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Model-Based Heart Rate Control during Robot-Assisted Gait Training

Alexander Koenig, Student Member IEEE, Antonello Caruso, Student Member IEEE, Marc Bolliger, Luca Somaini, Ximena Omlin, Manfred Morari, Member IEEE and Robert Riener, Member IEEE

Abstract— **In recent years, gait robots have become increasingly common for gait rehabilitation in non-ambulatory stroke patients. Cardiovascular treadmill training, which has been shown to provide great benefit to stroke survivors, cannot be performed with non-ambulatory patients. We therefore integrated cardiovascular training in robot-assisted gait therapy to combine the benefits of both training modi. We developed a model of human heart rate as a function of exercise parameters during robot-assisted gait training and applied it for automatic control purposes. This structural model of the physiological processes describes the change in heart rate caused by treadmill speed and the power exchanged between robot and subject. We performed physiological parameter estimation for each tested individual and designed a model-based feedback controller to guide heart rate to a desired time profile. Five healthy subjects and eight stroke patients were recorded for model parameter identification, which was successfully used for heart rate control of three healthy subjects. We showed that a model-based control approach can take into account patient-specific limitations of treadmill speed as well as individual power expenditure.**

I. Introduction

During gait rehabilitation of stroke survivors, cardiovascular training can be of great benefit to the patient [1]. Depending on the degree of impairments caused by the lesion, this training is performed either on treadmills for less severe cases or on stationary bicycles in severely affected patients. Particularly non-ambulatory patients cannot exercise on treadmills, but must use stationary bicycles, where the problems of coordination and balance during walking do not need to be taken into consideration.

Besides cardiovascular training, coordinative gait training play a major role in rehabilitation of stroke survivors [2]. Gait robots such as the Lokomat [3], the WalkTrainer [4] and the LOPES robot [5] allow even non-ambulatory patients to exercise walking by guiding the legs of the patient on a walking trajectory and were shown to cause significant improvement of gait function in patients suffering from stroke [6], [7].

Today, cardiovascular gait-training with robotic assistance has not been used with neurological patients, as patients can be too impaired to walk on a treadmill at speeds which would permit control of heart rate (HR). The present work is an effort to integrate cardiovascular training into robot-assisted gait training with the goal to combine the benefits of both training modi and to make gait training more efficient.

While treadmill-based HR control is well established in healthy subjects [8]–[10], none of these approaches can be used in stroke patients during robot-assisted gait training, as they do not address three major differences compared to standard treadmill walking. First, for patient safety, treadmill speed during robot-assisted rehabilitation training is typically limited to very slow walking speeds and does not allow fast walking or running. The Lokomat gait orthosis for example is limited to 3.2km/h, which is low compared to previous approaches, where HR control was performed with walking speeds starting at 3.6 km/h [10]. Second, for facilitation of stance, the patient can be body weight supported, which will decrease HR with increasing body weight support (BWS) [11]. And third, all gait robots use actuators to provide supportive guidance force (GF) in order to enable the walking movement in patients with little leg force or little coordinative capabilities. This GF can be expected to alter HR, as it decreases the energy required by the subject to perform the walking movement.

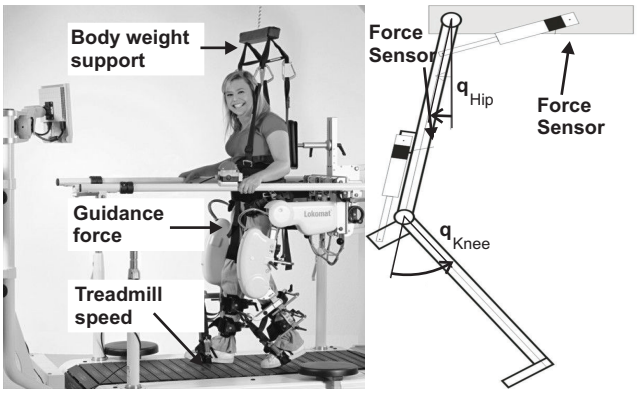

Fig. 1. The Lokomat gait orthosis. Left: Motors at hip and knee produce torques which move the subject's legs on a gait trajectory. The ankles are not actuated but kept in dorsiflexion to prevent foot drop (photo courtesy of Hocoma AG.). Right: schematic drawing of the orthosis. Potentiometers sense the position of the orthosis hip and knee joint, force sensors in series with the motors sense the interaction force between Lokomat and human.

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A.K., M.B., X.O. and R.R. are with the Sensory-Motor Systems Lab, ETH Zurich, Institute of Robotics and Autonomous Systems, Department of Mechanical Engineering and Process Engineering, ETH Zurich, Switzerland and also with the Spinal Cord Injury Center, University Hospital Balgrist, University of Zurich (email: koenig | bolliger | ximena.omlin | riener@mavt.ethz.ch).

A.C. and M.M. are with the Automatic Control Laboratory, ETH Zurich, Switzerland (email: caruso | morari@control.ee.ethz.ch).

L.S. was with the Sensory-Motor Systems Lab, ETH Zurich, Institute of Robotics and Autonomous Systems, Department of Mechanical Engineering and Process Engineering, ETH Zurich and is now with Hocoma Inc., Volketswil, Switzerland (email: luca.somaini@hocoma.com).

Taking these challenges into account, we first developed a structural model of physiological processes, describing the change in HR caused by changes of the Lokomat's treadmill speed, supportive force and the amount of BWS. We then estimated the physiological parameters of the model and designed a model-based feedback controller to guide HR to a desired time profile during Lokomat walking.

II. DEVELOPMENT OF A HEART RATE MODEL

To develop a model of HR as a function of robot-assisted gait training, we first analyzed the settings of a gait robot, which would influence HR. As power expenditure of a subject was shown to have major influence on HR, we analyzed the power exchange between an exoskeleton gait robot and a subject. To parameterize a model, we then performed one experiment with five healthy subjects and two experiments with a total of eight stroke patients.

A. Power exchange during Lokomat walking

During robot assisted gait training, the robot can exert large forces onto the patient's legs to guide them on a reference trajectory. This power exchange between the device and the patient has a major effect on the HR. At high GFs, i.e. with a stiff impedance controller, the patients have the possibility to walk actively, i.e. pushing into the orthosis with high forces, or behave passively, letting the robot move their legs.

We consider the torques exchanged between human and orthosis as the dominant port for power exchange in the system of Fig. 1. Due to the sensor placement in the Lokomat, we can only record the torques exchanged between the Lokomat's drives and the exoskeleton. Using the recorded gait trajectory, we can compute the torques necessary to move the exoskeleton on this trajectory and subtract these from the recorded torques. This permits us to determine the torques and thereby the power exchanged between Lokomat and human.

The power in the Lokomat during walking ($P_{Lokomat}$) can be computed as

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

where $\tau = [\tau_{hip\ left}\ \ \tau_{knee\ left}\ \ \tau_{hip\ right}\ \ \tau_{knee\ right}]^T$ is the interaction torque between the human and the Lokomat and $\dot{q} = [\dot{q}_{hip\ left}\ \ \dot{q}_{knee\ left}\ \ \dot{q}_{hip\ right}\ \ \dot{q}_{knee\ right}]^T$ is the angular velocity of the orthosis. This power gives an indication how active the human is. Positive values indicate that the human walks actively and the Lokomat has to brake. Negative power means that the Lokomat assists the human.

B. Model identification in healthy subjects

Parts of model identification in healthy subjects have been reported previously and are briefly repeated here for the sake of clarity [12]. The electrocardiogram (ECG) was recorded in five healthy individuals to define a model for the cardiovascular process of subjects during Lokomat walking (3 m and 2 f, 25.0 yr $\pm$ 2.3, 77.2 kg $\pm$ 8.0). The ECG was recorded with a gTec (www.gtec.at) amplifier, sampled at 512 Hz, filtered with a 50Hz notch filter and bandpassed with a 20-50Hz Butterworth filter of 4th order. HR was then extracted in real time using a custom steep slope detection algorithm. All software was implemented in Matlab 2008b (www.mathworks.com). The study protocol was approved by local ethics committees and subjects gave informed consent.

We varied the three Lokomat parameters: treadmill speed, GF and BWS. For experiments in healthy subjects, we implemented a velocity-dependent friction force for investigation of effects of changes in power expenditure. Friction was computed as

$$F_{friction} = \alpha v_{TM}^2 \tag{2}$$

where v_{TM}^2 is the treadmill speed and α is a scaling factor. The friction caused additional power expenditure adding up to the expenditure related to walking. Subjects walked at three walking speeds [1, 2 and 3 km/h], three GFs [0%, 50% and 100%], three different levels of BWS [0%, 30% and 60%] and three different levels of friction (α = 0, 0.5 and 1). Note that a GF of 100 % meant a maximally stiff impedance controller and 0 % a fully transparent orthosis. Maximal gait speed of 3 km/h was chosen, as the Lokomat is limited to 3.2 km/h for patient safety. Details on the experimental protocol can be found in [12].

The dependency between walking and HR of healthy subjects has been previously investigated. Increases in treadmill speed were shown to linearly increase HR [13]–[16]. This can be interpreted as lowpass-like reaction to a sudden increase of oxygen demand, which we modeled as a first order delay (PT) element ($\frac{k}{\tau s+1}$). Treadmill acceleration resulted in an overshoot in HR before steady state was reached [13], [14], [17]. An undershoot was observed after a negative acceleration. Holmgren reported a drop in arterial pressure that reached its minimum 10 seconds after onset of exercise [18]. The HR 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 [19]. The overshoot and undershoot behavior was modeled as a second order derivative (DT) element ($\frac{ks}{(s+\tau)^2}$). The power expenditure of a subject during exercise on a bicycle ergometer [20]–[22] and during arm cranking [21] was reported to correlate linearly with HR. Therefore, 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, which resulted in increased resting HR, was observed and described by several researchers [10], [13], [14]. We modeled this as a first order lowpass element. This resulted in a model with five scaling factors and six parameters.

Estimation of the the scaling factors and parameters for each subject were done using a genetic algorithm in combination with a gradient descent optimization, as we wanted to explore the whole parameter space for solutions. Validation of the model was performed with a velocity profile different from the one used for model identification (Fig. 2). The goodness of fit was assessed with the coefficient of

determination, R^2.

For real-time HR control, we needed to repeat the identification of the subject specific parameters at each training. We performed an online identification of subject specific parameters, optimizing only over the first 12 minutes of the speed profile in (Fig. 2). After the first 12 minutes, the model parameters were fixed and used for HR prediction during training sessions of 37 minutes.

C. Model identification in patients

We performed parameter estimation with eight chronic stroke patients (3w, 5m, 51.3y $\pm$10.7, all hemi-paretic), all with a Functional Ambulation Classification (FAC) score of 0, by looking at steady state increases in HR from baseline HR measured during standing. Investigating GF, treadmill speed and BWS at three different settings as done for healthy subjects would take 27 conditions each several minutes long. As patients can usually exercise around 30 to 45 minutes in the Lokomat, we split up our investigations into two patient groups (experiment I and II). The experimental protocol started with three minutes of standing baseline recording, followed by 5 minutes walking in the Lokomat to get acquainted to the machine. The conditions were presented in randomized order, with a condition length of three minutes, which was in pre-tests found to be a good tradeoff between experimental time and reaching steady state of HR. Ethical approval was obtained by the local ethics committee and all subjects gave informed consent. Although we did not plan on varying BWS during HR control (see Discussion), we still investigated its effects on HR for theoretical purposes.

In a first set of experiments (experiment I), four stroke subjects walked at two walking speeds [1.5 and 2.5 km/h], two levels of GF [50% and 100%] and two levels of unloading to [30% and 60%]. This resulted in eight conditions plus one baseline condition while standing in the Lokomat. Lower settings for walking speed were reported to feel uncomfortable by patients; 0% GF was not investigated, as only one patient was able to walk at less than 30% GF; higher values of BWS were not investigated, as they are not used in a clinical setting. Friction force, as used with healthy subjects for increase of power expenditure, was also not used as it would resist the GF provided by the Lokomat.

We performed a second set of experiments with four different stroke subjects (experiment II), to obtain additional data points for different treadmill speeds and unloadings. Investigation of GF changes were not included as a result of experiment I, as changes in GF did not cause changes in HR (see Results section). Treadmill speed was set to [1, 2 and 3 km/h] and BWS to [30%, 45% and 60%]. Walking speeds higher than 3 km/h were not investigated for patient safety; lower levels of BWS could not be chosen as the ones of healthy subjects, as no patient was able to walk at 3 km/h with less than 30% BWS. Experiment II had nine conditions plus one baseline condition with condition length set to 3 minutes. All results were computed as percent change in HR from baseline standing.

III. MODEL PREDICTIVE CONTROL

A. Problem setup

We used model predictive control (MPC) for closed loop HR control, as the MPC controller can predict HR based on a provided model. These predictions can predict HR constraint violations based on the model, in which the HR would rise over a desired maximal HR, before these situations can actually occur. The MPC can also limit treadmill speed, BWS and GF to be bound not only within the robot's safety limits, but it can also be configured to change the robot's parameters within safe, patient-adapted ranges. Also, the relationship between HR and power output (computed from angular velocity and torque exerted in the robot), can be used as a measured disturbance to improve controller performance.

B. Controller plant model

The Lokomat would allow manipulation of the patient's HR via treadmill speed, GF and BWS (Fig. 1, left), which would make the control problem a "multi input single output" (MISO) problem with HR as output variable. As a result of experiment I, we did not use GF as a control variable, as it did not significantly impact HR (Tab. I). Because of therapeutic reasons, we did also not use body weight as a control variable (see Discussion for details).

Using the model identified in equation 4, We validated that the HR model developed for healthy subject did hold for the patient data recorded in experiment I and II. We split the model up into a plant and a disturbance model and used the power measurement in equation 1 as a measured disturbance.

C. Experimental setup and controller evaluation

Using the model developed in chapter II in combination with the controller developed in III-B, we performed HR control in three healthy subjects. Subjects (2 m. and 1 f., 28.4 y $\pm$ 5.1) walked in the Lokomat at 30% BWS. The desired HR was set to the profile between 70 and 90 beats per minute (bpm), (as depicted in figure 3, A). As changes in treadmill speed between [0, 3] km/h would not have elicited a HR as high as 90 bpm, we turned off GF and enabled friction force as in eq. 2. Mean and standard error (STE) of HR were computed to quantify controller performance. Althought the model incorporated transient

TABLE I

RESULTS OF CHANGES IN GF FOR ALL PATIENTS IN EXPERIMENT I. PERCENT CHANGE IN HR FROM BASELINE, WHERE BASELINE WAS SET TO 100%

		GF [%]	
BWS	v_{TM}	50	100
30	1.5	131 $\pm$ 5	130 $\pm$ 6
30	2.5	117 $\pm$ 3	116 $\pm$ 7
60	1.5	115 $\pm$ 2	119 $\pm$ 3
60	2.5	113 $\pm$ 2	111 $\pm$ 2

BWS: body weight support, v_{TM}: treadmill speed

TABLE II

SUMMARY OF ALL HR CHANGES CAUSED BY BWS AND TREADMILL SPEED FOR ALL PATIENTS IN EXPERIMENT I AND II. PERCENT CHANGE IN HR FROM BASELINE, WHERE BASELINE WAS SET TO 100%.

	BWS [%]		
v_{TM}	30	45	60
1	114 ± 1	106 ± 3	103 ± 1
1.5	131 ± 5	n.a.	116 ± 3
2	108 ± 3	107 ± 1	102 ± 2
2.5	117 ± 2	n.a.	114 ± 2
3	112 ± 3	109 ± 1	108 ± 3

BWS: body weight support, v_{TM}: Treadmill speed

dynamics, we wanted to quantify the controller's ability to perform cardiovascular training, i.e. driving HR to a desired steady state. We therefore computed mean and STE over the last 60 seconds of each condition.

IV. RESULTS

A. Model identification

Neither healthy subjects nor patients showed an increase of HR for changes in GF (Tab. I). We therefore did not include GF in our HR model. Healthy subjects also did not react to changes in BWS, but only for changes in treadmill velocity and friction. In patients, BWS allowed increasing/decreasing HR by up to 31% (Tab. II). However, for therapeutic reasons, we set BWS constant and did not include it in the model (see Discussion for details). Therefore, the model only included the influence of treadmill speed and power output. HR was computed as an increase from baseline

$$HR = HR_{Baseline} + \Delta HR \tag{3}$$

with Δ HR = overshoot dynamics + undershoot dynamics + power expenditure + fatigue as described in section II B. The overall HR model for healthy subjects was parameterized as

$$\begin{aligned}\Delta HR \quad &= \frac{sk_1 v_{TM}}{s^2+\tau_{OS} d_{OS}+\tau_{OS}^2} + \frac{sk_2 v_{TM}}{s^2+\tau_{US} d_{US}+\tau_{US}^2} \\ &+ \frac{k_3 v_{TM}+k_4 P}{\tau_{fast} s+1} + \frac{k_5 P}{\tau_{slow} s+1}\end{aligned} \tag{4}$$

with P being the power exchanged between human and Lokomat, τ_{OS} and τ_{US} the time constant of overshoot (OS) and undershoot (US) respectively, τ_{fast} and τ_{slow} the time constants of the fast and slow dynamics and $k_i, i \in [1,4]$ are the scaling factors.

In the model (eq. 4), four of the five scaling factors were found to be subject-dependent, the other parameters were distributed within $\pm 10\%$ of their respective mean values and were therefore set to the mean [12]. The constant parameters were τ_{slow} = 575.03, τ_{OS} = 0.0575, d_{OS} = 1.0094, k_{US} = 0.1445, d_{US} = 1.0010, τ_{US} = 0.0302.

B. Heart rate responses of healthy subjects and patients

The model for treadmill velocity and power expenditure during robot-assisted gait rehabilitation successfully predicted the HR of healthy subjects with an R^2 of 0.80±0.15 if the parameter optimization was performed over the whole dataset, and with an R^2 of 0.79±0.14 when the parameter optimization was performed on the first 12 minutes of data. The R^2 value of the later case refers only to the predicted data from minute 12 on (Fig. 2). Whenever healthy subjects did not respond to increases of treadmill speed with increases in HR during model identification, we obtained a low quality of HR prediction.

Within the class of responders, we found decreasing HR for decreasing BWS. Contrary to clinical observations informally reported by physiotherapists, we found no change above or below the normal HR fluctuation for changes in GF (Tab. I). Linearizing the system for control purposes, the plant model of eq. 4 simplified to

$$A = \begin{pmatrix} -2\ \tau_{OS} & k_1 & -\tau_{OS}^2 & 0 \\ 1 & & 0 & 0 \\ 0 & & 0 & \frac{-1}{\tau_{fast}} \end{pmatrix} \quad B = \begin{pmatrix} 1 \\ 0 \\ \frac{k_2}{\tau_{fast}} \end{pmatrix}$$

$$C = \begin{pmatrix} k_1 & 0 & 1 \end{pmatrix} \qquad D = \begin{pmatrix} 0 \end{pmatrix}$$

where k_1 and τ_{OS} are the gain and time constant of the overshoot dynamics and k_2 and τ_{fast} are the gain and time constant of the fast dynamics describing the dependency between treadmill speed and HR increase. This system is controllable and observable for $k_i \neq 0$ and $\tau_{OS} \neq 0$.

C. Heart rate control

The results of HR control in the three healthy subjects (see Experimental setup) are summarized in Fig 3. It has to be noted that HR of healthy subjects fluctuated in average

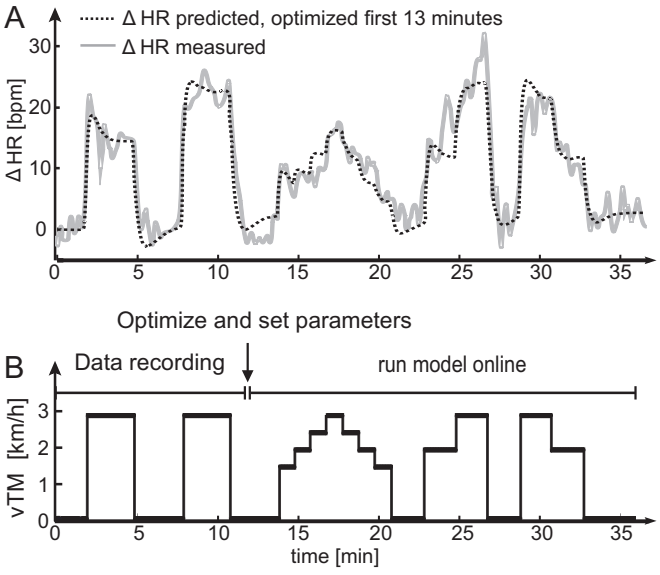

Fig. 2. Predicted and recorded HR of one healthy subject subject (A) and Lokomat treadmill velocity profile (B) for model verification. HR was recorded for the first 12 minutes. From this data, the model parameters were computed and fixed. From minute 12 on, the model predicted HR in real-time.

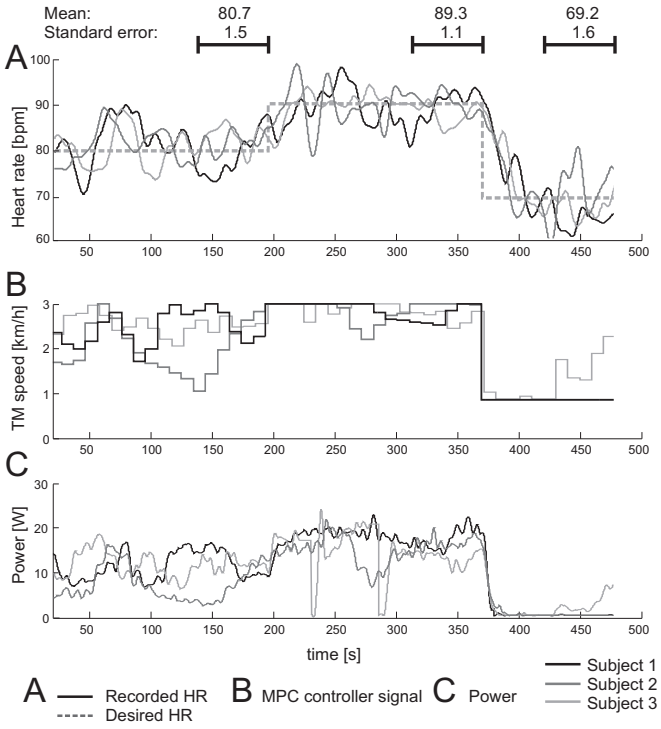

Fig. 3. A: HR of three healthy subjects controlled to a desired reference trajectory. HR is plotted filtered (4^{th} order Butterworth filter, cut-off at 0.8 Hz) to remove short term HR variability and show the underlying trend. The controller results (mean and standard error of the last 60 seconds, i.e. steady state of each condition) are displayed to quantify the controller quality. B: control signal to the treadmill of all three subjects. C: power expenditure of all three subjects.

by $\pm$ 4 bpm during standing and $\pm$ 3 bpm during walking. The controller was able to control HR to the desired values of 80, 90 and 70 respectively within the limits of these HR fluctuation. To mimic the training situation in which patients exercise, we limited the treadmill speed of the Lokomat to 3 km/h. Trying to control the subjects to 90 bpm, the control signal saturated. The maximally reachable HR was limited by the patient's HR at maximal treadmill speed.

V. DISCUSSION

We integrated HR control into robot-assisted treadmill training, as cardiovascular training was shown to be beneficial for stroke patients [1]. To achieve HR control as a function of the exercise parameters power expenditure and treadmill velocity, we developed a model of human HR during robot-assisted treadmill training and applied it for control purposes. A controller which stabilizes HR at a desired value opens the possibility of personalized cardiovascular training in the early phase of gait rehabilitation after stroke.

A. Influence of guidance force and treadmill speed on HR

Decreasing GF and increasing treadmill speed would have been expected to lead to an increase in metabolic cost and therefore to increase HR [21]. Surprisingly, changes in GF did not alter HR in healthy subjects and patients, and HR at treadmill speeds of 1.5 km/h were increased compared to HR at 2.5 km/h in patients (Tab. I). Both results are counter-intuitive.

A possible explanation for the GF results could be that decreased GF did increase metabolic cost, but allowed the subjects a larger step length, as they could deviate from the predefine trajectory. Larger step length could in return have decreased effort and kept the the overall energetic cost of walking constant.

The increase in HR for low gait speeds was informally confirmed by patients, who reported low gait speeds to be more exhausting than faster gait. This might have to do with the effects of leg's vein pumps, which can support the cardiovascular system better at higher gait speeds. Preliminary results of gait speeds up to 4.5 km/h in the Lokomat showed, that HR did increase monotonic between 2 km/h and 4.5 km/h. These higher gait speeds were not available at the time of the experiments. However, this finding will necessarily influence the design of HR control experiments in neurological patients and will require enforcing a lower limit of 2km/h on gait speed.

B. Comparison to other control approaches

HR control has been successfully demonstrated using other control techniques like PID or H_∞ control in healthy subjects using Hammerstein models [10]. However, our approach differs from the approaches above in its usability with severely affected stroke patients. In our model, the power exchange with the Lokomat accounted for up to 75% of the predicted increases in HR. Compared to PID, MPC enabled us in a straightforward way to include the influence of power expenditure in our controller. Additionally, walking speed in Lokomat is currently limited to 3.2 km/h, which severely limits the possibilities of HR control. Previous approaches to HR were performed at walking speeds starting at 3.6 km/h [10], which is already above the maximal possible Lokomat walking speed. From that point of view, the increase in HR of 20 bpm in healthy subjects only presents a hurdle for the current version of the gait robot Lokomat. As studies have found high walking speeds to be most beneficial for patients recovering after stroke [23], future gait rehabilitation robots might allow higher maximal walking speed, which might improve results achieved with our approach.

Controlling HR of healthy subjects to 90 bpm (Fig. 3), volitional pushing effort might have increased power expenditure of subjects and thereby HR even further. However, our approach was explicitly designed for stroke patients with severe impairments that do not have the cognitive or biomechanical ability to perform task oriented, voluntary movements. While HR control via active, voluntary pushing effort might have worked better than MPC, it would require cognitive understanding of the task as well as the biomechanical ability to voluntarily push against the orthosis.

C. Control variables for heart rate control in patients

Although HR control could have been improved by varying not only treadmill speed but also BWS, we decided not to use BWS as a control variable. Increased BWS reduces

the loading the patient has to carry during walking. High loading of the patient during treadmill training was shown to be a key factor for rehabilitation success [24], [25]. In order to maximize the quality of coordinative training, it was decided to set BWS patient-specifically to a fixed minimal value. Minimum BWS was identified for each patient individually by decreasing unloading at maximal walking speed in steps of one kg. Minimum BWS was set right before the gait pattern degraded visibly as rated by the attending physiotherapist. The loading was then kept constant over the whole training session. This approach reduced control variables to the treadmill speed.

Not all patients could be classified as responders to our intervention. It was not possible to clearly identify a clinical indicator that would predict pre-training whether or not the patient would react. Beta blocking medication, which is known to decrease HR variability and limit the adaptation of HR to physical stress [26], was ruled out as a reason. Post hoc analysis of power expenditure could also not explain the observed phenomena. In stroke survivors, medullar brain stem and hemispheric infarctions were shown to impair autonomic cardiovascular regulation [27]. Patients with these diagnoses should be excluded from HR control in future trainings.

D. Heart rate control

HR control was well possible in healthy subjects within the bounds imposed by limiting the treadmill speed to values between [0, 3.2] km/h. From a clinical point of view, the necessity to re-identify each subject anew is currently the largest drawback of our approach, as the identification phase can take up to 12 minutes (Fig. 2, bottom). These 12 minutes would significantly shorten the Lokomat therapy. With four parameters, an automatic learning algorithm might be used to identify the parameters automatically in the beginning.

Although

VI. CONCLUSION AND OUTLOOK

We were able to show that a model-based control approach can take subject specific limitations on treadmill speed as well as individual power expenditure into account. Our approach is limited to subjects who show increased HR for increases in treadmill speed. The next step will be to perform model based HR control in neurological patients.

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