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Sensitivity of vertical jumping performance to changes in muscle stimulation onset times: a simulation study

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Abstract. The effect of muscle stimulation dynamics on the sensitivity of jumping achievement to variations in timing of muscle stimulation onsets was investigated. Vertical squat jumps were simulated using a forward dynamic model of the human musculoskeletal system. The model calculates the motion of body segments corresponding to STIM(t) of six major muscle groups of the lower extremity, where STIM is muscle stimulation level. For each muscle, STIM was allowed to switch "on" only once. The subsequent rise of STIM to its maximum was described using a sigmoidal curve, the dynamics of which was expressed as rise time (RT). For different values of stimulation RT, the optimal set of onset times was determined using dynamic optimization with height reached by the center of mass as performance criterion. Subsequently, 200 jumps were simulated in which the optimal muscle stimulation onset times were perturbed by adding to each a small number taken from a Gaussian-distributed set of pseudo-random numbers. The distribution of heights achieved in these perturbed jumps was used to quantify the sensitivity of jump height to variations in timing of muscle stimulation onsets. It was found that with increasing RT, the sensitivity of jump height to timing of stimulation onset times decreased. To try and understand this finding, a post-hoc analysis was performed on the perturbed jumps. Jump height was most sensitive to errors in the delay between stimulation onset times of proximal muscles and stimulation onset times of plantar flexors. It is explained how errors in this delay cause aberrations in the configuration of the system, which are regenerative and lead to relatively large jump height deficits. With increasing RT, the initial aberrations due to erroneous timing of muscle stimulation are smaller, and the regeneration is less pronounced. Finally, it is speculated that human subjects decrease or increase RT depending on the relative importance of different performance criteria.

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1 Introduction

In vertical jumping, the skeletal system is mechanically analogous to a multi-link inverted pendulum. Also, execution time is so short that neural feedback can play at best a secondary role in adjusting control signals for the on-going movement. From a control point of view, vertical jumping is therefore a highly demanding task. In the search for control strategies, researchers have turned to forward dynamic simulation models of the human musculoskeletal system, using muscle stimulation as independent input (Hatze 1981a; Pandy et al. 1990; Soest et al. 1993). An important conclusion drawn from results of forward simulations of vertical jumping is that intrinsic muscle properties can act as a zero-lag negative feedback loop: aberrations of kinematics cause immediate adjustments of muscle forces, which help to limit the effects of movement perturbations on jump height (Soest and Bobbert 1993). This may be exploited to achieve, with one muscle stimulation pattern, successful performance in jumps starting from different initial positions (Soest et al. 1994). Unfortunately, although jump height of the simulation models is rendered relatively insensitive to kinematic perturbations by the intrinsic muscle properties, it still remains highly sensitive to muscle stimulation onset times: when optimal onset times are perturbed by just a few milliseconds, jump height may drop by several centimeters (Bobbert and Soest 1994).

The sensitivity of jump height to timing of muscle actions in our simulation model, which obviously constitutes a problem in the search for control strategies, seems greater than the sensitivity in human subjects. Figure 1, for instance, presents for five vertical squat jumps from one human subject time histories of the vertical ground reaction force and SREMG (electromyographic activity which was first rectified and subsequently smoothed using a 7-Hz low-pass filter) of gastrocnemius and gluteus maximus, where gluteus maximus is one of the muscles activated first. Although the onset time of stimulation of gastrocnemius varied some 50 ms relative to that of gluteus maximus, the difference between the highest and lowest of these five jumps was only 2 cm. In the simulation model developed

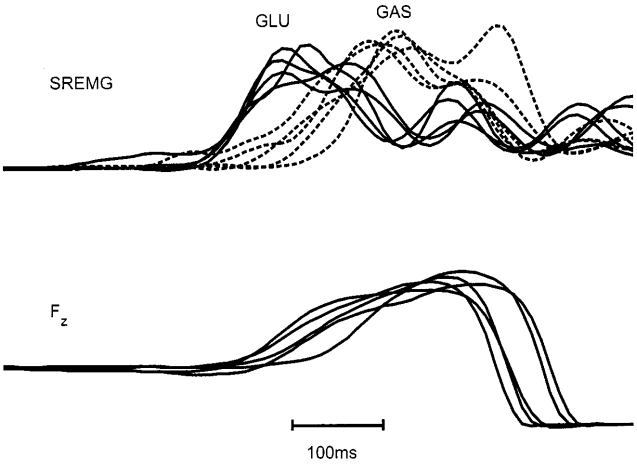

Fig. 1. Time histories of smoothed rectified EMG (*SREMG*) of gastrocnemius (*GAS*) and gluteus maximus (*GLU*) (*top*) and vertical component of the ground reaction force F_z (*bottom*), measured in five maximum height squat jumps of the same subject

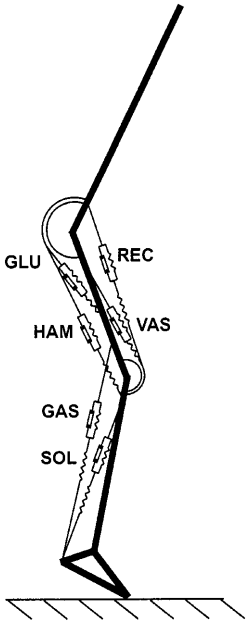

Fig. 2. Schematic drawing of the model of the musculoskeletal system used for forward dynamic simulation of vertical jumps. The model consists of four interconnected rigid segments (feet, lower legs, upper legs and head-arms-trunk) and six muscle groups of the lower extremity (*HAM*strings, *GLU*teus maximus, *REC*tus femoris, *VAS*ti, *GAS*trocnemius and *SOL*eus), all represented by Hill-type muscle models

previously (Soest et al. 1993), a similar timing difference is sufficient to cause a difference in jump height of more than 10 cm, as will become clear in the remainder of this paper. It seems, therefore, that the sensitivity of jump height to variations in timing of muscle actions is greater in the model than in human subjects. This calls for a reconsideration of the differences between the simulation model and the real system.

An obvious difference between our simulation model and the real system exists in the stimulation dynamics, i.e. the dynamics of the development of muscle stimulation. In the simulation model, muscle stimulation is assumed to switch instantaneously between initial and maximal values, as proposed by other investigators (e.g. Levine et al. 1983; Zajac et al. 1984; Pandy and Zajac 1991). However, as illustrated in Fig. 1, in human subjects performing maximum height squat jumps the changes in muscle stimulation occur more gradually (it was confirmed, of course, that this was not due to smoothing). In fact, SREMG rise times (RTs) are typically on the order of 100–200 ms (Bobbert and Zandwijk 1999). It has already been shown that stimulation RTs affect force RTs (Zandwijk 1998; Bobbert and Zandwijk 1999), and the question may be raised whether they also affect the sensitivity of jumping achievement to variations in timing of muscle stimulation onset times. The purpose of the present study was to answer this question for a model of the musculoskeletal system with given properties. Preliminary results of this study have been presented elsewhere (Bobbert and Zandwijk 1996).

2 Methods

The push-off phase of human vertical squat jumps was simulated using the two-dimensional forward dynamic model of the human musculoskeletal system shown in Fig. 2. The model, which calculates the motion of body segments corresponding to muscle stimulation input, has been described in detail elsewhere (Bobbert and Soest 1993). It consists of four rigid segments representing feet, lower legs, upper legs and head-arms-trunk. These segments are interconnected in hinge joints representing hip, knee and ankle joints. In this skeletal submodel, six major muscle-tendon complexes (MTCs) contributing to extension of the lower extremity are embedded: hamstrings, gluteus maximus, rectus femoris, vasti, gastrocnemius and soleus. A Hill-type muscle model is used to represent each of these six MTCs. It consists of a contractile element (CE), a series elastic element (SEE) and a parallel elastic element (PEE), and is also described in full detail elsewhere (Soest and Bobbert 1993; Zandwijk et al. 1996). Behavior of SEE and PEE is determined by a non-linear force-length relationship. Behavior of CE is more complex; contraction velocity depends on active state, CE length, and force, with force being directly related to the length of SEE. This length can be calculated at any instant from the state variables CE lengths and joint angles, because the latter directly determine MTC lengths. Following Hatze (1981b), the relationship between active state, representing the fraction of cross-bridges attached, and muscle stimulation STIM was modeled as a first-order process. STIM, ranging over 0–1, is a one-dimensional representation of the effects of recruitment and firing frequency of α-motoneurons.

In the present study, the rise of STIM(t) to its maximum was described by the following sigmoidal curve (Fig. 3): $\mathrm{STIM}(t) = \mathrm{STIM}_0 + (1.0-\mathrm{STIM}_0) \cdot \sin^2[\alpha(t-t_{\mathrm{onset}})]$ for $0 < (t-t_{\mathrm{onset}}) < (\pi/2\alpha)$, $\mathrm{STIM}(t) = \mathrm{STIM}_0$

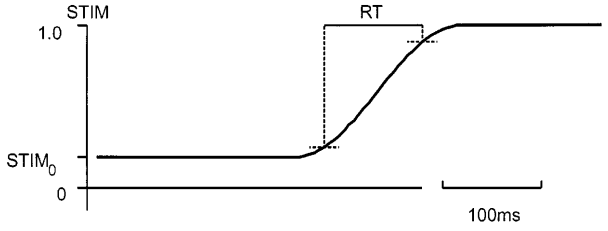

Fig. 3. The rise of muscle stimulation (*STIM*) to its maximum of 1 was described by a sigmoidal curve. The dynamics of this curve are expressed in terms of rise time (*RT*), which is defined as the time taken by STIM to increase from $STIM_0 + 0.1(1.0-STIM_0)$ to $STIM_0 + 0.9(1.0-STIM_0)$, with $STIM_0$ being the STIM level required to maintain equilibrium in the starting position

for $(t-t_{onset}) \leq 0$ and $STIM(t) = 1.0$ for $(t-t_{onset}) \geq (\pi/2\alpha)$, where $STIM_0$ is the initial STIM level required to maintain equilibrium in the starting position, and α and t_{onset} are parameters. For different values of α (same value for all muscles), a set of six values of t_{onset} (one for each muscle) was obtained by dynamic optimization, using as criterion the maximum height attained by the mass center of the system. This yielded the maximum jump height as a function of the speed of stimulation dynamics, with jump height being defined as the height attained by the mass center of the system at the apex of the jump relative to the height of this mass center in upright standing. Subsequently, for each value of α, 200 jumps were simulated in which each of the six t_{onset} values was perturbed by adding a small error Δt_{onset} out of a set of pseudo-random numbers. The set of numbers had a Gaussian distribution with zero mean and standard deviation σ. The distribution of heights achieved in the 200 jumps was used to quantify the sensitivity of jump height to variations in timing of muscle actions. This procedure was repeated for σ values of 1, 3 and 5 ms. In the remainder of this paper, values for α will be converted to RTs, where RT is defined (see Fig. 3) as the time taken by STIM to increase from $STIM_0 + 0.1(1.0-STIM_0)$ to $STIM_0 + 0.9(1.0-STIM_0)$.

3 Results and discussion

3.1 Sensitivity of jump height to control

The distribution of heights obtained in 200 jumps with randomly perturbed t_{onset} was determined for different values of RT and different values of noise standard deviation σ. Figure 4 shows cumulative frequency distribution plots obtained with zero RT and σ values of 1, 3 and 5 ms, and Table 1 presents values for the median value of the jump height distributions. Even for $\sigma = 1$ ms, jump height could be more than 4 cm below maximum. With increasing noise amplitude, the fraction of bad jumps increased and larger jump height errors occurred. Henceforth, results will be presented for a σ value of 5 ms. Figure 5 shows for this σ value cumulative frequency distribution plots of jump heights for three different RTs: 0, 150 and 300 ms. Values for the maximum and median values of the jump height distributions are given in Table 1.

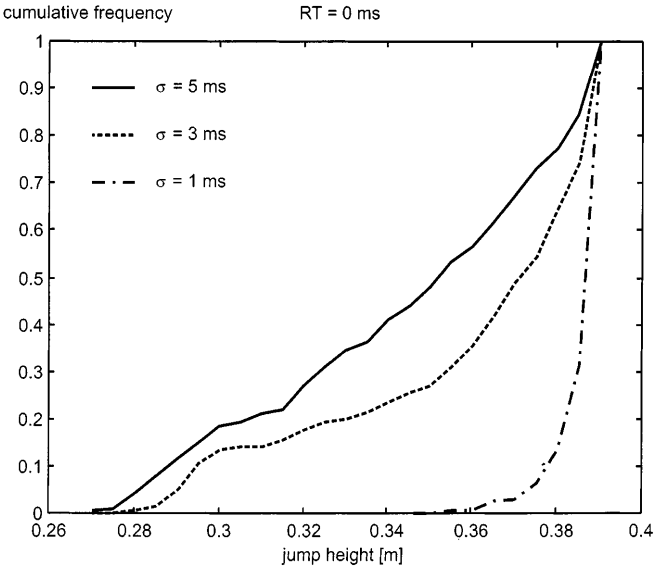

Fig. 4. Cumulative frequency distributions of jump heights obtained in simulated jumps in which the stimulation onset time of each of the six muscles was perturbed by adding a small timing error. This error was drawn randomly out of a set of pseudo-random numbers, which had a Gaussian distribution with zero mean and standard deviation σ. Each curve represents the jump height distribution of 200 jumps

Table 1. Values for median jump height obtained in simulated jumps in which the stimulation onset time of each of the six muscles was perturbed by adding a small timing error. This error was drawn randomly from a set of pseudo-random numbers, which had a Gaussian distribution with zero mean and standard deviation σ. Each value is the median of 200 jumps. Note that $\sigma = 0^{ms}$ yields maximum jump height

σ	Rise time						
	0 ms	50 ms	100 ms	150 ms	200 ms	250 ms	300 ms
0 ms	0.390	0.389	0.386	0.383	0.378	0.371	0.362
1 ms	0.388	0.388	0.385	0.382	0.377	0.371	0.357
3 ms	0.372	0.375	0.377	0.377	0.371	0.366	0.355
5 ms	0.351	0.357	0.362	0.363	0.360	0.357	0.352

The first observation to be made in Fig. 5 and Table 1 is that maximum jump height decreases when RT increases. This may be explained easily if we first consider the optimal solutions in kinematic terms. Figure 6 presents time histories of joint angles and joint angular velocities corresponding to the optimal solutions at RTs of 0 ms and 150 ms. Although the push-off lasts 70 ms longer in the latter case, the differences between the curves occur primarily in the first part of the push-off and, in the case of joint angles, can hardly be distinguished. Virtually the same joint angle trajectories were also found in the optimal solutions for other RTs; regardless of RT, virtually the same body configurations are passed through in the optimal solution. Thus, the reason for the drop in maximum jump height with increasing RT is the following: because of the slower force development, a greater part of the range of joint extension is traveled at submaximal force and, therefore, the total amount of work produced is less. The magnitude of this effect is limited, however, because when the rate of

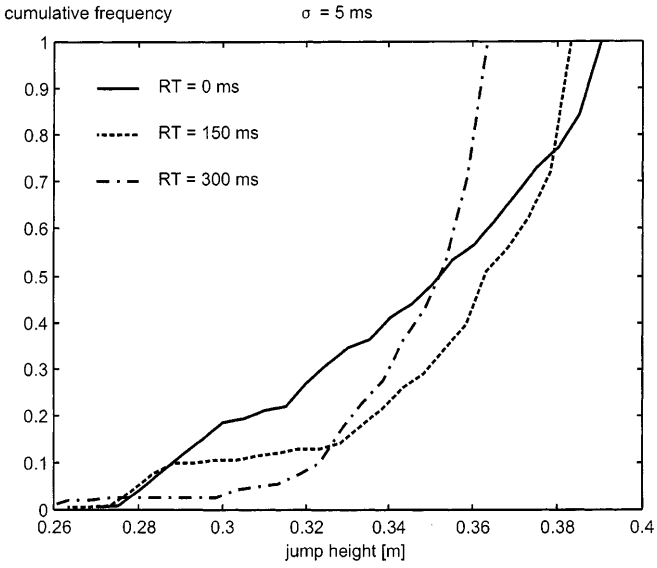

Fig. 5. Cumulative frequency distributions of jump heights obtained in simulated jumps with randomly perturbed stimulation onset times (cf. Fig. 4), for rise times of 0, 150 and 300 ms. In all simulations σ, the standard deviation of noise in stimulation onset times, was 5 ms

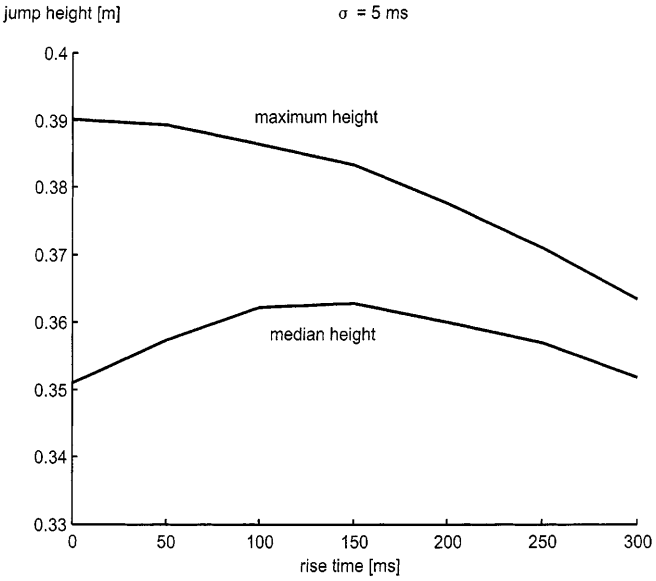

Fig. 7. Values for maximum and median jump height obtained in simulated jumps with randomly perturbed stimulation onset times at different rise times of muscle stimulation. The standard deviation of noise in stimulation onset times (σ) was 5 ms

force development decreases, so does the rate at which accelerations increase. Consequently, more time for force development is available before a given part of the range of joint extension has been covered.

The second observation to be made in Fig. 5 and Table 1 is that the jump height distributions become more narrow as RT is increased. Thus, with increasing RT, the sensitivity of jump height to changes in timing of stimulation onset times decreases. In other words, jumping achievement becomes more robust. Surprisingly, when σ is greater than 1 ms, even in terms of absolute jump height, measures of central tendency are not in favor of very short RTs! For instance, at a σ value of 5 ms, the median of the jump height distribution increases by more than 1 cm when RT is increased from 0 ms to some 150 ms, (Table 1 and the graphical representation of selected results in Fig. 7). A further increase of RT does lead to a further reduction of the sensitivity of jump height to noise in stimulation onset times, but maximum jump height and median jump height both decrease. It may be concluded that if there is noise on stimulation onset times and median jump height is used as criterion, RT has an optimum value of some 150 ms.

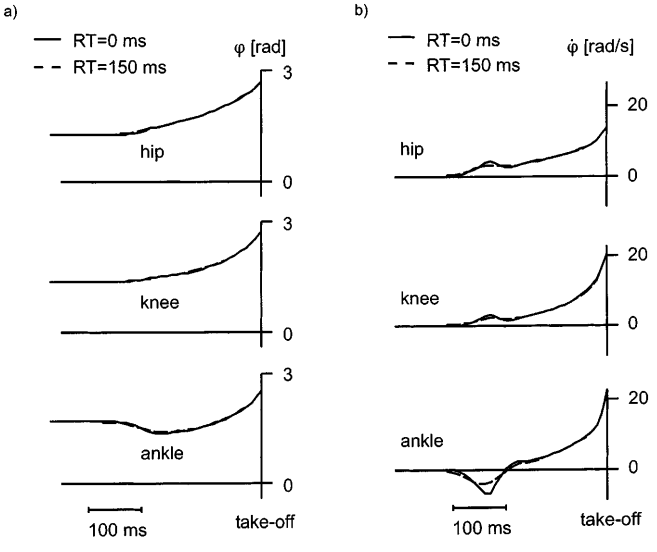

Fig. 6. Time histories of joint angles (**a**) and joint angular velocities (**b**) for maximum height jumps when muscle stimulation RT is 0 ms (*solid lines*) or 150 ms (*dashed lines*)

3.2 Why does sensitivity of jump height to control depend on stimulation RT?

It was found in this study that when stimulation RT is increased, jumping performance of the model becomes more robust. An important question is, of course, whether this phenomenon holds only for jumping because of the instability of the system in this task, or whether it holds for other movements too. To answer this question, we need to understand why the sensitivity of jump height depends on stimulation RT. To gain this understanding, we have first attempted to relate jump height to the errors in timing of muscle stimulation onsets. In a post hoc analysis of jump heights corresponding to randomly perturbed stimulation onset times it appeared that, given a stimulation RT, jump height was strongly dependent on errors in the delay between stimulation onset times of proximal muscles and stimulation onset times of plantar flexors. This is illustrated in Fig. 8, which shows jump height as a function of the errors in this delay, obtained by subtracting for each jump the average error in stimulation onset times of hamstrings, gluteus maximus and vasti,

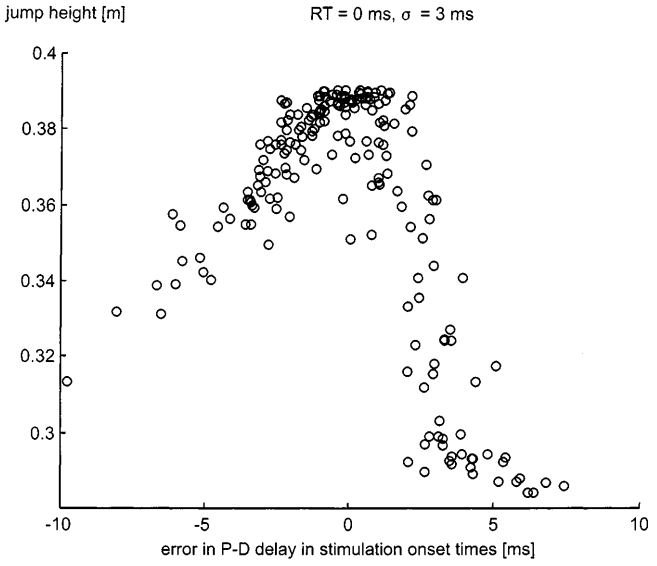

Fig. 8. Jump height plotted as a function of the errors in P-D delay, i.e. the delay between average stimulation onset of proximal muscles and that of plantar flexors. Results were obtained by post hoc analysis of simulated jumps in which stimulation onset times were randomly perturbed by noise with a standard deviation (σ) of 3 ms. Errors in P-D delay were calculated by subtracting for each jump the average error in stimulation onset times of hamstrings, gluteus maximus and vasti, from the average error in stimulation onset times of gastrocnemius and soleus (thus, a positive error means that the plantar flexors were activated too late). The P-D delay in the optimally timed jump amounted to 58 ms

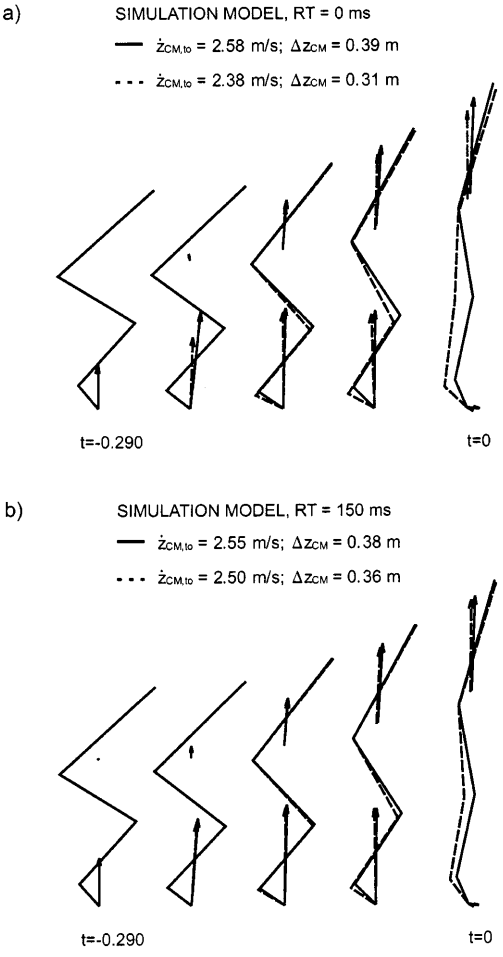

Fig. 9. Stick diagrams for simulated jumps in which stimulation rise times were 0 ms (**a**) or 150 ms (**b**). *Solid lines* are for jumps in which stimulation onset times were optimal (unperturbed), *dashed lines* for jumps in which the stimulation onset time of soleus was delayed by 6 ms relative to its optimal value. All diagrams are equidistant in time. Each stick diagram shows the velocity vector of the center of mass plotted with its origin in the location of the center of mass, as well as the vertical ground reaction force vector plotted with its origin in the center of pressure. $\dot{z}_{CM,to}$ Vertical velocity of the center of mass at take-off; Δz_{CM} jump height, i.e. height of the center of mass at the apex of the jump relative to height of the center of mass in upright standing

from the average error in stimulation onset times of gastrocnemius and soleus. Large errors in jump height occurred especially when stimulation onset of the plantar flexors was erroneously delayed relative to that of the proximal muscles.

The next challenge is to explain why jump height depends so strongly on errors in the delay between stimulation onset times of proximal muscles and those of plantar flexors. The top panel of Fig. 9 shows for a RT of 0 ms stick diagrams of a jump in which stimulation onset times were optimal, as well as for a jump in which stimulation onset of soleus was delayed 6 ms relative to the optimal value. The top panel of Fig. 10 shows for these jumps the vertical component of the ground reaction force as a function of the height of the center of mass. The area under each curve reflects the amount of effective work performed by the muscles, with effective work being defined as work contributing to an increase in energy related to height and vertical velocity of the center of mass. It can be observed that two factors are contributing to the lower effective work production in the erroneously timed jumps. First, at each height of the center of mass, the vertical ground reaction force is lower in the erroneously timed jump than in the optimally timed jump (Fig. 10). Second, take-off occurs at a lower height of the center of mass in the erroneously timed jump (Fig. 10), at a body configuration in which the ankle joint is plantarflexed less than in the optimally timed jump (Fig. 9).

Let us begin with a discussion of the origin of the vertical force deficit in the erroneously timed jump. Since the plantar flexors are activated too late, they are temporarily producing too little force and the plantar flexion moment is too small. As a result, the downward acceleration of the ankle is greater and the upward acceleration of the center of mass lower in the erroneously timed jump than in the optimally timed jump. This leads to higher hip and knee extension velocities and more extension of the knee against the ankle in the erroneously timed jump (note the third stick diagram in Fig. 9). Due to the force-velocity relationship, the higher hip and knee extension velocities lead to lower forces of gluteus maximus and vasti, and thereby lower hip and knee extension moments. One might expect that because of these lower extension moments, the aberration in configuration of the system would be partly corrected as soon as the plantar flexors are activated, but this is not the case. At the instant that the plantar flexors are

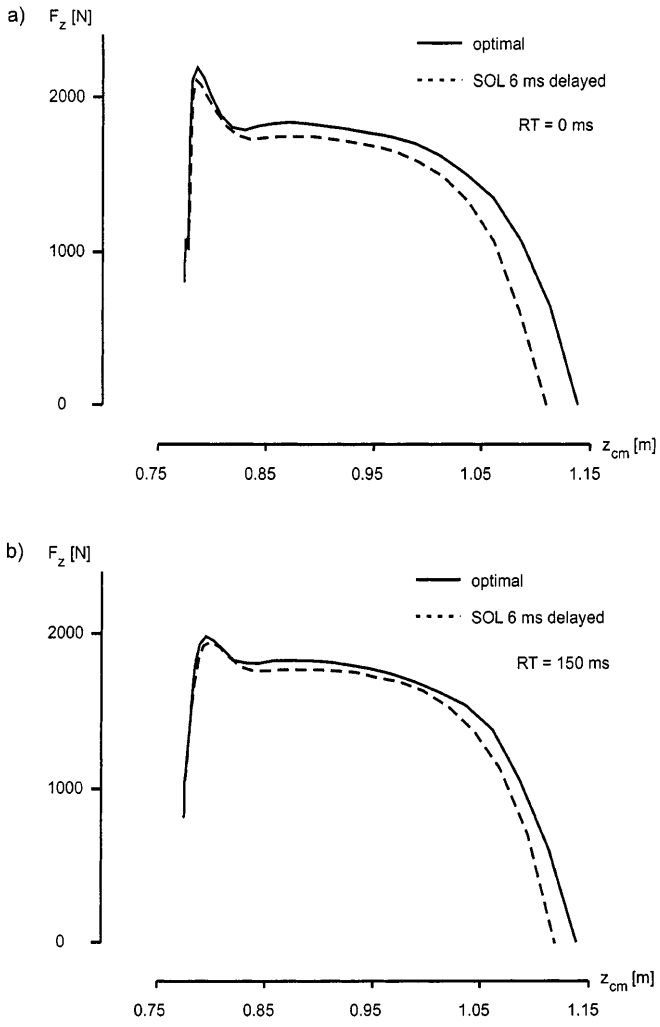

Fig. 10. Vertical component of the ground reaction force (F_z) plotted as a function of the height of the center of mass (z_{CM}) for simulated jumps in which stimulation rise times were 0 ms (**a**) or 150 ms (**b**). *Solid lines* are for jumps in which stimulation onset times were optimal, *dashed lines* for jumps in which the stimulation onset time of soleus was delayed by 6 ms relative to its optimal value

activated, the configuration of the system is different in the erroneously timed jump than in the optimally timed jump, and therefore the effects of intersegmental forces are different (intersegmental forces are caused by gravity and muscular forces, which are transmitted through the system by dynamic coupling). Specifically, in the erroneously timed jump, the segments proximal of the ankle are oriented more vertically, so that the intersegmental forces, which are directed almost vertically, have smaller moment arms; the foot, in contrast, is oriented more horizontally so that the intersegmental forces on the foot segment have a larger moment arm. The latter explains why the vertical ground reaction force can be smaller even though the plantar flexion moment is higher in the erroneously timed jump. The overall result is that, instead of a partial correction, a further increase the aberrations in kinematics of the system occurs; the hip and knee angular velocities and angles increase more and more relative to the values in the optimally timed jump and plantar flexion lags more and more behind (note the last two stick diagrams in Fig. 9).

In both the optimally timed jump and the erroneously timed jump, the increase in angular velocities of the hip and knee joints (see Fig. 6b for the optimally timed jump), and therewith in shortening velocities of the mono-articular extensor muscles, causes a decrease in the hip and knee extension moments. The plantar flexion moment is no longer the weakest link in the chain, and the angular velocity of plantar flexion increases so that a (faster and faster) drop occurs in the plantar flexion moment and the vertical ground reaction force. In the erroneously timed jump, this happens prematurely and too rapidly because the trunk and leg segments are oriented more vertically at the same height of the center of mass and, consequently, the transfer from angular motion of these segments to linear motion of the center of mass is less favorable (Bobbert and Ingen Schenau 1988; Ingen Schenau 1989). At a height of the center of mass of about 1.05 m, reached some 50 ms before take-off, the angular velocities are higher and the joint moments lower at all the joints in the erroneously timed jump compared to the optimally timed jump. Due to the less favorable configuration of the system in the erroneously timed jump, the rate of increase in angular velocities is higher in all joints, but especially in the ankle joint. As a result, the maximum shortening velocity of the plantar flexors, i.e. the velocity where these muscles cannot produce force anymore, is reached at a smaller ankle angle, and take-off occurs prematurely, at a lower height of the center of mass compared to the optimally timed jump. It follows that in the erroneously timed jump, the ranges of motion of the joints are travelled at smaller moments (due to the higher angular velocities) so that less work is produced. Moreover, compared to the optimally timed jump, a needlessly large amount of work is transformed into rotational energy of the segments at take-off rather than effective work (see also Bobbert and Ingen Schenau 1988).

In essence, the course of events described above is a case of positive feedback: a small delay in onset of muscle stimulation causes a small delay in force and a small kinematic aberration, but the aberration is regenerative because of the effect of the errors in the configuration of the system on the transfer from angular to linear variables and vice versa. It seems plausible that such a regenerative loop may be initiated by certain errors in timing of muscle actions in all explosive movements (i.e. movements in which the force-velocity relationship of the muscles has a strong limiting effect on performance) involving two or more joints in series. However, performance of explosive movements in which the musculoskeletal chain behaves like an inverted pendulum are likely to suffer more from timing errors because of the de-stabilizing effect of gravity.

The final challenge is to explain why the severity of the problem described above depends on RT. Figures 9b and 10b present, for 150 ms of stimulation RT, curves similar to those which were presented in Figs. 9a and 10a for 0-ms RT. The figures clearly show that, in the case of 150-ms RT, the difference between the errone-

ously timed jump and the optimally timed jump is similar but less pronounced than in the case of 0-ms RT. Apparently, a smaller force difference acting over a longer time interval, as in the case of 150-ms RT, has less effect than a large force difference acting over a short time interval, as in the case of 0-ms RT, even if the integral of the force difference with respect to time is, in principle, the same. This can be understood intuitively because we have just learned that not only the force impulse but also its transmission through the system, which depends on the configuration of the system, is important. In the case of 0-ms RT, the force error develops rapidly in a given configuration of the system, whereas in the case of 150-ms RT, the force error develops slowly, while the configuration of the system is changing. In the latter case, the erroneous moments and the resulting initial kinematic aberrations, which trigger the positive feedback loop, are smaller, and the regeneration is less pronounced.

3.3 Other factors which may influence the sensitivity of jump height to control

It was shown in this study that when stimulation RTs are increased, jumping achievement becomes less sensitive to errors in muscle stimulation onset times. Even with relatively large RTs of 300 ms, however, the sensitivity remains rather high; certain errors in stimulation onset times of less than 10 ms, such as the late onset of plantar flexors, still lead to errors in jump height of more than 10 cm. Which factors other than stimulation RT may affect the sensitivity of jump height to errors in muscle stimulation onset times? One factor is the force-velocity relationship of the muscles. Zandwijk et al. (1998) recently found in experiments on five human subjects that the force of their plantar flexors decreased faster with velocity than the force of the muscles in the model. When the experimentally derived force-velocity properties are substituted in the model, maximum jump height decreases but the system becomes less sensitive to errors in muscle stimulation onset times (Zandwijk 1998). A second factor, prompted by the results of our analysis of what goes wrong in erroneously timed jumps, may be the pliability of the foot. In the model, the foot is a rigid body with a point support when the heel is off the ground. In the real system, however, the foot has several joints actuated by muscles, which allows for a larger contact area and variation in the location of the center of pressure, even when the heel is off the ground. Perhaps these factors provide an additional explanation for the apparent robustness of jumping performance in human subjects.

3.4 Do human subjects manipulate stimulation RT?

In a previous study (Bobbert and Zandwijk 1999) SREMG RTs were found to vary from 50 ms to more than 200 ms among subjects. The question was raised whether this variation has any functional significance. In the present study it was found that if there is noise on stimulation onset times and median jump height is used as criterion, it is better to slow down RT to some 150 ms. In a quantitative sense, it seems unwarranted to translate the stimulation RTs in the model to stimulation RTs in human subjects. It is not known, for instance, whether muscle active state achieved in maximum voluntary contractions of human subjects is comparable to that reached in the model at a stimulation of 1.0. If stimulation in the model is not increased to 1.0 but to 0.5, the active state will no longer saturate, the force response will slow down, and the sensitivity of performance to stimulation onset times will decrease. Nevertheless, in a qualitative sense we may speculate that subjects decrease or increase RT depending on the relative importance of different performance criteria. At a given level of noise in stimulation onset times, long RTs lead to robustness of performance at the expense of jump height. Short RTs lead to short execution times, which may be important in interceptive tasks requiring fast reactions, but at the expense of robustness of performance. Possibly, noise can be reduced by training so that robustness increases at the same RT, or that a shorter RT, leading to shorter execution time, can be afforded at the same level of robustness. Perhaps the variability in SREMG RTs among subjects reflects differences among the subjects in sports background or training level.

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