand, the heart rate should be monitored during training for security concerns. The Lokomat should take preventive counter measures before the patient becomes physically overstressed and reduce the amount of physical load the patient has to carry. On the other hand, a control mode which stabilizes the heart rate at a desired value would offer the possibility of patient specific cardiovascular training in the early phase of rehabilitation in the Lokomat.

Heart rate control based on control of treadmill speed has been employed previously using PID controllers and fuzzy logic [16] as well as robust control [4], [5]. All of these controllers were developed for subjects running on a treadmill up to 8km/h. That makes them inapplicable for the Lokomat, which is limited to walking speeds up to 3.2

All authors are with the Sensory Motor Systems Lab, ETH Zurich, Institute of Robotics and Intelligent Systems, Department of Mechanical Engineering and Process Engineering, ETH Zurich, Switzerland.

A. Koenig and R. Riener are also with the Spinal Cord Injury Center, University Hospital Balgrist, University of Zurich. koenig@mavt.ethz.ch

km/h for patient safety. The only controller with explicit constraints on the control input was the approach of Su [15], who developed a model predictive controller with an upper boundary on treadmill velocity and treadmill acceleration.

Previously developed cardiovascular models are not applicable to the Lokomat, as the patient can walk either actively or passively in the Lokomat. Previously, we showed that this difference in physical effort (or power exchanged between Lokomat and human) results in significantly different heart rates ($p \leq 0.05$) [11]. Large forces are employed by the orthosis that guides the patient's legs on the reference trajectory. The patients have the possibility to walk actively, thereby pushing with high forces into the orthosis, or behave passively and getting moved by the Lokomat.

Based on a cardiovascular model that describes the dependency between heart rate and the treadmill speed, we want to predict the heart rate of subjects walking in the Lokomat gait orthosis. As our approach is model-based, it can foresee the temporal evolution of the heart rate and can predict when the heart rate might increase over a security threshold caused by the physical stress of the Lokomat training. The model will also be the basis for model-based control during Lokomat walking, in which the controller takes the power exchanged between Lokomat and patient into account.

II. METHODS

A. Power during Lokomat walking

As active or passive behavior during Lokomat walking will result in significantly different heart rates, we had to include the influence of power expenditure of the human during the training as part of the model. The power output during exercise on a bicycle ergometer [18], [3], [2] and during arm cranking [3] were reported to correlate linearly with heart rate. Power during Lokomat walking can be computed as

$$P_{Lokomat} = \tau^T \dot{q}, \tag{1}$$

where τ and $\dot{q}$ refer to the moments and angular velocities exerted by the subject on the Lokomat. Due to the sensor placement in the Lokomat, we can only record the combination of torques exerted by the human and the torques exerted by the Lokomat actuators. Having recorded the gait trajectory, we computed the torques that would be needed to move the empty Lokomat on this trajectory and subtract it from the recorded torques. This results in the torque exchanged between Lokomat and human. If these torques are positive, the human has provided kinetic energy to the Lokomat, which means that the Lokomat had to slow the human down. A negative torque means that the Lokomat
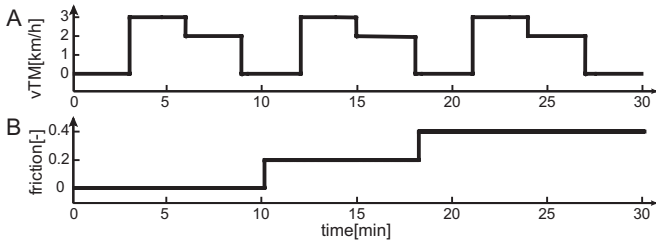
Fig. 1. Speed profile (A) and friction profile (B) for model identification experiments

Fig. 2. The heart rate model as a block chart. Input are the treadmill speed and the power exchanged between the human and the Lokomat

provided additional torque to the human, who was not generating enough torque to walk actively.

B. Model identification

To define a model for the cardiovascular process of subjects during Lokomat walking, we identified the main effects of change in treadmill speed and change in power output on the heart rate. We extracted heart rate from ECG recordings using a steep slope algorithm. We recorded heart rate data at different walking speeds (Fig. 1) from five subjects (3male, 25.0 years$\pm$2.3, 77.2kg$\pm$8.0) subtracted baseline heart rate during standing and averaged the resulting heart rate increase. For investigation of effects of power expenditure on the heart rate, we applied a velocity dependent friction force onto the gait orthosis. The friction was scaled such that 100% friction force simulated walking in hip deep water as previously described in [12]. We recorded the total power when subjects walked in zero impedance mode at different levels of treadmill speed and three different levels of friction. Friction was computed as

$$F_{friction} = \nu v_{TM}^2 \qquad (2)$$

where ν is the friction coefficient and v_{TM} the Lokomat treadmill speed. The friction force was projected back as torques onto the orthosis. This friction caused additional power expenditure adding up to the expenditure related to walking.

Inputs to the model were treadmill speed and power expenditure of the human; output of the model was the change in heart rate from baseline heart rate recorded during standing. We used Matlab Simulink 2007b (www.mathworks.com) for implementation of real time heart rate prediction. Increases in walking speed linearly increase heart rate [7], [9], [14], [1], which we modeled as a first order PT element. This can be interpreted as lowpass-like reaction to a sudden increase of oxygen demand. A treadmill acceleration results in an overshoot in heart rate before steady state is reached [7], [9], which we modeled as second order DT element. Holmgreen reported a drop in arterial pressure that reached its minimum 10 seconds after onset of exercise [10]. The heart rate overshoot might be caused by a first overreaction of the cardiovascular system to compensate for the blood pressure drop. Feroldi et al argued that the overshoot might be a result of changes in the balance between sympathetic and parasympathetic activity [8]. An undershoot can be observed
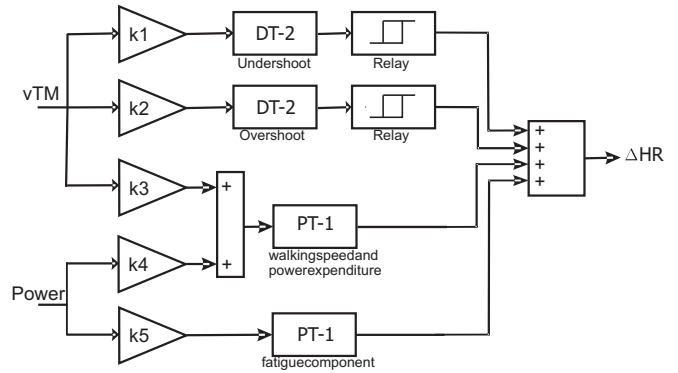
after a negative acceleration, modeled as a second order DT element as well. To separate the overshoot and undershoot dynamics, a nonlinearity was inserted after the overshoot and undershoot dynamics. The output of the undershoot was only added to ΔHR (Fig. 2), if its value was negative, whereas the output of the overshoot was added to ΔHR if its value was positive. This was implemented using relay blocks in Simulink, which made the model nonlinear. The power expenditure of the human was taken as a linear input parameter modeled as a first order PT element. After longer training durations, a fatigue effect is present [7], [9], [17] that results in a higher resting heart rate after a physically demanding task than before the tasks. This fatigue effect was modeled as a first order PT element. All inputs were scaled with a scaling factor. This resulted in a model with eleven parameters: five scaling factors and six time constants (Fig. 2).

C. Intra and inter subject variability

To perform the parameter identification of single subjects, we investigated the parameter variability inter and intra subject. Inter subject variability refers to the question, if some parameters can be seen as constant across subjects and which ones are left to be subject specific. As the system is not convex, we used a genetic algorithm to minimize the squared error between predicted and recorded data. We compared the optimized parameters of all subjects and analyzed which ones could be seen as subject specific and which ones could be set constant for all subjects. Intra subject variability refers to the question, if subjects needed to be re-identified before each training or if previously identified parameters could be reused. We recorded three data sets from two subjects (both male,24 and 25 years) with the same velocity profile in the Lokomat. Twice on day one (morning, afternoon) and once on day two (afternoon).

D. Model verification

We used the r^2 coefficient of determination for evaluation of our simulation results. To verify our model, we used a speed and friction profile shown in (Fig. 3). Five subjects walked in the profile (3male, 24.6 years$\pm$0.6, 69.0kg$\pm$10.8).
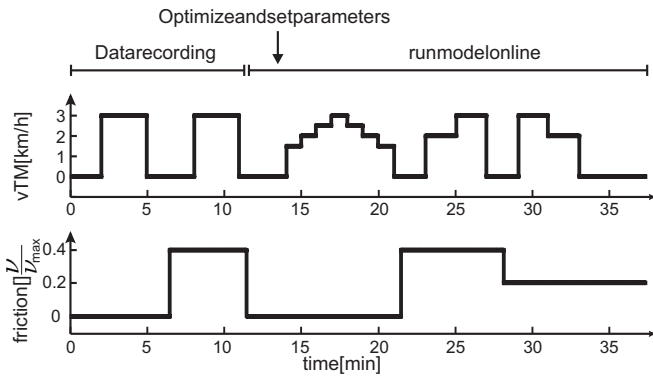
Fig. 3. Verification and validation of the heart rate model. A: Speed profile B: friction profile
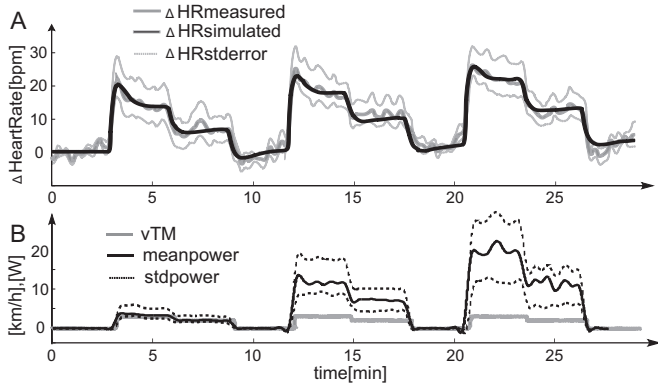

Fig. 4. A: Mean heart rate and standard error of five subjects for model identification using the speed and friction profile shown in B.

We first optimized over the whole dataset to obtain an optimal prediction of the recorded heart rate.

E. Heart rate prediction

For the future application of real time heart rate control, we need to identify the subject specific parameters at each training anew. This has to be done as quickly as possible, as one training session is typically between 30 and 45 minutes long. We performed an online identification of subject specific parameters, optimizing only over the first 13 minutes of the speed profile in (Fig. 3). After the first 13 minutes, the model parameters were fixed and used for heart rate prediction. We compared the r^2 values for the whole trial and the optimal parameters for both cases.

III. RESULTS

A. Results for model ID

We identified the eleven parameters of our heart rate model for the averaged heart rate curve of five healthy subjects during Lokomat walking. Our data showed the dependency of heart rate on treadmill speed and power expenditure as depicted in Figure 4.

Initial trials showed that subjects had similar heart rates in reaction to the velocity profile shown in (Fig. 1) when recorded on consecutive days at the same time. Recordings at different daytimes showed different results in heart rate

	R^2 for optimization of eleven parameter	R^2 for optimization of four parameters
Subject 1	0.90	0.90
Subject 2	0.32	0.30
Subject 3	0.93	0.88
Subject 4	0.93	0.92
Subject 5	0.94	0.94

(data not shown). We therefore decided to re-identify each subject before the Lokomat training.

Optimizing over all 11 parameters took several hours on a standard Pentium IV with 2GHz. It was therefore desirable to reduce the amount of parameters needing to be optimized. Optimization for each individual dataset over all eleven parameters revealed that only the scaling gains were subject specific. All parameters of the dynamics (overshoot, undershoot and the fatigue parameters) remained within plus minus 10% of their respective means. The same was true for the slow exhaustion gain parameter. They were therefore set constant. All other gain parameters were subjective, time specific and needed to be identified for each training session and for each subject anew.

We quantified the loss in prediction quality caused by optimizing over 4 instead of 11 parameters. Using 11 optimized parameters, we obtained coefficients of determination larger than 88% for all but one subject. Only subject 2 could not be identified properly. This was due to the fact that the intervention (increase in treadmill speed) did not cause a significant increase in heart rate. The heart rate increase caused by the intervention was in average 5 beats per minute, which is too close to the heart rate variability of healthy subjects (4 beats per minute).

The r^2 values of optimizing over the whole dataset with eleven parameters are compared with the r^2 values in Table I for an optimization with seven globally constant values and four variable parameters. In exchange for a mean decrease of r^2 of 0.02, we were able to reduce the time necessary for optimization to approximately two minutes.

B. Model verification

Model verification was performed with the velocity and friction profile shown in Fig. 3. The best result is shown in Fig. 5. When we reduced the identification time to 13 minutes (see Methods section), we saw only a slight decrease in prediction quality. (Table II).

IV. DISCUSSION AND OUTLOOK

For most subjects, the identification could be performed within the first 13 minutes of Lokomat training. The performance of our model was thereby limited by two factors: the heart rate variability (HVR) and the signal to noise ratio (SNR). With increasing HRV, the r^2 values droped, as we did
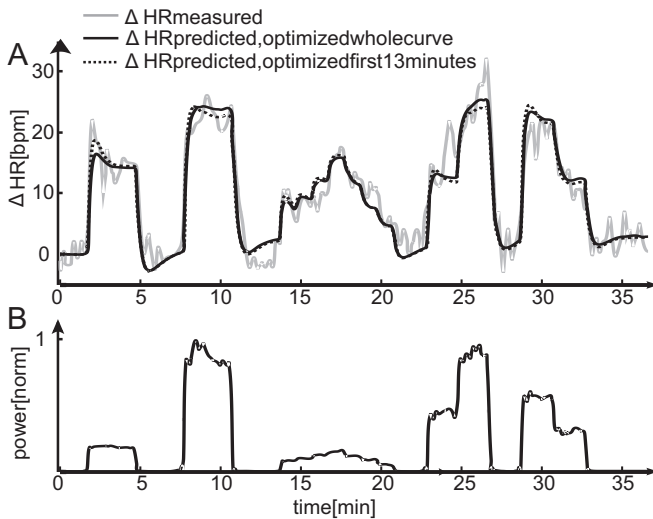
Fig. 5. Best result plot of subject 5 of the model verification. A: Recorded heart rate (gray) and two model predictions. Once optimized with four parameters and seven constants over the whole curve (black, solid) and only optimized over the first 13 minutes (black - dashed). B: Speed and power profile during the validation.

TABLE II

PREDICTION QUALITY OF THE HEART RATE MODEL WITH OPTIMIZATION OF 4 PARAMETERS. COMPARISON BETWEEN AN OPTIMIZATION OVER THE WHOLE DATA WITH AN OPTIMIZATION WHICH OPITMIZES OVER THE FIRST 13 MINUTES AND THEN PREDICTS THE REST OF THE DATASET. SUBJECT 1 AND 2 WERE BOTH RECORDED THREE TIMES.

Subject	Recording number	R^2 for optimization over entire dataset	R^2 for optimization over first 13 min
1	1	0.59	0.47
	2	0.82	0.81
	3	0.73	0.68
2	1	0.90	0.89
	2	0.73	0.67
	3	0.91	0.91
3	1	0.68	0.28
4	1	0.75	0.72
5	1	0.86	0.81

not explicitly model HRV. But it is not desirable to control HRV to zero, as a very low HRV can indicate a cardiac disease and down-regulation of HRV might become dangerous for the subject. Seeing the heart rate variability as noise, the controller can only perform well, when the intervention, i.e. the treadmill acceleration, causes an increase of heart rate that is clearly above the HRV. We only performed tests with healthy, well trained subjects, some of which showed little increase in HR for a treadmill speed up to 3.2km/h. The subjects with the worst r^2 results were also the ones that showed the smallest reaction to our intervention.

The model identification process suggests adaptive control, which could identify the model parameters online. However, heart rate is not only influenced by physical effort, but also by psychological events such as frightening, stressful or relaxing external stimuli. As these stimuli potentially elicit increases in heart rate due to stress, we decided to not use adaptive control, as we do not want our model to learn these quickly changing psychological responses.

We are currently working on implementing model predictive heart rate control of healthy subjects, necessitating performed validation of our model with data from spinal cord injury and stroke patients. In these patients, the mechanisms that control the heart rate, like the baroreflex or vaso-dihilation, might be imparied as response to physical effort. On the other hand, we expect a much larger increase in heart rate in patients caused by a treadmill speed of 3.2km/h, which would increase SNR and thereby increase the performance a model based controller.

REFERENCES

[1] J. Achten and A. E. Jeukendrup, "Heart rate monitoring: applications and limitations," *Sports Med*, vol. 33, pp. 517-38, 2003.
[2] F. J. Arts and H. Kuipers, "The relation between power output, oxygen uptake and heart rate in male athletes," *Int J Sports Med*, vol. 15, pp. 228-31, Jul 1994.
[3] G. Borg, P. Hassmen, and M. Lagerstrom, "Perceived exertion related to heart rate and blood lactate during arm and leg exercise," *Eur J Appl Physiol Occup Physiol*, vol. 56, pp. 679-85, 1987.
[4] T. Cheng, S. A, S. Su, B. Celler, and L. Wang, "A robust control design for heart rate tracking during exercise," in *IEEE EMBS* Vancouver, 2008, p. 4.
[5] T. Cheng, A. Savkin, B. Celler, S. Su, and L. Wang, "Heart Rate Regulation During Exercise with Various Loads: Identification and Nonlinear H infinity Control," in *17th World Congress of the International Federation of Automatic Control*, Seoul, Korea, 2008, p. 6.
[6] G. Colombo, M. Joerg, R. Schreier, and V. Dietz, "Treadmill training of paraplegic patients using a robotic orthosis," *J Rehabil Res Dev*, vol. 37, pp. 693-700, Nov-Dec 2000.
[7] L. Fagraeus and D. Linnarsson, "Autonomic origin of heart rate fluctuations at the onset of muscular exercise," *J Appl Physiol*, vol. 40, pp. 679-82, May 1976.
[8] P. Feroldi, M. Belleri, G. Ferretti, and A. Veicsteinas, "Heart rate overshoot at the beginning of muscle exercise," *Eur J Appl Physiol Occup Physiol*, vol. 65, pp. 8-12, 1992.
[9] M. Hajek, J. Potucek, and V. Brodan, "Mathematical-model of heart rate regulation during exercise," *Automatica*, vol. 16, p. 5, 1980.
[10] A. Holmgren, "Circulatory changes during muscular work in man; with special reference to arterial and central venous pressures in the systemic circulation," *Scand J Clin Lab Invest*, vol. 8, pp. 1-97, 1956.
[11] A. Koenig, M. Bolliger, X. Omlin, M. Wieser, and R. Riener, "Controlling physiology during robot automated treadmill training," in *Technically assisted rehabilitation (TAR)*, vol. in press Berlin, Germany, 2009.
[12] A. Koenig, M. Wellner, S. Koneke, A. Meyer-Heim, L. Lunenburger, and R. Riener, "Virtual gait training for children with cerebral palsy using the Lokomat gait orthosis," *Stud Health Technol Inform*, vol. 132, pp. 204-9, 2008.
[13] G. Kwakkel, B. J. Kollen, and R. C. Wagenaar, "Long term effects of intensity of upper and lower limb training after stroke: a randomised trial," *J Neurol Neurosurg Psychiatry*, vol. 72, pp. 473-9, Apr 2002.
[14] G. L. Pierpont, D. R. Stolpman, and C. C. Gornick, "Heart rate recovery post-exercise as an index of parasympathetic activity," *J Auton Nerv Syst*, vol. 80, pp. 169-74, May 12 2000.
[15] S. Su, S. Huang, L. Wang, B. Celler, A. Savkin, Y. Guo, and T. Cheng, "Nonparametric Hammerstein Model Based Model Predictive Control for Heart Rate Regulation," in *IEEE EMBS*, Lyon, France, 2007.
[16] S. Su, L. Wang, B. Celler, and A. Savkin, "Heart Rate Control During Treadmill Exercise," in *IEEE Conference in Engineering in Medicine and Biology (EMBC)* Shanghai, China, 2005, p. 4.
[17] S. W. Su, L. Wang, B. G. Celler, A. V. Savkin, and Y. Guo, "Identification and control for heart rate regulation during treadmill exercise," *IEEE Trans Biomed Eng*, vol. 54, pp. 1238-46, Jul 2007.
[18] Z. Vokac, H. Bell, E. Bautz-Holter, and K. Rodahl, "Oxygen uptake/heart rate relationship in leg and arm exercise, sitting and standing," *J Appl Physiol*, vol. 39, pp. 54-9, Jul 1975.