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RESEARCH ARTICLE

Control of Movement

Interactions between initial posture and task-level goal explain experimental variability in postural responses to perturbations of standing balance

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Abstract

Postural responses to similar perturbations of standing balance vary widely within and across subjects. Here, we identified two sources of variability and their interactions by combining experimental observations with computational modeling: differences in posture at perturbation onset across trials and differences in task-level goals across subjects. We first collected postural responses to unpredictable backward support-surface translations during standing in 10 young adults. We found that maximal trunk lean in postural responses to backward translations were highly variable both within subjects (mean of ranges = 28.3°) and across subjects (range of means = 39.9°). Initial center of mass (COM) position was correlated with maximal trunk lean during the response, but this relation was subject specific (R^2 = 0.29–0.82). We then used predictive simulations to assess causal relations and interactions with task-level goal. Our simulations showed that initial posture explains the experimentally observed intrasubject variability with a more anterior initial COM position increasing the use of the hip strategy. Differences in task-level goal explain observed intersubject variability with prioritizing effort minimization leading to ankle strategies and prioritizing stability leading to hip strategies. Interactions between initial posture and task-level goal explain observed differences in intrasubject variability across subjects. Our findings suggest that variability in initial posture due to increased sway as observed in older adults might increase the occurrence of less stable postural responses to perturbations. Insight in factors causing movement variability will advance our ability to study the origin of differences between groups and conditions.

NEW & NOTEWORTHY Responses to perturbations of standing balance vary both within and between individuals. By combining experimental observations with computational modeling, we identified causes of observed kinematic variability in healthy young adults. First, we found that trial-by-trial differences in posture at perturbation onset explain most of the kinematic variability observed within subjects. Second, we found that differences in prioritizing effort versus stability explained differences in the postural response as well as differences in trial-by-trial variability across subjects.

initial posture; postural control; predictive simulation; task-level goal

INTRODUCTION

Redundancy in the neuromotor system enables kinematic variability (1, 2). However, the factors governing differences in how motor tasks are performed across different instances within the same individual (intrasubject variability) as well as systematic differences across individuals (intersubject variability) are not well understood (3). In reactive balance control, often studied through observation of postural responses to perturbations of standing balance (4), kinematic strategies in response to the same external perturbation differ in the amount of trunk lean and the occurrence of steps both within and across subjects. In addition, intrasubject variability varies across individuals. Intrasubject variability in step incidence following a perturbation of standing has been associated with center of pressure (COP) position at perturbation onset (initial posture) (5), but it is yet unclear whether initial posture also explains intrasubject variability in nonstepping responses. Intersubject variability of postural strategies can be caused by both musculoskeletal

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(6–8) and neural factors with differences in task-level goal contributing largely to intersubject variability in healthy young adults (9, 10). However, no prior studies investigated interactions between sources of intrasubject and intersubject variability, and hence, it is unclear what causes differences in intrasubject variability. Here, we developed a framework for understanding both the isolated and interaction effects of initial posture and task-level goal as sources of intrasubject and intersubject variability in balance-correcting responses to perturbations of standing in a group of young healthy adults. Since experiments alone do not allow us to establish causal relations or to analyze factors that are not directly measurable, such as the task-level goal, we complemented experiments with model-based simulations.

Intrasubject and intersubject variability in postural responses to backward support surface translations during standing result in different kinematic strategies, including stepping and nonstepping balance strategies. Even with the feet remaining in place, there is a continuum of responses between two strategies commonly referred to as the ankle strategy and the hip strategy (4). In the ankle strategy, the body sways about the ankles, relying on ankle torque shifting the COP within the base of support (BOS) to counteract the perturbation (11, 12). COP displacement can therefore be used to quantify the contribution of the ankle strategy to a postural response. As task difficulty increases, for example, when the perturbation magnitude increases or the base of support decreases, the ankle strategy is typically combined with a hip strategy characterized by a combination of ankle plantarflexion, knee flexion, and, most prominently, hip flexion (4, 13, 14). Hip flexion causes trunk rotation, generating a change in angular momentum that counteracts the perturbation. The extent to which the hip strategy contributes to a postural response has been quantified by different variables: the change in horizontal component of the ground reaction force (15), the change in angular momentum (11), or the maximal trunk lean angle (10).

Reactive balance responses consist of three temporally distinct phases, but it is yet unclear when variability originates and how it propagates over the different phases. During the preperturbation phase, both voluntary and random movements affect posture at perturbation onset (initial posture) (16). We will refer to the phase lasting from perturbation onset to 300 ms into the response as the early response (ER) (17). In the first 80 to 100 ms after perturbation onset, alterations in muscle activity are typically absent (18, 19). Intrinsic joint impedance and skeletal inertia, which might depend on initial posture, determine the response during this time. The first alterations in muscle activity are the result of short (onset 100–120 ms) (20, 21) and long latency reflexes (onset 120–180 ms) (19, 22–24) originating from spinal and supraspinal feedback loops. Movements caused by these early muscle activities may only be observable up to 100–200 ms after their onset (25). Movement observed within the first 300 ms after perturbation onset may thus mainly result from musculoskeletal mechanics and automatic control. From 300 ms onward, movements and the muscle activation patterns that induce them become more complex, as voluntary components may accompany reflexes (26–29). We will refer to this phase as the later response (LR).

Posture at perturbation onset contributes to intrasubject variability in step incidence, but it is unclear to which extent changes in initial posture also explain intrasubject variability in nonstepping postural responses. Changes in initial posture result from postural sway (30), i.e., the natural oscillation of the hip, knee, and ankle angles during quiet standing or anticipation to an expected perturbation (16). Anticipatory postural adjustments, changes in muscle co-contraction (31), and changes in reactive control (24) lead to less frequent stepping responses. In this study, we focused on the effect of changes in initial posture resulting from postural sway by applying unpredictable perturbations to prevent anticipation. A prior study showed that altered initial postures can reduce step incidence when the COP is displaced in the same direction as the platform translation (5). It has not been assessed whether trial-by-trial changes in initial posture also explain intrasubject variability in nonstepping strategies.

During the early and the later response, differences in task-level goal rather than musculoskeletal properties might contribute to intersubject variability in young healthy adults. The task-level goal defines a variable (e.g., COM kinematics, trunk orientation, stability, metabolic efficiency) that appears to be controlled or optimized when performing a specific task but that is not directly encoded by any unique sensory input or motor output (22). Prior studies showed that musculoskeletal properties might play a role in corrective balance responses: increased body mass and inertia alter the dynamics of the body, increasing the resistance to a perturbation (7), and measures of strength allow discriminating fallers from nonfallers (32). Some studies have shown that increased strength and rate of force development might improve balance recovery (6, 8), but other studies have not confirmed these results [e.g., (33)]. These studies contrast groups with large differences in musculoskeletal properties and, therefore, do not explain intersubject variability across young healthy adults with rather homogenous musculoskeletal properties. Differences in task-level goals, resulting from personal preference, prior movement training (34), expectation of the disturbance (24), and emotional state [e.g., fear of falling (35–37)], might explain intersubject variability across individuals with similar musculoskeletal properties.

Computer simulations are a useful tool to investigate cause-effect relations in human movement. There are typically multiple strategies to perform a movement task. Optimal control simulations enable us to identify movement strategies that satisfy different task-level goals (10, 38–41). Such optimal control simulations capture important features of movement kinematics across movement tasks [reaching (42–45), standing balance (10, 38–41, 46), gait (47–49)]. Simulations of standing balance are often based on simple mechanical models that do not account for muscle physiology. Despite being simplified representations of the complex neuromusculoskeletal system, sagittal multilink models controlled through feedback-generated torques have yielded useful insights in postural control (39, 40). Using optimal control simulations, we recently tested the effects of varying task-level goals on movement strategies during reactive balance (9). Variations in the trade-off between minimizing effort and COM excursion resulted in a continuum of ankle and hip strategies that was in agreement with the experimentally

observed range of strategies in a group of healthy young adults.

Although origins of both intrasubject and intersubject variability have been identified, it is yet unclear why intrasubject movement variability differs across subjects. Both experimental and simulation studies using models with different complexities strongly suggest that initial posture contributes to intrasubject variability (8, 50, 51), and task-level goal contributes to intersubject variability in postural strategies (9, 10, 52), but no prior studies assessed the interactions between these two origins of variability.

Therefore, our goal was to dissociate the effects of initial posture (COM position at perturbation onset) and task-level goal (prioritization of stability versus effort) on intrasubject and intersubject variability in the kinematic strategy in response to backward support surface translations. We quantified the kinematic strategy by the maximal trunk lean angle during the response and combined experiments and optimal control simulations to test the following hypotheses:

1) Posture at perturbation onset contributes to intrasubject variability in kinematic strategies, with a more anterior initial COM position increasing the use of the hip strategy (higher maximal trunk lean angle) and step incidence.

2) Differences in task-level goal contribute to intersubject variability in kinematic strategies, with prioritizing effort minimization leading to ankle strategies and prioritizing stability leading to hip strategies.

3) The interaction between initial posture and task-level goal contributes to observed differences in intrasubject kinematic variability, with prioritization of stability increasing intrasubject variability.

We performed perturbation experiments and established correlations between initial posture and postural strategy in response to unpredictable backward support-surface translations. We then used predictive simulations to demonstrate the causal relation between initial posture and movement variability and its interaction with task-level goal. We performed predictive simulations of the postural responses to the different perturbations assuming optimal feedback control. We separately (*hypotheses 1* and *2*) and simultaneously (*hypothesis 3*) varied initial posture and the objective function reflecting the task-level goal to test the hypothesized effects of initial posture and task-level goal on the kinematic strategy. We compared simulated and experimentally observed effects of initial posture. We performed all simulations using a generic model, assuming that musculoskeletal differences had no major contribution to movement variability in this group of young healthy adults.

MATERIALS AND METHODS

Experiments

Participants and experimental protocol.

All experimental protocols were approved by the ethical committee of UZ Leuven (Protocol No. S61361). Ten healthy young adults (Table 1) provided written informed consent before participating in this study, which was part of a larger study aiming at understanding differences in reactive balance between healthy young and older adults.

Table 1. *Participant age, height, mass and gender information*

	Age, yr	Height, cm	Mass, kg	Gender
Mean	24	177	68	7 female
Range	22–28	163–198	52–95	3 male

Participants stood barefoot on a movable platform (CAREN, Motek) with their feet at shoulder width looking forward and wore a safety harness to catch them in case of a loss of balance. Participants were instructed to maintain balance without taking a step when perturbed and were allowed to move their arms freely. If the perturbation elicited a stepping response, participants were instructed to return themselves to their original position before the subsequent perturbation. To standardize foot placement, the heel position was marked on the platform.

Participants received a total of six support-surface perturbation types: anterior-posterior translations, mediolateral translations, and pitch rotations in two directions inducing either ankle plantar- or dorsiflexion. Subjects were first familiarized with the motion of the platform while being informed on the type of perturbation. During this familiarization, perturbations were applied with progressively larger magnitudes until subjects needed to take a step, which ended familiarization with the specific perturbation direction. The first perturbation magnitude that induced a step response was the highest magnitude included in the randomized experimental part of the protocol. When no step response was evoked at the highest magnitude, all perturbations for that direction were included. Up to six different perturbation magnitudes (see Fig. 1*A* for position, velocity, and acceleration profiles) were presented for posterior translations, whereas up to four different perturbation magnitudes were presented in the other directions. Next, each perturbation condition was applied five times in random order. Our analysis focused on backward platform translations because the kinematic variability was largest in response to backward perturbations.

Subjects were instrumented with 33 reflective markers on anatomical landmarks (full body plug-in-gait) and four clusters on the left and right shanks and thigh. Three markers were placed on the platform to acquire its motion. The marker trajectories were captured using seven Vicon cameras at a frequency of 100 Hz. Subjects stood with each foot on a different AMTI force plate embedded in the platform, measuring forces and moments at 1,000 Hz. A static trial in anatomical position was acquired before starting the perturbation experiment.

Electromyography (EMG) (Cometa) was collected at 1,000 Hz from seven muscles per leg: medial and lateral gastrocnemius, soleus, peroneus longus, vastus medialis, vastus lateralis, and rectus femoris.

Data analysis.

Collected data for the posterior platform translations were preprocessed to get joint, platform, COM, and COP kinematics. All marker trajectories were labeled in Vicon Nexus 2.4. Generic musculoskeletal models (OpenSim 3.3) were scaled based on anatomic marker positions acquired during the

static trial (53). Joint angles were computed using OpenSim's Inverse Kinematics tool (OpenSim 3.3). Marker tracking root-mean-square errors were smaller than 2 cm, and maximal errors were smaller than 4 cm. Finally, OpenSim's Body Kinematics tool was used to compute segment and whole body kinematics. The platform positions and velocities were derived from the trajectories of the three markers attached to the platform. Center of pressure (COP) locations were derived from the forces and moments recorded by the force plates. The COP trajectories were expressed in the world frame by taking the platform offset and motion into account. A correction of the force plate data was performed to remove forces and moments registered due to the inertia of the force plate (54). We corrected for these forces and moments by subtracting the forces and moments registered during all perturbations when no mass was on the platform from the data acquired with the subject on the platform.

EMG signals were band-pass filtered using a fourth-order Butterworth filter with frequencies of 20 Hz and 400 Hz. Next, the signals were rectified and low-pass filtered using a fourth-order Butterworth filter with a cutoff frequency of 12 Hz. Each EMG channel was normalized with respect to the maximal value measured throughout the whole experiment.

The anterior functional base of support (BOS) was approximated as the horizontal distance from the origin of the talus segment (ankle joint) to the origin of the toe segment in the scaled model during the static trial. The posterior BOS was determined as the horizontal distance from the heel to the origin of the talus segment. Perturbation onset was determined using a 2 cm/s threshold on the platform velocity. We determined perturbation onset based on platform velocity rather than on control signal onset since there was a large delay between control signal onset and the platform reaching a 2 cm/s velocity that depended on perturbation magnitude. Step responses were determined by detecting time intervals of more than 100 ms where the vertical ground reaction force (GRF) was smaller than 10 N at either force plate.

We used different measures to quantify postural strategies (Table 2). Use of the ankle strategy was quantified by the position of the COP within the BOS (COP/BOS). Reliance on the hip strategy was quantified based on maximal trunk lean angle ($\theta_{trunk,max}$). To quantify the trial-by-trial differences in the position and velocity of the COM, we computed the ratio of the extrapolated center of mass (xCOM) and the BOS (xCOM/BOS) at both perturbation onset ($xCOM/BOS_{onset}$) and at the transition from the early to the later response phase ($xCOM/BOS_{300ms}$). We divide the xCOM by the BOS to normalize between subjects. The extrapolated center of mass xCOM was calculated following the description in Ref. (55) based on the inverse kinematics results [OpenSim 3.3 BodyKinematics (53)]:

$$xCOM = COM_x + \frac{\dot{COM}_x}{\sqrt{g/l}},$$

with COM_x and $\dot{COM}_x$ the horizontal position and velocity of the whole body COM (with respect to the ankle joint to account for platform motion), g the gravity constant, and l the vertical position of the center of mass during the static trial.

To analyze when different muscles contributed to the postural responses, we quantified the muscle onset latencies and mean muscle activity during the ER (Table 2). Muscle onset was determined as the time instant at which the signal amplitude was 3 standard deviations above the mean signal during the 1-s interval before perturbation onset. We visually confirmed whether the algorithm determined a correct onset timing. If timing was clearly off due to some artifact, we removed the trial from the analysis rather than manually selecting an onset time.

Table 2. *Outcome variables of perturbation experiments*

Variable	Quantifies
$\theta_{trunk,max}$	Postural strategy—larger trunk lean indicates greater reliance on hip strategy (10)
$xCOM/BOS_{onset}$	Stability at perturbation onset
$xCOM/BOS_{300ms}$	Stability at transition from ER to LR
COP	Reliance on ankle strategy (12)
EMG onset latency	Latency of muscle activity with respect to perturbation onset
Mean EMG during ER	Mean muscle activity during the first 300 ms (ER) of the postural response

BOS, base of support; COP, center of pressure; ER, early response; LR, later response; xCOM, extrapolated center of mass; $\theta_{trunk,max}$, maximal trunk lean angle.

Statistical analysis.

We performed a nonparametric repeated-measures ANOVA (Friedman's test) to examine the effect of perturbation magnitude on postural strategies (within-subject mean of $\theta_{trunk,max}$ across trials) and within-subject variability in postural strategies (within-subject range of $\theta_{trunk,max}$ across trials). To examine the directional effect of perturbation magnitude, a Tukey's HSD (honestly significant difference), correcting for multiplicity, was executed on the results of the ANOVA test with $\alpha \leq 0.05$.

As learning effects might have contributed to intrasubject variability in kinematic strategy, we evaluated whether we could detect learning effects. We used a Wilcoxon signed-rank test to test whether the maximal trunk lean angle was different between the first and second and between the first and last repetition for perturbations magnitudes 1 to 5. We left out perturbations at magnitude 6, as these perturbations were not included for all subjects.

We compared onset times and activations of the muscles from which we collected EMG by conducting a one-way ANOVA (Fig. 6). To evaluate individual differences in onset times between different muscles, a Tukey's HSD (honestly significant difference), correcting for multiplicity, was executed on the results of the ANOVA test with $\alpha < 0.05$.

We associated initial posture and stability at the end of the early response by performing a robust linear regression analysis [iterative least-squares; robustfit MATLAB (56)] between $xCOM/BOS_{onset}$ and $xCOM/BOS_{300ms}$, with perturbation magnitude as a categorical variable affecting the intercept but not the slope.

To analyze whether stability at the end of the early response and the use of the hip strategy were related, we performed a robust linear regression analysis [iterative least-squares; robustfit MATLAB (56)] between $xCOM/BOS_{300ms}$ and $\theta_{trunk,max}$ for each subject. A one-way ANOVA was

performed on the estimated slope coefficients to test whether we could reject the null hypothesis that the slopes were similar for all subjects ($\alpha = 0.05$). We used this approach as opposed to fitting one model with subject as a categorical variable to all the data because the robust regression method tended to consider all data from some subjects as an outlier, which defeated our purpose of studying intersubject variability.

Predictive Simulations

To perform predictive simulations, we assumed that the response to perturbations could be modeled by optimal feedback control of a multisegment inverted pendulum model describing skeletal mechanics. We performed predictive simulations for different initial postures, task-level goals (encoded by different cost functions), and perturbation magnitudes to assess causal relations between these factors and the kinematic strategy in response to the perturbation.

Model.

Skeletal mechanics and feedback control were modeled differently for the three phases of the response (Fig. 5, *B* and *E*) based on the experimentally observed joint motions and assumptions on underlying control mechanisms. The *1*) initial mechanical response (0–60 ms), the *2*) early response (60–300 ms), and the *3*) later response (>300 ms) were simulated separately while imposing continuity between phases. During the initial mechanical and early responses, the body was modeled by a torque-driven single inverted pendulum as we observed very little knee and hip motion during the first 300 ms after perturbation onset. During the later response, the body was modeled by a torque-driven triple inverted pendulum describing ankle, knee, and hip movement. Joint torques consisted of a passive and an active component. The passive component was modeled by linear damping with coefficient of 0.5 Nm · s/rad. The active component only contributed during the early and later responses. Active torques resulted from delayed linear feedback of the horizontal COM position and velocity. Feedback and electromechanical delays were lumped and modeled by a first-order delay with a time constant of 60 ms (57). Maximal torques ($\boldsymbol{T}_{max}$) for ankle, knee, and hip joints were set at 130 Nm, 200 Nm and 200 Nm, respectively. Due to the different number of degrees of freedom of the models used to describe the skeletal system during the early and the later response, the number of feedback gains differed between phases ($n \times 2$ matrix $\boldsymbol{K}^{n\times 2}$ with the number of degrees of freedom $n = 1$ for the early response and $n = 3$ for the later response).

States, controls, dynamics.

The state of the model at time t is described by the vector s containing joint positions $\boldsymbol{q}(t)$, velocities $\dot{\boldsymbol{q}}(t)$, and activations $\boldsymbol{a}(t)$. For the early response phase, the state vector is:

$$\boldsymbol{s}_{ER}(t) = \left[q_{ankle}(t)\ \ \dot{q}_{ankle}(t)\ \ a_{ankle}(t)\right]^T,$$

For the later response phase, the state vector is:

$$\boldsymbol{s}_{LR}(t) = \left[q_{ankle}(t)\ q_{knee}(t)\ q_{hip}(t)\ \dot{q}_{ankle}(t)\ \dot{q}_{knee}(t)\ \dot{q}_{hip}(t)\ a_{ankle}(t)\ a_{knee}(t)\ a_{hip}(t)\right]^T.$$

The ankle position, velocity, and activation at the beginning of the later phase are constrained to be equal to those at the end of the early response, while the other states are zero at the beginning of the later phase.

During the early response phase, there is a single control:

$$\boldsymbol{e}_{ER}(t) = [e_{ankle}(t)] \in [-1, 1],$$

whereas there are three controls for the later response phase:

$$\boldsymbol{e}_{LR}(t) = \left[e_{ankle}(t)\ \ e_{knee}(t)\ \ e_{hip}(t)\right] \in [-1, 1].$$

These controls are determined by linear feedback from the horizontal COM position and velocity:

$$\boldsymbol{e}_{phase}(t) = \boldsymbol{K}^{n\times 2}\begin{bmatrix} COM_x(t) - COM_x(0) \\ \dot{COM}_x(t) - \dot{COM}_x(0) \end{bmatrix},$$

with phase referring to ER or LR. The controls are subject to a lumped neuromechanical delay that is modeled as first-order dynamics with a time constant of 60 ms:

$$\dot{\boldsymbol{a}} = \frac{\boldsymbol{e} - \boldsymbol{a}}{0.06}.$$

The joint activations are scaled to joint torques $T(t)$ by the maximal torque values T_{max}. The equations of motion were derived based on the Euler–Lagrange equations:

$$\boldsymbol{M}\left(\boldsymbol{q}(t), \dot{\boldsymbol{q}}(t)\right) \cdot \ddot{\boldsymbol{q}}(t) - \boldsymbol{G}\left(\boldsymbol{q}(t)\right) - \boldsymbol{C}\left(\boldsymbol{q}(t), \dot{\boldsymbol{q}}(t)\right) - \boldsymbol{T}(t) - \ddot{p}(t) \cdot \boldsymbol{P}\left(\boldsymbol{q}(t)\right) = 0,$$

with $\boldsymbol{M}$ the mass matrix, $\boldsymbol{G}$ the gravity vector, $\boldsymbol{C}$ the Coriolis vector, $\boldsymbol{T}$ the joint torques, $\ddot{p}(t)$ the platform acceleration, and $\boldsymbol{P}$ the vector mapping the platform acceleration into equivalent external joint torques.

The movement of this model is completely determined by the initial state, the feedback gains, and the perturbation. The initial activations were determined by solving for static equilibrium for the imposed initial posture, and the feedback gains were estimated by minimizing the multiobjective cost function described in the following paragraph.

Cost function.

Feedback gains were computed by minimizing a multiobjective cost function (9, 10, 40, 46) consisting of the following three terms:

$$\phi_{stability} = \int_{t_{start}}^{t_{end}} \left(COM_x(t) - COM_x(0)\right)^2 dt$$

$$\phi_{effort} = \int_{t_{start}}^{t_{end}} \left(e_{ankle}(t)^2 + e_{knee}(t)^2 + e_{hip}(t)^2\right) dt$$

$$\phi_{regularization} = \int_{t_{start}}^{t_{end}} \left(\ddot{q}_{ankle}(t)^2 + \ddot{q}_{knee}(t)^2 + \ddot{q}_{hip}(t)^2\right) dt + \sum_{ij}(K_{ij}^{1\times 2})^2 + \sum_{ij}(K_{ij}^{3\times 2})^2$$

$\phi_{stability}$ was included to minimize the excursion of the COM with respect to the initial position of the COM. Although we do not have contributions of separate muscles, ϕ_{effort} is a term often used to represent effort minimization in simulations of walking (47, 49, 58) and standing (9, 40, 41). $\phi_{regularization}$ is included to improve the numerical condition of the optimal control problem (OCP).

During the first 60 ms, there was no active torque, so the movement was fully determined by the initial state. Feedback gains during the early response (60–300 ms) were computed by minimizing

$$J_{ER} = \phi_{stability} + w_{regularization} \cdot \phi_{regularization},$$

whereas feedback gains during the later phase were computed by minimizing

$$J_{LR} = (1-w)\cdot \phi_{stability} + w\cdot \phi_{effort} + w_{regularization}\cdot \phi_{regularization}.$$

For all simulations, $w_{regularization}$ was small ($1e^{-8}$). We varied w from 0.01 to 0.999 to simulate different task-level goals.

Task constraints.

The predictive simulation was further constrained to model the balance task. The final joint positions at time 1.8 s were constrained to be equal to the initial positions. For simulating the response for different initial postures, we chose the initial ankle angle so that COM_x of the inverted pendulum model ranged from +2.05 to +6.67 cm (similar to experimental observation). The COP was constrained to stay within the BOS, i.e., between −5 cm and +17 cm with respect to horizontal position of the ankle, throughout the motion.

We used a direct collocation approach to transform the OCP into a nonlinear programming problem (NLP). We formulated the problems in MATLAB (The MathWorks Inc.) using CasADi (59). For all simulations, we used a third-order Radau quadrature collocation scheme with a mesh frequency of 100 Hz. We solved the resulting NLP with the solver IPOPT (60).

Data analysis.

We analyzed the same variables from the optimal simulated motions as from the experimentally collected data (Table 2). This allowed us to compare our simulations with the observed behavior and thus to evaluate which causal relations underlie the experimentally established relations.

RESULTS

Postural Strategies in Response to Backward Support-Surface Translations Vary between and within Subjects

We observed large intrasubject and intersubject variability in the postural strategies in response to backward support-surface translations. The range of perturbation levels (Fig. 1*A*) was large enough to elicit a wide range of maximum trunk lean angles ($\theta_{trunk,max}$) (Fig. 1*B*). Seven of the 10 subjects also took steps at larger perturbation magnitudes; the three subjects who did not step were able to maintain feet-in-place responses for the largest perturbation that we could deliver with the platform. Within perturbation, intrasubject variability quantified by the range of maximal trunk lean is large. For the largest perturbation performed by all subjects (magnitude 5), the within-subject range of $\theta_{trunk,max}$ averaged over the 10 subjects was 28.3° (SD = 15.2°) (Fig. 1*B*). Intersubject variability was large as well. For perturbation magnitude 5, the standard deviation and range of the subject mean $\theta_{trunk,max}$ (calculated over the different trials for each subject) were 12.5° and 39.9°, respectively.

Perturbation magnitude had a significant effect on the mean maximal trunk lean angle ($P < 0.0001$) and on the range of the maximal trunk lean angle ($P < 0.001$). As perturbation magnitude increased, the maximal trunk lean angle (Fig. 2*A*) as well as the range of the maximal trunk lean angle (Fig. 2*B*) increased ($P < 0.05$; Tukey's HSD post hoc test). We excluded perturbation magnitude 6 from our statistical analysis, as not all participants executed the task for this magnitude.

Within each subject, the COM position at perturbation onset was variable and associated with stepping responses (Fig. 3). The average range of initial COM positions was 5.02 cm (SD = 1.44 cm) over all trials. Almost without exception, the perturbations that led to stepping responses were large perturbations that started with more anterior initial COM (Fig. 3, *crosses*).

We could not detect learning effects described as changes in the maximal trunk lean angle throughout repeated perturbations ($P > 0.1$ for all Wilcoxon signed-rank tests).

Two Distinct Phases of the Postural Response: Ankle Strategy during Early Response, Combined Strategy during Later Response

During the early response (<300 ms after perturbation onset), subjects used an ankle strategy characterized by a large shift in COP and little trunk movement, whereas during the later response (>300 ms after perturbation onset),

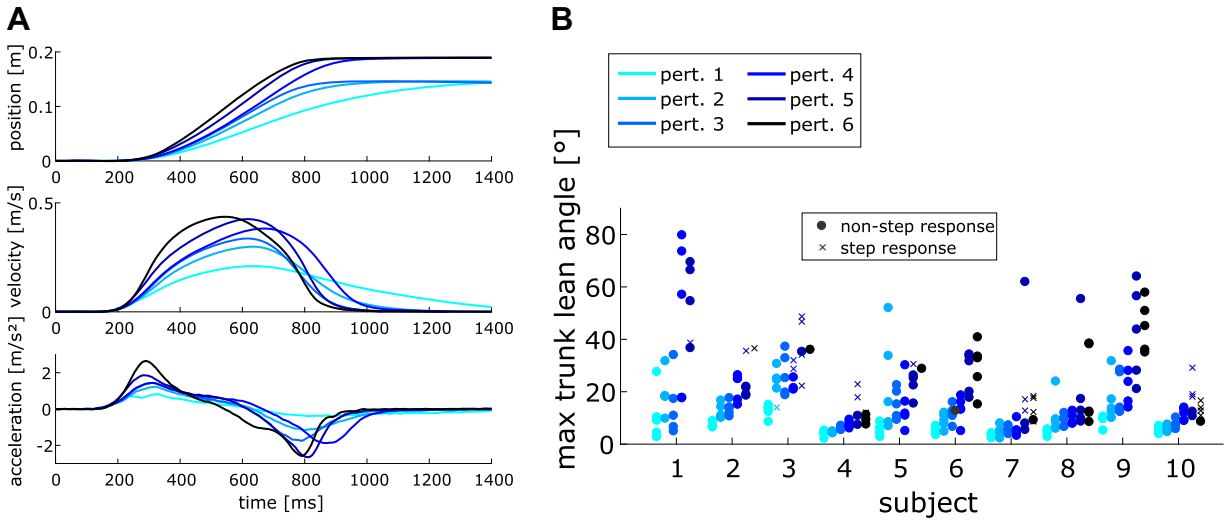

Figure 1. *A*: position, velocity, and acceleration profiles for the six backward support-surface translations. *B*: maximal trunk lean angles for the 10 subjects. Not all perturbations magnitudes were applied to all subjects.

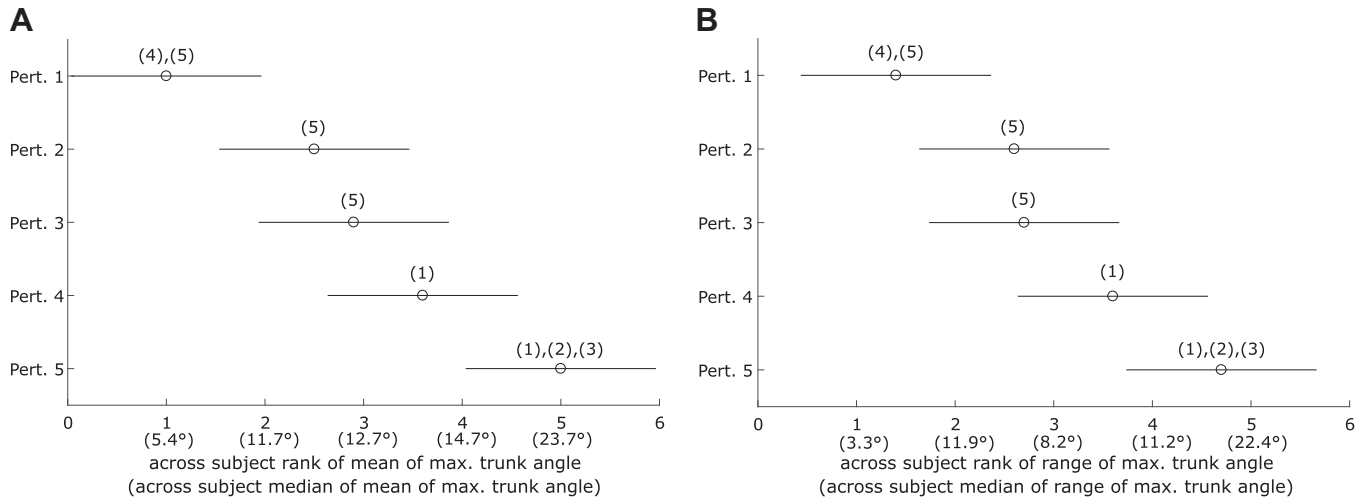

Figure 2. Effect of perturbation magnitude on across subject mean of maximal trunk angle (*A*) and across subject range of maximal trunk angle (*B*). Means with comparison intervals (P = 0.05) are shown for perturbation magnitudes 1 to 5. Numbers in brackets "()" indicate statistical difference with respect to the specified perturbation magnitude.

subjects used a combination of an ankle and hip strategy characterized by larger maximal trunk lean (Fig. 4).

During the early response (ER), the COP moved forward and trunk lean was relatively constant for all perturbation magnitudes. The trunk lean at 300 ms with respect to the maximal trunk lean, $\tilde{\theta}_{trunk,ER}$, was small, whereas the change in COP at 300 ms relative to the BOS, $\Delta\widetilde{COP}_{ER}$, was large across all perturbation magnitudes (Fig. 4*B*). Given the limited motion of the trunk, the extrapolated COM (xCOM) is a reliable measure of (dynamic) posture (55), and the proposed inverted pendulum model is sufficiently complex to describe the kinematic strategy during the early response.

In contrast, during the later response (LR), the COP remained relatively constant and the trunk lean increased (Fig. 4, *A* and *B*), demonstrating the suitability of the maximal trunk lean angle as an outcome parameter to describe the kinematic strategy during the later response.

Early Response: Initial Posture Explains Intrasubject Variability While Intersubject Variability Is Low

During the early response, initial posture is correlated with the kinematic response. xCOM/BOS at perturbation onset and after 300 ms are positively correlated (Fig. 5*A*), with R^2 values ranging from 0.53 to 0.72; all model coefficients were significant ($P < 0.001$). A single correlation explained most of the variability across all individuals and trials, indicating that there is little intersubject variability during the early response. This can qualitatively be confirmed by observing the low variability in $\widetilde{COP}$ and θ_{trunk} during the early response (ER) in Fig. 4*A*. The offset of the linear relation increased with perturbation magnitudes, which corresponds to higher perturbation magnitudes causing a larger response.

The plantarflexors but not hip flexors contributed to active control during the early response. The onset of muscle activity (measured through EMG) happens very consistently for the ankle plantarflexors between 100 and 150 ms after perturbation onset (Fig. 6*A*). At the end of the early response, hip flexor muscles are consistently activated. The mean normalized EMG was significantly higher in the ankle plantarflexors than in the hip flexors during the first 300 ms (Fig. 6*B*).

Predictive simulations of the early response based on a generic inverted pendulum model confirmed that variability in initial posture could cause experimentally observed trial-by-trial intrasubject variability during the early response (Fig. 5, *A*–*C*). The simulated effect of varying initial COM position was qualitatively and quantitatively similar to the experimental results. Increased perturbation magnitude and more anterior initial COM positions (higher xCOM/BOS at onset) resulted in larger xCOM/BOS at 300 ms (Fig. 5*C*). Similar to the experimental data, the offset of the simulated relation between xCOM/BOS at onset and xCOM/BOS at 300 ms increased with perturbation magnitude, and the relations for perturbation magnitudes 3 and 4 were very similar due to the almost identical platform acceleration during the first 300 ms of these two perturbation magnitudes (Fig. 1*A*).

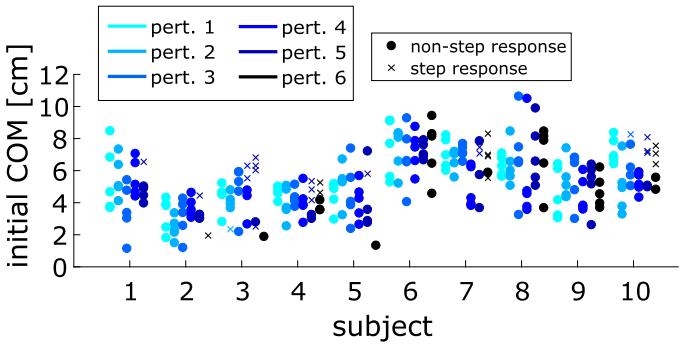

Figure 3. Initial center of mass (COM) positions for different subjects and different trials. Dots represent nonstepping responses; crosses are stepping responses.

Later Response: Initial Posture Explains Intrasubject Variability While Different Task-Level Goals Explain Intersubject Variability

Posture at the onset of the later response is correlated with the maximal trunk lean angle within subjects but not between

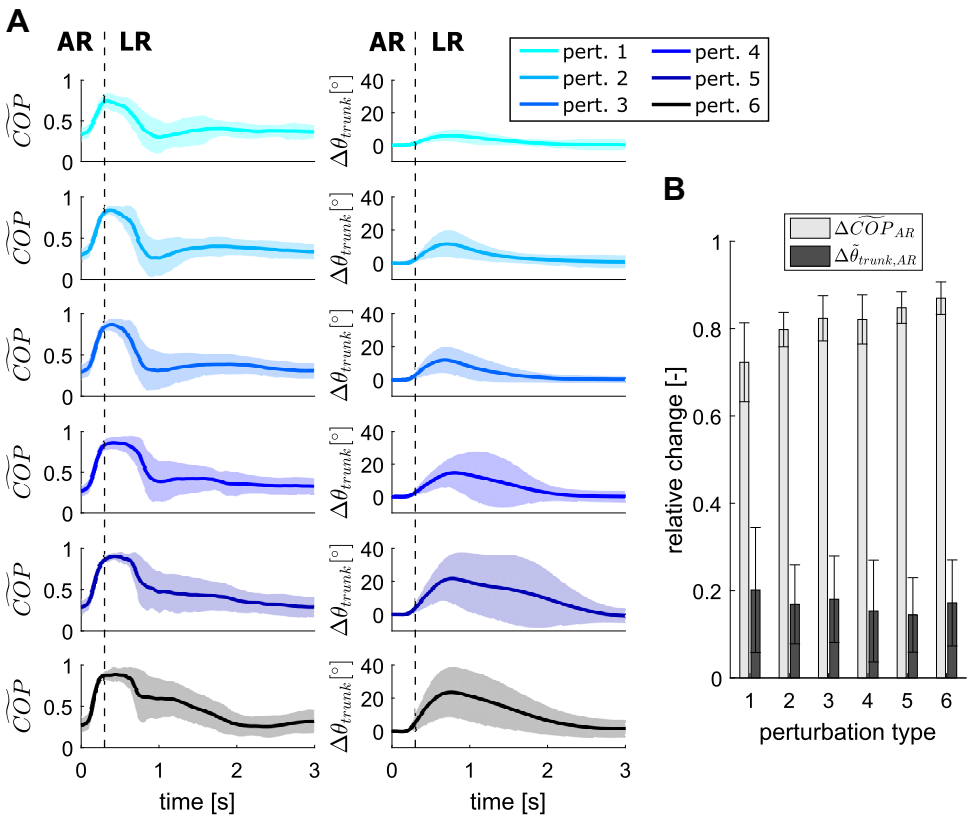

Figure 4. *A*: center of pressure (COP) and trunk lean during the response to a backward support-surface translation. Trajectories for the COP normalized to BOS ($\widetilde{COP}$) and trunk lean with respect to perturbation onset ($\Delta\theta_{trunk}$). We show average trajectories with standard deviation (shaded area) for all subjects and trials. The vertical dashed line indicates the transition from early response (AR) to later response (LR). *B*: relative change in COP position and trunk lean during the early response. Mean and standard deviation of trunk lean at 300 ms relative to the maximal trunk lean over the entire trial ($\Delta\tilde{\theta}_{trunk,AR}$) and change in COP at 300 ms relative to the BOS ($\Delta\widetilde{COP}_{AR}$).

subjects. Within subjects, the correlation between the xCOM/BOS at the end of the early response (300 ms after perturbation onset) and the maximal trunk lean is high (R^2 values from 0.29 to 0.81) across perturbation magnitudes (Fig. 5*B*). All slope coefficients were significant ($P < 0.01$). At least one slope coefficient was significantly different from all other slope coefficients, indicating a subject-specific relation.

Predictive simulations of the later response based on a triple inverted pendulum model showed that variability in initial posture combined with between-subject differences in the task-level goal can cause the experimentally observed kinematic variability. We changed the task-level goal by sweeping the weight determining the trade-off between the effort and stability term in the optimality criterion and assessed the same range of initial postures as used in the simulations of the early response (Fig. 5, *D*–*F*). We performed simulations for the different perturbations that were imposed experimentally. For clarity, we only show simulation results for three perturbation magnitudes (levels 1, 2, and 6) that cover the full range of kinematic responses based on the maximal trunk lean.

Differences in task-level goal caused differences in simulated kinematic strategies that were similar to the observed intersubject variability. We found that prioritizing stability over effort (Fig. 5*F*, compare between colors) as well as increasing perturbation magnitude (Fig. 5*F*, compare between symbols) led to higher maximal trunk lean angles, and hence favored the use of a hip strategy. The simulations that strongly prioritized effort minimization ($w \approx 0$) exhibit a sudden strong increase in the maximal trunk lean for large perturbation magnitudes and large xCOM/BOS at 300 ms (Fig. 5*F*, red simulations). This steep increase in trunk lean is not in agreement with experimental data, which is due to the use of a model that cannot describe a stepping response, hence in simulation, the only remaining option to restore an upright posture is a sudden switch to a hip strategy.

Differences in initial posture caused differences in simulated kinematic strategies that were similar to the observed intrasubject variability. Higher xCOM/BOS at 300 ms led to larger maximal trunk lean for the same task-level goal (Fig. 5*F*, compare within color). The effect of initial posture (xCOM/BOS at 300 ms) on maximal trunk lean angle is larger for increasing perturbation magnitude in agreement with the observed increase in variability in trunk lean angle with perturbation magnitude (Fig. 2*A*). The relation between initial posture and maximal trunk lean angle is steeper when prioritizing stability over effort (Fig. 5*F*). This is in agreement with the experimental observation that the variability in maximal trunk lean angle is larger in subjects who make more use of the hip strategy (Fig. 1*B*). Finally, the relation between initial posture and maximal trunk lean angle is steeper with increasing perturbation magnitude (Fig. 5*F*), which is in agreement with the increasing average range of maximal trunk lean angles with increasing perturbation magnitude (Fig. 2*B*).

DISCUSSION

By combining experimental observations with computational modeling, we demonstrated that intertrial differences in initial posture and intersubject differences in task-level goals can cause observed movement variability in response

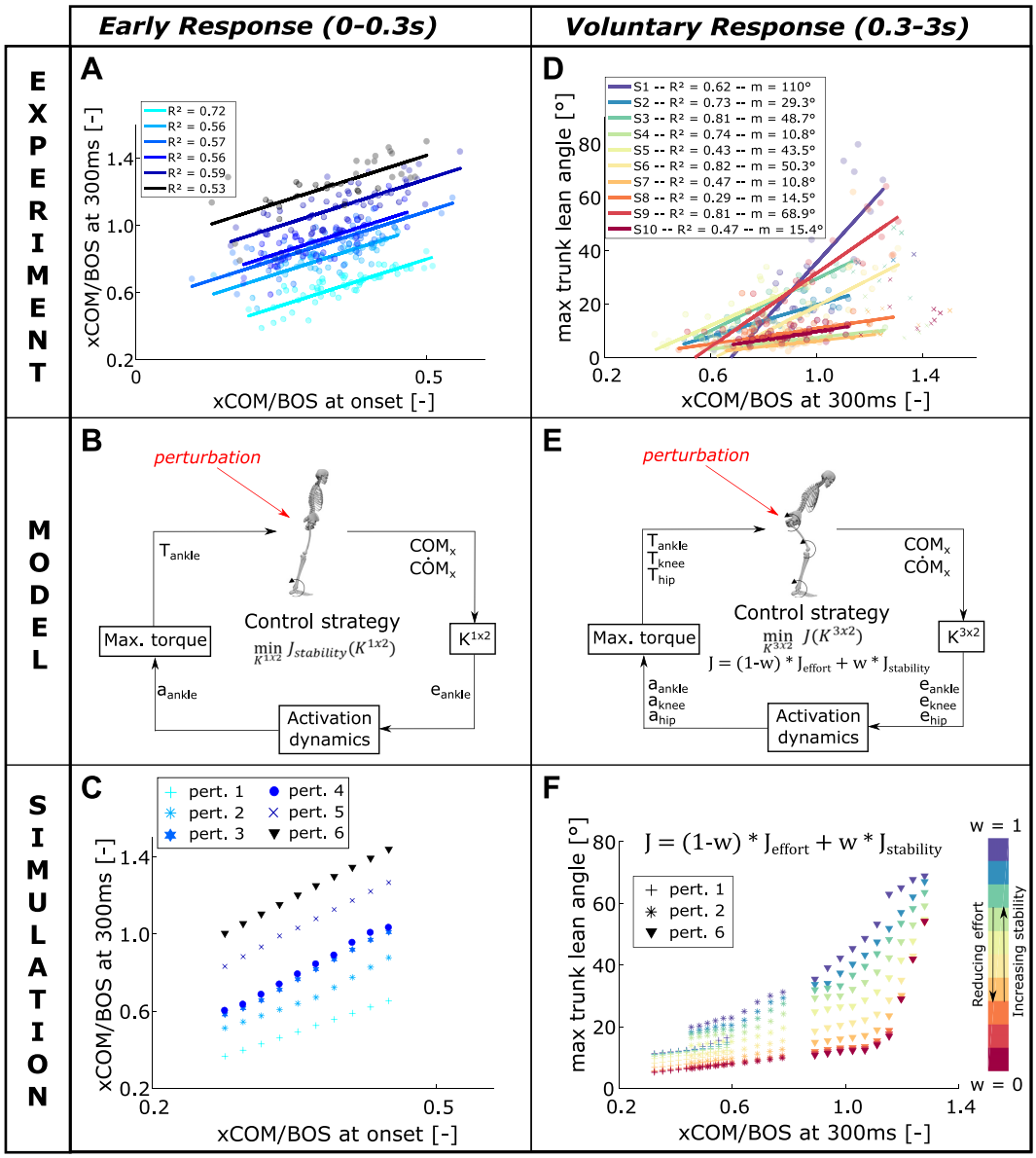

Figure 5. Experiment—*A*: experimental data for all subjects and trials of xCOM/BOS at perturbation onset and at the end of the early response. Colors encode for the different perturbation types. All linear regressions were statistically significant ($P < 0.001$); R^2 values are given. *D*: experimental data for all subjects and trials of xCOM/BOS at the end of the early response and maximal trunk lean angle. Colors encode for the different subjects; dots encode nonstepping responses, and crosses encode step responses. All linear regressions were statistically significant ($P < 0.01$); R^2 values are given; m is the slope of the regression that is subject-specific. Model—Neuromechanical models used for predictive simulations of the postural response in two phases. *B*: torque (T)-driven single inverted pendulum model, during the early response. Feedback (gains $K^{1\times2}$) of the COM kinematics (position: COM and velocity: $\dot{COM}$) generates an excitation signal ("e") that is delayed through activation dynamics to an activation signal ("a"). *E*: a triple inverted pendulum is used to model motion around ankle, knee, and hip joint during the later response. Feedback (gains $K^{3\times2}$) from the COM kinematics drives the model. Simulation—Results from predictive simulations for different trade-offs of effort vs. COM excursion minimization, different initial conditions, and different perturbation magnitudes (perturbation magnitudes 1, 2, and 6). *C*: predictive simulations of the early response. Increased xCOM/BOS at onset results in increased xCOM/BOS at 300 ms. *F*: predictive simulations of the later response. Both xCOM/BOS at onset and trade-off of task-level goal affect the simulated response. The colors encode for the different trade-offs in the cost function. BOS, base of support; COM, center of mass; xCOM, extrapolated center of mass.

to backward support-surface translations during standing in healthy young adults. By using a generic musculoskeletal model, we demonstrated that initial posture, task-level goal, and their interactions can explain movement variability independent of subject-specific variations in musculoskeletal properties. The observed relationship between initial posture and movement variability was similar between subjects in the early response but differed substantially across subjects in the later response. This intersubject variability in the later reactive balance response could be explained in simulations by the interaction between initial posture and intersubject differences in task-level goal, i.e., in the trade-off between stability and effort when reacting to perturbations. Our findings have two important implications. First, small differences in initial

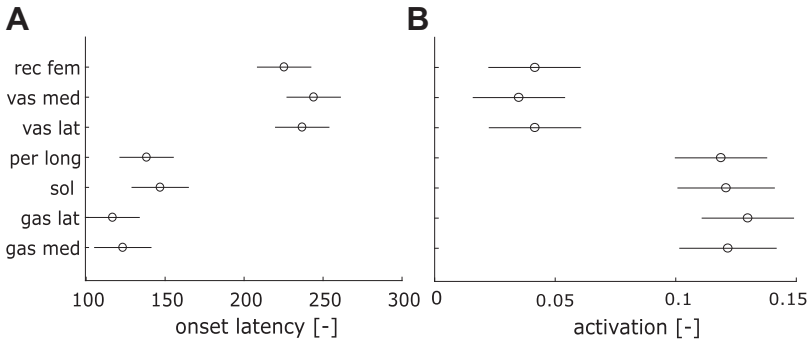

Figure 6. *A*: latency of muscle activity with respect to perturbation onset of ankle plantarflexors and hip flexors. *B*: mean muscle activation in first 300 ms after perturbation onset of ankle plantarflexors and hip flexors. Mean and comparison intervals of the muscle responses averaged per subject over the performed trials of all perturbation types. Muscle latency and mean activation are different ($\alpha = 0.01$) for ankle and hip actuators, resulting in disjoint comparison intervals. gas lat, lateral gastrocnemius; gas med, medial gastrocnemius; per long, peroneus longus; rec fem, rectus femoris; sol, soleus; vas lat, vastus lateralis; vas med, vastus medialis.

posture might increase the occurrence of kinematic strategies that are typically associated with larger perturbations, i.e., hip and stepping strategies, and might limit postural robustness in older adults with increased postural sway and pathological populations that adopt aberrant postures. Second, accounting for the contribution of variability in the execution of balance strategies is necessary to isolate the contributions of various neural and musculoskeletal factors contributing to balance deficits in older adults and populations suffering from balance disorders.

Random variations in initial posture lead to large variability in the kinematic response to perturbations by reducing the effectiveness of the ankle strategy in restoring balance. Previous research has shown that stepping responses are more frequent if the COP location is shifted in the same direction as the effect of the perturbation (5) but did not examine the effects on nonstepping responses as we did here. Consistent with the decreased ability to shift the COP adequately using an ankle strategy when the initial COP is shifted forward in the BOS (12), we observed increased occurrence of hip and stepping strategies with more anterior initial COM. Because we used a randomized perturbation protocol, the observed changes in initial posture could be considered the result of random processes including natural postural sway. Our data further show that the range of initial COM positions during the reactive balance task (5.02 cm on average) exceeded COP sway amplitudes during a 30-s quiet standing task (average peak-to-peak amplitude of 1.3 cm) in the same subjects on the same day. This discrepancy between a standing task and a reactive balance task might have multiple causes. First, the instruction to stand as still as possible during the quiet standing task may have resulted in sway amplitudes below those observed in daily life. Second, the quiet standing task lasted only 30 s (vs. 20 min for the perturbation experiment) and might therefore have failed to catch the larger but slower rambling component of postural sway (61). Third, subjects may have adopted a different resting position after each perturbation (61, 62). Finally, subjects might have explored different initial postures throughout the randomized experiment in search of a posture that is optimal to withstand the perturbations.

Our results suggest that beyond musculoskeletal or sensory acuity declines, increased postural sway may play a significant role in the increased incidence of hip and stepping strategies in older adults. One study found that increased preperturbation sway in the frontal plane improved reactive balance responses to perturbations in the sagittal plane by improving proprioceptive input (63). Therefore, it is possible that sagittal plane sway might also improve proprioceptive input. However, our results suggest that sagittal sway has dominant mechanical consequences leading to increased kinematic variability. Previous studies attributed the increased use of hip and stepping strategies to reduced strength (8, 64) or sensory acuity (38). However, anterior-posterior sway amplitudes in older adults (>65; 25 ± 4 mm) are ~50% greater than in young adults (18 ± 5 mm) (65), leading to increased variability in initial posture. Hence, older adults will more often require hip or stepping strategies when confronted with a balance perturbation, which might increase fall risk (66–68).

The causal relation between initial posture and response implies that systematic differences in standing postural configuration in individuals with neurological or musculoskeletal impairment compared with healthy controls might contribute to balance problems. For example, individuals with Parkinson's disease (PD) typically adopt a stooped posture, moving the COM anteriorly; this has been suggested to be a protective mechanism against backward loss of balance, reducing stabilizing long-latency responses (69). Our simulations show that such anterior shifts in initial posture would also reduce the effectiveness of ankle strategy in forward COM perturbations. Similarly, children with cerebral palsy (CP) often adopt crouched postures (70, 71) that may also contribute to abnormal postural control. Indeed, when typically developing children adopt a crouched posture, their muscle activation responses to a perturbation become similar to those of CP children (72). Predictive simulations, as applied in this study, can be a useful tool to dissociate the effects of aberrant postures and neural control deficits, which often occur simultaneously in pathological populations.

Intersubject variability in postural responses to perturbations only appears during the later response and can be attributed to intersubject differences in task goal that were absent during the early response. Simulations based on a single task-level goal, i.e., maximize stability, explain the observed kinematics during the early response across subjects, initial postures, and perturbation magnitudes. The lack of variability in postural control during the early response might be a consequence of our protocol consisting of unpredictable multidirectional perturbations, as postural

control during the early response is known to be adapted to predictable experimental conditions. For example, adaptation leading to reduced COM movement during the ER has been observed within a series of identical perturbations (24). Given that our simulations already optimize stability by minimizing COM movement, further improvements in stability during adaptation might be obtained through factors not accounted for in our simulations, e.g., anticipatory postural adjustments, reduced delays, multisegment movement, or feedforward control enabled by prior knowledge of the perturbation direction. In contrast to the early response, we observed large intersubject variability during the later response, where voluntary components might contribute to the movement. In agreement with our previous simulation study (9), prioritizing minimization of COM excursion (maximizing stability) over effort led to increased reliance on the hip strategy. Here we used a model with three instead of two degrees of freedom (addition of knee joint) and accounted for a neural delay to find the same causal relation between task level goal and strategy. The larger potential of the hip strategy to generate balance restoring impulse (12, 14) explains why it is used to minimize COM movement. Accelerating and decelerating the heavy trunk segment during the hip strategy require additional effort, explaining the trade-off between effort and stability. The origin of intersubject differences in this trade-off is difficult to identify, as it might be shaped by factors that are difficult to control such as prior experience with related tasks (34) and emotional state (36, 37).

Our results suggest that the effect of initial posture on movement variability can be reduced by selecting an appropriate control strategy. Variation in initial posture leads to larger variability in the kinematic response in individuals who prioritize stability over effort. Hence, the interaction between variable initial postures and task-level goal explains the larger range of maximal trunk lean angle in individuals using hip strategies (Fig. 1*B*). This interaction between initial posture and control strategy might have implications beyond reactive balance control, as it has previously been suggested that elite gymnasts select a control strategy that minimizes the effect of uncertainty in initial posture on performance (73).

Our simulations based on simple models explained important aspects of variability in reactive standing balance kinematics but provide limited insight in how control of standing balance is realized by the complex neuromusculoskeletal system. The use of more complex models is unlikely to alter our main conclusions but could advance our insight in the underlying mechanisms. First, we did not model individual muscle contributions to joint torques. As a result, our model does not provide insight in how muscle mechanics contributed to the response. Muscle short-range stiffness has an important contribution to the joint torques during the initial mechanical response to support surface perturbations (74). This contribution is likely influenced by alterations in initial posture since short-range stiffness depends on muscle activity, and muscle activity will be different for different standing postures. Furthermore, alterations in resting state of the muscle might alter reflexes (43). Second, not only musculoskeletal mechanics but also motor control is simplified in our model. We assumed optimal feedback control, and hence, we computed feedback gains by optimizing a cost function representing the task-level goal. While this approach captures important aspects of movement, it does not capture the underlying processes within the central nervous system. In addition, we made assumptions about the feedback structure. Feedback from center of mass kinematics has been shown to explain observed joint torques and muscle activity (19, 22). Similarly, deviations in joint positions following a support surface perturbation lie largely in a joint subspace in which center of mass position is constant (75). Third, we did not model sensorimotor noise (76), which has been shown to contribute to motor variability (2). Sensorimotor noise might have contributed to the variability in initial posture, which we accounted for, but likely also shapes the kinematic strategy (38).

Improved insight in factors that cause intrasubject movement variability will advance our ability to study the origin of differences in postural strategies between groups and conditions. Movement trials are typically averaged when comparing conditions and groups and important information is lost in this process. When studying alterations in reactive balance due to aging or pathology, it might be important to dissociate contributions from alterations in initial posture and control. Our results suggest that controlling initial posture in experiments or accounting for variability in initial posture when processing experimental data might facilitate assessment of differences in reactive balance control between groups. For example, accounting for the contribution of increased variability in initial posture to the higher incidence of hip and stepping strategies in older adults will allow us to more accurately identify the contribution of strength deficits and sensory deterioration to altered balance control in older adults.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

T.V.W., L.H.T., and F.D.G. conceived and designed research; T.V.W. performed experiments; T.V.W. analyzed data; T.V.W. and F.D.G. interpreted results of experiments; T.V.W. prepared figures; T.V.W. drafted manuscript; T.V.W., L.H.T., and F.D.G. edited and revised manuscript; T.V.W., L.H.T., and F.D.G. approved final version of manuscript.

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