Balance control impairment in obese individuals is caused by larger balance motor commands variability

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ABSTRACT

It is acknowledged that various factors impaired balance control. Among them, heavy body weight is associated with poor balance control because the location of the center of mass is further away from the ankle joint. Thus, a larger active ankle torque is required to counter the greater gravitational torque. Because balance motor commands have signal-dependent noise whose standard deviation increases with the absolute value of the neural control signal, it was hypothesized that faster center of pressure speed observed in obese individuals would be related to larger balance motor commands variability. A feedback-control model and parametric system identification technique was used to estimate the variability in the balance motor commands and neural controller parameters based on previously published experimental data. Results of the neuromechanical model confirmed that the balance motor commands of obese individuals are more variable than that of lean individuals.

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1. Introduction

To control upright balance, the brain needs to correct small deviations from an upright body position to reduce the consequence of the gravity-induced torque that tends to accelerate the body further away from the upright position. To counter the destabilizing torque, a corrective torque is exerted by the feet against the support surface [1]. Although this process of corrective torque generation is not completely understood, there is evidence suggesting that visual, vestibular and proprioceptive (e.g., plantar sole mechanoreceptors and ankle proprioception) systems contribute to postural control. For instance, various studies have demonstrated that either stimulation, or reduction of cue, to visual [2–4], vestibular [5–7], plantar sole mechanoreceptor [8] or proprioceptive [9,10] systems evoke body sway. These results imply that feedback-control mechanisms contribute to corrective torque generation based on motion detection by the sensory systems.

During the last decades, various studies have identified factors, such as bodyweight, that impaired balance control [11]. For instance, Hue et al. [12] have shown that the speed of body sway of obese (i.e., body mass index (BMI) > 30 kg m^{-2} and <35 kg m^{-2}) and morbid obese (i.e., BMI > 35 kg m^{-2}) individuals is faster than that of normal weight individuals (i.e., BMI < 25 kg m^{-2}). Improvement in balance control following weight loss provides further support for the role of weight and rule out the suggestion that balance impairment, in these studies, resulted from lower limb peripheral neuropathy [13].

Based on these observations, it has been hypothesized that because the location of the center of mass is further away from the ankle joint (associated with an increased abdominal mass), compared to normal weight individuals, obese and morbid obese individuals would need to counter a greater gravitational torque, and consequently a larger corrective ankle torque is necessary. The outcomes of a mathematical model confirmed that to maintain their balance, individuals with BMI larger than 30 kg m^{-2} needs to generate larger active ankle torque [14]. An inherent feature of the human motor system is that the standard deviation of motor-neuronal firing increases with the mean level of the signal [15]. Given the fact that the variability in the motor command increases with an increased magnitude of the motor command, it is likely that faster center of pressure speed observed in previous studies also reflected greater variability in the balance motor commands (not measured in the cited studies).

Therefore, the purpose of this study was to determine whether faster center of pressure speed measured in obese and morbid

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obese individuals could be related to greater variability in the balance motor commands. To pursue this goal, a feedback-control model [16] and parametric system identification techniques were used to determine the variability in the balance motor commands and model parameters based on experimental data. This modeling approach allowed the assessment of the neural controller parameters and the variability in the balance motor commands while predicting center of pressure speed during upright balance control in lean and obese individuals.

2. Methods

The body dynamics and kinematics during upright balance control were described using an inverted pendulum model (Fig. 1). The neuromechanical model is based on the assumptions that the inverted pendulum rotates around the ankle joint therefore inputs and outputs are restricted to the sagittal plane and the active ankle torque is controlled by a linear neural controller (i.e., PID controller). It is assumed that the neural control system detects a deviation of the body away from the upright reference position (i.e., earth-vertical) and following sensorimotor transformation, a motor command generates a corrective torque reducing the deviation of the body position with respect to the reference position (i.e., error signal). Consequently, the components of the neural controller are scaled according to the angular position error signal (K_P), the derivative of this error signal (K_D), and the integral of this error (K_I). Although these three components are efficient to stabilize an inverted pendulum, it is acknowledged that the corrective torque could contain two components: one proportional to the angular position error (K_P) and the other proportional to angular velocity error (K_D). These two components refer to ankle joint stiffness and damping [1,17]. Although it is not necessary for stability, the integral component (K_I) contributes in adding low frequency error correction allowing for better fits to human data [18]. The input to the body model is the torque exerted at the ankle joint and the output is the center of pressure (COP) and center of mass (COM) kinematics. The torque at the ankle joint includes a random disturbance generating body sway patterns (i.e., band-limited white noise) similar to those measured experimentally and it includes the balance motor commands generated by the brain in

response to body motions. To simulate body sway of normal weight, obese and morbid obese individuals, three different body model parameter sets were calculated according to participant's characteristics (weight: 71.1 ± 7.9 kg, 101.5 ± 14 kg and 153.3 ± 23.7 kg; height: 177 ± 5.6 cm, 175 ± 6.8 cm and 174 ± 6.1 cm; age: 38.6 ± 9.4 years, 37.9 ± 7.7 years and 44.4 ± 8.9 years, for control ($n = 16$), obese ($n = 14$) and morbid obese ($n = 14$) groups respectively). These data were taken from a previously published manuscript investigating the effect of weight loss on balance control in obese and morbidly obese Caucasian men [13]. Participants stood barefoot on a force platform with feet together for 35 s (14 trials). Although participants performed 7 trials with their eyes closed (at 5 s a computer-generated tone indicated to close their eyes; only the last 30 s served for computing the COP displacement), only the data with eyes open are considered in the modeling approach.

For each group, the average height of the body center of mass above the ankle joint (db = 0.575 × height), body mass (Mb) and body inertia (Jb = Mb × db^2) were used to simulate COP displacement. The sensory feedback time delay from the ankle joint to the brain, determined by sensory-evoked potential method [19], is approximately 0.040 s whereas the time taken by the motor commands to travel from the brain to the ankle joint muscles (i.e., measured from evoked motor response) is approximately 0.030 s [20]. We hypothesized that the time required for performing a sensorimotor transformation was 0.100 s. Therefore, the overall time delay of the feedback loop was set to 0.170 s. A similar time delay has been successfully used to simulate balance control in healthy young adults [16].

Simulations (30-s duration) were performed using Simulink and Matlab (The Mathworks, Natick, MA, USA). The Dormand–Prince algorithm with a fixed step size of 0.002 s was used for all simulations to solve the differential equations associated with the model. For each set of body model parameters, we used the Optimization toolbox (i.e., function fmincon) to determine the gain of the neural controller (i.e., proportional (K_P) and derivative (K_D) components) and the variability level (i.e., gain of the 1st order transfer function: K_N) in the balance motor commands minimizing an error function based on the square of the scalar differences between simulated COP speed and empirical COP speed. We kept
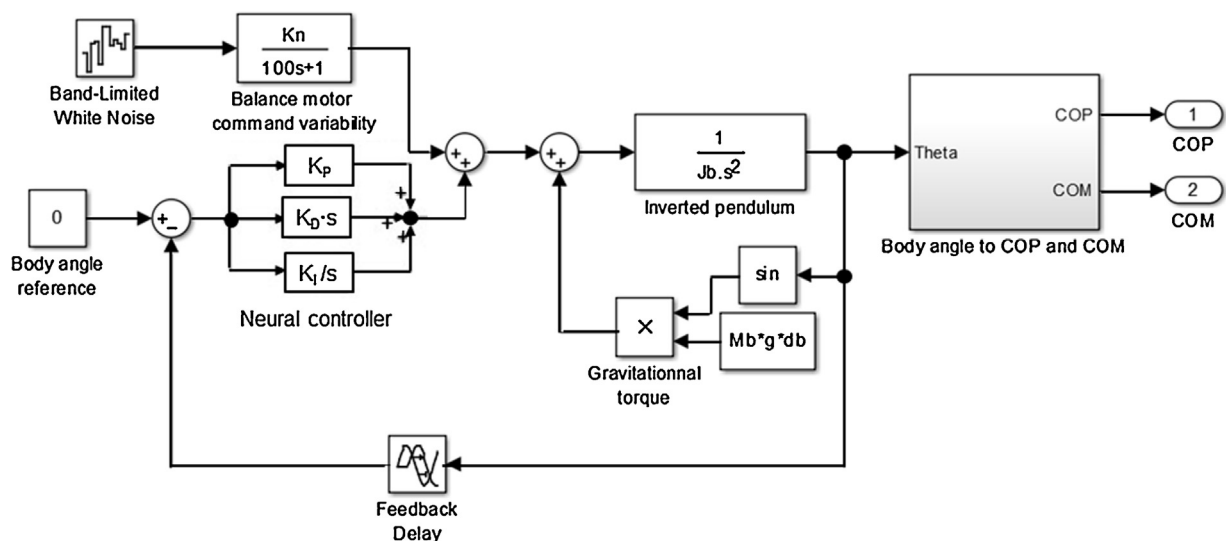

Fig. 1. Feedback control model of balance control. The body is modeled as an inverted pendulum. Variability within the balance motor command is simulated using a band-limited white noise and a first order filter (K_N; variability amplitude). The mechanical plant includes the angular moment of inertia (Jb) and the gravitational torque (db × Mb × g × sin(θ); Mb: mass; g: gravitational acceleration; db: distance between the ankle joint and center of mass). Body sway angle is feedback, following a delay, and compared to earth-fixed vertical (body angle reference). In response to body orientation with respect to body angle reference, the proportional-integrative-derivative (PID) controller generates a corrective torque. The body angle is converted in center of pressure (COP) and center of mass (COM) displacement along the anterior-posterior axis.