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Intrinsic and Extrinsic Contributions to Seated Balance in the Sagittal and Coronal Planes: Implications for Trunk Control After Spinal Cord Injury

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Abstract

The contributions of intrinsic (passive) and extrinsic (active) properties of the human trunk, in terms of the simultaneous actions about the hip and spinal joints, to the control of sagittal and coronal seated balance were examined. Able-bodied (ABD) and spinal-cord-injured (SCI) volunteers sat on a moving platform which underwent small amplitude perturbations in the anterior-posterior (AP) and medial-lateral (ML) directions while changes to trunk orientation were measured. A linear parametric model that related platform movement to trunk angle was fit to the experimental data by identifying model parameters in the time domain. The results showed that spinal cord injury leads to a systematic reduction in the extrinsic characteristics, while most of the intrinsic characteristics were rarely affected. In both SCI and ABD individuals, passive characteristics alone were not enough to maintain seated balance. Passive stiffness in the ML direction was almost 3 times that in the AP direction, making more extrinsic mechanisms necessary for balance in the latter direction. Proportional and derivative terms of the extrinsic model made the largest contribution to the overall output from the active system, implying that a simple proportional plus derivative (PD) controller structure will suffice for restoring seated balance after spinal cord injury.

Keywords

seated balance; functional neuromuscular stimulation (FNS); posture; spinal cord injury

Paralysis due to spinal cord injury results in many health complications such as pressure sores, difficulty breathing, and bowel and bladder dysfunction. Functional neuromuscular stimulation (FNS) of the paralyzed muscles can help alleviate these problems by providing a means to exercise, thus improving blood flow and muscle mass and reducing seating interface pressures.^{1,2} A common effect of spinal cord injury is the inability to maintain a balanced seated posture, which can hinder independent function by compromising reaching and manual wheelchair propulsion. For these reasons trunk stability has been identified as

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one of the highest priorities for motor system recovery in individuals with spinal cord injury.³ Thoroughly understanding the mechanisms for seated balance is important for designing suitable systems to restore it with FNS.

Most previous studies on trunk stability have concentrated on the passive (intrinsic) characteristics of the spine.^{4,5} Many have examined changes in the angle of the trunk with the pelvis held fixed, thus ignoring contributions of the hips to seated postural stability.⁶ In these studies, trunk stability was expressed in terms of a single stiffness measure. Since both passive and active mechanisms can act differently to maintain trunk balance, it is important to isolate their individual contributions, since the target of most FNS interventions is to restore the latter.

Goodworth and Peterka⁷ investigated both active and passive contributions to trunk stability in the coronal plane and assumed that upper body control either followed a similar trend as whole body control, in which sensory integration mechanisms were dominated by long feedback delays, or was dominated by intrinsic mechanisms and/or rapid stretch reflexes characterized by short time delays. That study was the first to derive explicit values for extrinsic and intrinsic parameters that mediate postural balance in the seated human. Another study⁸ has established that tonic and phasic characteristics of intact muscles could also contribute to mechanisms of seated balance in other ways. This particular study found that there was low tonic activity of the abdominal muscles during erect sitting while each muscle produced phasic activity that was linked to the direction of perturbation.

The work reported in this paper contributes to our understanding of the predominant mechanisms of seated balance control in the sagittal and coronal planes by subjecting both able-bodied (ABD) and spinal-cord-injured (SCI) individuals to random perturbations and measuring the resulting responses in terms of trunk orientation angle. These measures combine the effects of trunk movement about the hips and the lumbar joints, which may have significant implications for design of control systems for seated balance. Preliminary studies on simple on-off control of FNS to the hip and lumbar trunk extensors have shown promise of immense benefits.^{9,10} Ideas from the current study will help in designing more advanced interventions that can improve the daily independent functioning of SCI individuals.

The objective of this work was to determine the extent to which the active and passive control mechanisms of seated balance in humans are affected by spinal cord injury. We hypothesized that (1) an inverted pendulum model of human seating characterized by 7 parameters was adequate to capture the essential elements of seated balance both in ABD and SCI individuals; (2) there would be significant differences in all 7 parameters between ABD and SCI individuals, as well as whether the motion of the trunk was in the anterior-posterior (AP) or in the medial-lateral (ML) direction; and (3) intrinsic (passive) torque alone is insufficient to counteract the effects of external perturbations on the trunk.

Methods

Volunteers

Nine ABD and 9 SCI volunteers with age and anthropometric characteristics shown in Table 1 participated in the study. Regarding neurological deficits, 4 of the SCI volunteers had sensory/motor complete (AIS A), 4 had sensory incomplete/motor complete (AIS B), and 1 had motor/sensory incomplete (AIS C). Of the SCI volunteers' injuries, 3 had sustained upper cervical (C6/C7), 5 midthoracic (T6/T7), and 1 lower thoracic (T10). All SCI volunteers were able to attain and keep a fairly erect seated posture. All volunteers signed the consent form approved by the local institutional review board.

Experimental Setup

Volunteers were seated on a Balance Manager (NeuroCom, Inc., Clackamas, OR) modified with a custom-designed seat bolted to the standing surface, as depicted in Figure 1. All volunteers sat on a 3-in (7.6-cm) wheelchair cushion with gel bladder (Drive Medical, Port Washington, NY) to protect the skin from high contact pressures and wore a safety harness to ensure against falls while allowing full range of trunk motion. SCI volunteers were spotted by a physical therapist. The Balance Manager was placed within the work volume of 8 motion capture cameras (Vicon, Oxford Metrics, UK). Retro-reflective markers were placed on the left and right shoulders, the seventh cervical spine, the clavicle, and the left and right posterior–superior iliac spines. The thighs were secured to the seat with a pair of 5-cm wide straps and the shanks were strapped to padded vertical cuffs to maintain the knees close to 90°. Volunteers were instructed to apply enough effort with only their trunk and hip muscles to remain as erect as possible with their arms folded across their chests. All trials where the volunteer's posture strayed too far away from erect were noted and eliminated from the subsequent analysis.

Perturbation Input Signals and Responses

Sagittal plane perturbations were obtained by translating the platform with the volunteer facing the fore-aft direction. Coronal plane perturbations were obtained by the same movement of the platform, but with the chair and volunteer rotated 90°. Low-amplitude sum-of-sines (multisine) perturbation signals were used because of their ability to generate signal power within a specified frequency range and smoother operation,¹¹ in contrast to pseudorandom ternary⁷ or single-pulse perturbations.^{12,13} Additionally, there is strong evidence that a pseudorandom waveform composed of a sum of as few as 3 sinusoids cannot be predicted by human subjects.¹⁴ The signals were defined as:

$$x(t) = \sum_{k=1}^N 2A_k \cos(2\pi f_k t + \phi_k), \quad (1)$$

where N is the number of components, A_k the amplitude, f_k ($f_{\min}$ f_k $f_{\max}$) the frequency, and ϕ_k the phase.¹¹ f_k was defined as $f_k = f_0 \cdot 1_k$, with 1_k a positive integer (1_1 1_k 1_N). [$f_{\min}$ $f_{\max}$] defined the frequency band within which the amplitude spectrum of $x(t)$ would be

constant. f_0 is the fundamental frequency. The discrete frequencies f_k were defined by the integers l_k , chosen as odd integers such that:

$$f_{\min}=l_1 \cdot f_0; f_{\max}=l_N \cdot f_0. \quad (2)$$

The harmonic spacing between any two integers, l_i and l_{i+1} , was chosen as 2 for this study. The phase was computed using Schroder's formula:¹¹

$$\phi_k = -\frac{\pi k(k-1)}{N}. \quad (3)$$

We chose $f_{\min} = 0.1$ Hz, $f_{\max} = 6.5$ Hz, and $f_0 = 0.01$ Hz. Figure 2 shows the displacement, velocity, and acceleration of a typical cycle, along with the envelope of power/frequency for the velocity signal. The same signal was used for perturbations in the AP and ML directions.

The maximum translational amplitude of the perturbation signals was set at 2.82 cm, which was the maximum that could be tolerated by most of the SCI volunteers without veering too far from their erect postures. This choice dictated a constant value of $A_k = 0.25$. The maximum platform velocity was limited by the drive motors to 20 cm/s.

The response signal was the change in trunk angle computed from marker positions on the trunk.

Experimental Protocol and Output Responses

For ABD volunteers, 5 trials were captured, each consisting of the application of 5 cycles in 100 seconds. Because of postural challenges, as well as to avoid prolonged sitting in one position and minimize risk of pressure sore formation, the trials for SCI volunteers were 60 seconds in duration, consisting of 3 cycles per trial. Adequate rest of 5–10 minutes was allowed between trials. For 3 of the SCI volunteers, only 7–8 cycles could be collected because they were either unable to withstand the perturbations in all trials or they moved their upper extremities to maintain balance. For each ABD and SCI volunteer, 5 and 3 cycles, respectively, were randomly selected to perform forward simulations to validate the model identified with the remaining trials.

Trunk Parametric Model

A linear inverted pendulum model of the trunk (shaded box in Figure 3) was used. With thighs assumed to be fixed with respect to the chair, the hip and lumbar joints were lumped into a single joint about which a solid trunk segment rotated in the sagittal and coronal planes. The system was assumed under influence of combined intrinsic passive control mediated by passive structures such as ligaments, tendons, and inactivated muscle tissue. Active neural control was mediated by spinal circuits and active contractions of the muscles of the hips and trunk. Passive control was characterized by 3 parameters: stiffness (K), damping (B), and short latency time delay (τ_{sl}). The path containing B and τ_{sl} modeled a short-latency stretch-reflex pathway.⁷ Active control was assumed to be a proportional-

integral-derivative (PID) control, characterized by 4 parameters: gains K_p , K_d , and K_i respectively, and long-latency time delay (τ_{l1}). Each of the 2 types of control elements contributed a torque to maintain balance as a function of the error between the desired and actual output signals (passive controller output $[T_p]$ and active controller output $[T_a]$). T_d was the translational disturbance of the platform. The resultant torque on the system, T_R , was the sum of the 3 input torques.

Using the transfer function between the change in trunk angle (θ_B) and platform translation (X) in Figure 3, the linear model of the trunk in the frequency domain was written as:^{7,15}

$$\frac{\theta_B(s)}{X(s)} = \frac{-mhs^3}{[Js^3 + e^{-\tau_{l1}s}Bs^2 + (K - mgh) \cdot s + M(s) \cdot e^{-\tau_{l1}s}]}, \quad (4)$$

where θ_B = change in trunk angle, m = mass of the trunk, h = height of the center of mass from the support platform, J = moment of inertia about the combined hip/lumbar joint, g = acceleration due to gravity, $X(s)$ is the Laplace transform of the platform input disturbance, and s is the Laplace transform parameter. The quantity $M(s)$ in Equation 4 is defined as:

$$M(s) = k_d s^2 + K_p s + K_i. \quad (5)$$

The anthropometric parameters J , m , and h were derived for each volunteer based on body mass and height tables.^{16,17} The quantities were determined separately for the AP and ML directions, as the hip joint was the dominant joint for movements in the AP direction, while in the ML direction the dominant joint was assumed to be located between the first sacral and the fifth lumbar vertebrae.¹⁸ The mean and standard deviations of the main anthropometric parameters are given in Table 1.

Data Processing

One approach for identifying the parameters of the model in Figure 3 involves fitting the model in the frequency domain.⁷ In that method, magnitude and phase of the experimental data are computed and fit to equivalent quantities of the linear model (Equation 4). The approach requires extensive preprocessing of the experimental data and smoothing of the frequency-domain signal functions.⁷ To avoid these steps, we identified the parameters of the model by direct simulation in the time domain using the Parameter Estimation Toolbox of Simulink (The Mathworks, Inc., Natick, MA). In this approach a Simulink model of Equation 4 was built with the parameters serving as unknown variables. The input velocity signal of the platform was differentiated using central differences to obtain acceleration which was input to the Simulink model. The model and actual outputs (trunk angle trajectories) were compared and the error was used to adjust the model parameters by minimizing the sum of squares of the difference between the 2 trajectories. The optimization was terminated when 1 of 4 criteria was satisfied: (1) successive parameter values changed by less than $1.0e-6$, (2) the number of function evaluations exceeded $1e+3$, (3) the number of iterations exceeded $1e+2$, or (4) successive function values were less than $1.0e-6$. Most of the simulations were stopped due to satisfaction of either condition 1 or condition 4. The

resulting parameter sets for all ABD and for all SCI volunteers were averaged to determine mean and standard deviations for the 2 populations.

Results

The simulated trunk angle from the inverted pendulum model closely tracked the experimental trunk angle, in this case, with a root-mean-square error (RMSE) of 0.3° (Figure 4). The main areas of divergence between the 2 trajectories were the initial part (around 0–1 s) and most of the peaks. The mean ABD RMSE values were 0.2° and 0.4° in the AP and ML directions, respectively, and 0.3° in both AP and ML directions for SCI volunteers. The RMSE between the simulated (model) and experimental angles for all trials were $0.68 \pm 0.13^\circ$ and $0.49 \pm 0.09^\circ$ for ABD and SCI subjects, respectively, in the AP direction, and $0.57 \pm 0.22^\circ$ and $0.72 \pm 0.34^\circ$ in the ML direction.

All the 7 control parameters (K , B , K_p , K_d , K_i , τ_{sl} , and τ_{11}) were significantly different ($P = .0$) between the groups except for B , K_d , and K_i for movements in the ML direction (Figure 5). In the ML direction, B was not affected by spinal cord injury ($P = .7$, $CI = -1.8, 2.7$). Similarly, both K_d and K_i were not affected by spinal cord injury ($P = .1$ and 0.9 and $CI = -9.3, 1.1$ and $-1.8, 2.1$, respectively). Values of all parameters in the AP direction were significantly affected by spinal cord injury ($P \approx .0$). The long-latency time in the AP direction was much prolonged after spinal cord injury, but had a smaller increase in the ML direction. The short-latency time, on the other hand, increased slightly from about 12 milliseconds to 16 milliseconds in the AP direction, while it actually decreased from 17 milliseconds to 12 milliseconds in the ML direction as a consequence of spinal cord injury. Values of the intrinsic parameter K in the AP direction were 2.1 ± 0.7 and 2.8 ± 0.4 (N·m/rad)/kg·m for ABD and SCI volunteers, respectively, and the corresponding values in the ML direction were 6.3 ± 1.0 and 5.1 ± 0.6 . Values of B in the AP direction were 1.5 ± 0.1 and 0.5 ± 0.2 (N·m/rad)/kg·m for ABD and SCI volunteers, respectively, and the corresponding values in the ML direction were 1.3 ± 0.2 and 1.2 ± 0.3 (N·m·s/rad)/kg·m. The extrinsic parameters K_p , K_d , and K_i in the AP direction were 14.0 ± 1.0 (N·m/rad)/kg·m, 2.0 ± 0.4 (N·m·s/rad)/kg·m, and 0.7 ± 0.2 (N·m/rad·s)/kg·m for ABD volunteers, and 5.8 ± 1.6 , 0.9 ± 0.5 , and 0.4 ± 0.2 for SCI volunteers; while in the ML direction they were 11.4 ± 1.8 , 1.8 ± 0.7 , and 0.9 ± 0.2 for ABD volunteers and 7.3 ± 2.4 , 2.4 ± 0.6 , and 0.9 ± 0.3 for SCI volunteers. The values for τ_{11} were 124.6 ± 26.1 and 197.1 ± 23.1 in the AP and ML directions, respectively, for ABD volunteers, and 233.4 ± 20.1 and 274.1 ± 22 in the AP and ML directions, respectively, for SCI volunteers; whereas τ_{sl} values were 11.6 ± 3 and 16.7 ± 2 in the AP and ML directions, respectively, for ABD volunteers and 15.8 ± 2.5 and 12 ± 2.7 in the AP and ML directions, respectively, for SCI volunteers.

The proportional and derivative terms tend to dominate the extrinsic parameter contributions to the joint torques (Table 2). The relative contributions of the passive and active (PID) elements to the total neural torque output were normalized by the mean of the neural torque after rectifying and averaging each signal. The predominant contributions to the extrinsic active torque resulting from neural drive were made by the derivative and then the proportional components, irrespective of injury status and direction of perturbation. There was virtually no contribution from the integral component in all cases.

Discussion

This paper explored the contributions of passive intrinsic and active reflexive properties of the human musculoskeletal system to the control of seated balance. We examined the problem of the rotation of the trunk (combined pelvis and thorax) relative to the support surface in both the AP and ML directions. A parametric model was fit to the experimental data directly in the time domain.

The 2 intrinsic parameters were the stiffness (K) and damping (B). Values for K were relatively low (in the range 2–3 [N·m/rad]/kg·m) in the AP direction for all volunteers and higher (in the range 5–6 [N·m/rad]/kg·m) in the ML direction. The lower values in the AP direction could be attributed to low resistance to trunk forward flexion at the hips represented as ball-and-socket joints, whereas movement of the trunk about the lumbar joints was represented as a flexible beam.¹⁹ The values for K in the AP direction closely matched the values of approximately 2.1 (N·m/rad)/kg·m for SCI individuals and 4.8 (N·m/rad)/kg·m for ABD individuals obtained by Amankwah et al²⁰ in isotonic experiments at the hips, indicating that the largest contributor to the value of K was perhaps due to the passive stiffness at the hip joints. For the larger values in ML direction, the range 5–6 (N·m/rad)/kg·m was close to the value of 7.8 (N·m/rad)/kg·m obtained by Goodworth and Peterka⁷ and 10.7 (N·m/rad)/kg·m by McGill et al⁴ in their studies of trunk stability in the ML direction. There is little or no movement at the hips in the ML direction and hence bending at the lumbar region dominated the values of trunk stiffness.

There is little information on values of intrinsic damping (B) at the trunk and hips. Values obtained in a previous study⁷ were about 1.25 (N·m·s/rad)/kg·m, which could be associated with values in the ML direction in our experiments. Brown and McGill⁶ reported values of around 26.2 (N·m·s/rad)/kg·m in the AP direction and 0.022 (N·m·s/rad)/kg·m in the ML direction. The values we obtained ranged between 0.5–1.5 (N·m·s/rad)/kg·m in both AP and ML directions. The difference in values could be due to the fact that the pelvis was immobilized in those studies. Figure 5 clearly shows that spinal cord injury does not significantly change the intrinsic parameter K in the AP direction, nor K or B in the ML direction. A similar observation was made by Mirbagheri et al²¹ for intrinsic stiffness (K) and damping (B) at the ankle joint after spinal cord injury.

We compared the moment generated by intrinsic stiffness K with static gravity moment in both AP and ML directions, computed using $M_g = mgh \sin \theta_B$, where θ_B is the angle of trunk rotation away from the erect vertical posture, m is the mass of the trunk, and h is the height of its center of mass from the assumed rotation joint. Using nominal values of $m = 42$ kg and $h = 0.27$ m in the AP direction and $m = 33$ kg and $h = 0.18$ m in the ML directions gives $M_g = 111$ N·m/rad and $M_g = 58$ N·m/rad in the AP and ML directions, respectively. The moment due to nominal values of passive stiffness K were about 30 and 45 N·m/rad, respectively, in the 2 directions. Thus the passive stiffness alone cannot be relied upon to generate enough torque to compensate for gravity and acceleration effects, and therefore other mechanisms such as extrinsic stiffness must be brought into play. The low tonic muscle activity reported by Masani et al⁸ however was an indication of the importance of intrinsic muscle activity in the maintenance of erect seated posture.

Since most previously reported estimates for PID controller gains K_p , K_d , and K_i in the AP direction involved experiments with the pelvis held fixed, it was difficult to find comparative values in the literature. Values of the 3 parameters in the ML direction from our experiments were much lower than the approximate values of 18.7 (N·m/rad)/kg·m, 0.8 (N·m·s/rad)/kg·m, and 12.1 (N·m/rad·s)/kg·m reported in Goodworth and Peterka⁷ for experiments with ABD volunteers with eyes open. However, we should emphasize that there were other subtle differences between our approaches to testing. In their experiments the volunteers were in an erect standing posture (hips and knees approximately 0° each), while in our case volunteers sat with hips and knees flexed at about 90° each. The systematic reduction in all extrinsic parameters in the AP direction may all be indicative of the effects of spinal cord injury in reducing active control as a result of damage to the nervous system.

The 2 time-delay parameters examined were the short and long latencies, τ_{sl} and τ_{ll} . Both were larger in the AP direction than in the ML direction. This difference parallels the predominance of the neural controller gains in the AP direction. Values for τ_{ll} and τ_{sl} reported by Goodworth and Peterka⁷ were around 280 milliseconds and 25 milliseconds, respectively. The value for τ_{ll} was comparable to our value for this parameter in the ML direction but not in the AP direction, but our values of τ_{sl} for both directions and injury groups were lower than those reported by Goodworth and Peterka.⁷ The increase in values of τ_{ll} in both AP and ML directions after spinal cord injury may again underscore the observation that spinal cord injury not only reduces the magnitudes of the extrinsic parameters but also results in further delay of their effects, perhaps as a consequence of damage to the central and/or peripheral nervous structures.

One advantage of estimating the parameters in the time domain was the ability to simultaneously obtain the joint torques generated by the model. The intrinsic and extrinsic joint torques from the 2 control mechanisms were of interest. Table 2 illustrates that the proportional and derivative terms tend to dominate the extrinsic parameter contributions to the joint torques. The relatively small torque contributions from the integral part of the active controller may suggest that intact balance control is probably less concerned with steady-state error. Therefore, replicating it with stimulation after spinal cord injury probably should not need anything more than proportional-derivative (PD) action.

From Table 2, the extrinsic torques were 2–3 times larger than the intrinsic torques in the AP direction, and about the same order of magnitude in the ML direction. This underscores the earlier observation that intrinsic torque alone is insufficient to counteract the effects of external perturbations on the trunk. Since the greatest neural effort in the case of spinal cord injury intervention with FNS comes from stimulated muscles, it is imperative that these muscles must generate enough torque to counter disturbances. The most critical values were those required to restore balance in a forward fall (in the AP direction). These torques were generated by stimulating the extensor muscles of the hips and back. In isometric and isotonic experiments with SCI volunteers,²² values of the order of 30 N·m were obtained by stimulating the trunk and hip extensors. Compared with approximately 20 N·m required to maintain seated balance in an erect posture in a dynamic environment, it appears that trunk control using FNS was a promising possibility and warrants further exploration.

Although much insight into the mechanisms for trunk control has been gained from this study, there are still a number of limitations that reduce its generalizability. (1) The parametric model assumed was characterized by a single joint that defined rotation of the trunk about the combined hips and lumbar joints. A more realistic approach would consider the 2 joints individually thus leading to a model with 2 or more degrees of freedom and perhaps a single degree of freedom in the sagittal and coronal planes, respectively. Such a model would be more complicated to analyze, and it is uncertain to result in better insight than the simpler one assumed in this study. (2) Unlike previous works, we made no attempt to separate the neural torque into proprioceptive, visual, and vestibular components.⁷ (3) Values of intrinsic and extrinsic controller gains obtained can be considered adequate for balance studies in an erect seated posture. Many activities of daily living by SCI individuals require seated postures that are far away from erect, such as reaching forward to retrieve/deliver objects. Further gain scheduling studies are required to extend applicability to other clinically useful nonerect postures. (4) Determination of the properties was restricted to the AP and ML planes separately. Real-life activities involve movements in both planes simultaneously. Experimental study of such movements would require equipment that could exert perturbations in multiple directions simultaneously.⁸ (5) The PID model of human reflex control has not been validated, even though several other studies have referred to and used it in a similar manner.^{7,23,24} (6) While the multisine has several advantages as an identification signal, the length of time per trial may be too long to be tolerated by individuals with compromised sitting stability. Other signals such as the pulse signal^{2,13} may lead to shorter trial times. (7) Although the multisine signal was sufficiently random, there was always the possibility that when used in multiple trials the subjects could learn its pattern and prepare to counteract its effect. We tried to mitigate this problem by taking lengthy trials (100 s and 60 s, respectively) for ABD and SCI volunteers. (8) For stronger statistical inference, larger sample sizes than the 9 used for each of the 2 populations may be required. This issue will be examined critically in future studies.

We have quantified the parameters that mediate seated balance in able-bodied individuals and those with spinal cord injury by perturbing them on a moving platform. The results of our study expand our knowledge of the changes in the mechanism of seated balance as a function of injury and direction of perturbation. This knowledge would be invaluable in the development of control systems for maintaining seated balance in individuals with spinal cord injury or other paralyzing diseases.

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Figure 1.

A volunteer with a spinal cord injury sits on the modified Balance Manager platform. The chair mounted on the platform can be rotated to make the volunteer face forward (for sagittal plane experiments) or face to the side (for coronal plane experiments).

Figure 2.

Displacement, velocity, and acceleration patterns of one cycle of a multisine signal used in the experiments. The lower right figure shows the plot of the envelope of the power/frequency of the velocity component of the signal.

Figure 3.

A conceptual model of trunk feedback control. Rotation of the trunk and pelvis about the hips and lumbar joints are combined into a single trunk angle which is used as the feedback signal for the extrinsic neural (active) control and the intrinsic (passive) control elements. Seven parameters characterize the control elements: K , B , K_p , K_d , K_i , τ_{11} , and τ_{sl} .

Figure 4. Mean (thick lines) ± 1 SD (thin lines) of experimentally-measured and model-simulated trunk angles in the anterior-posterior direction for all able-bodied (ABD) and spinal-cord-injured (SCI) volunteers over all trials. The plots for movement in the medial-lateral direction have similar variations.

Figure 5.

Mean and SD bar graphs of the parameter estimates obtained from the model described by Equation 1. The darker shading represents the values for able-bodied (ABD) individuals, while the lighter shading represents the values for spinal-cord-injured (SCI) individuals. In each pane, the first group is for anterior-posterior (AP) perturbations while the second group is for medial-lateral (ML) perturbations. Five parameters, K , B , K_p , K_d , and K_i , were normalized with respect to the product of trunk mass and distance of trunk center of mass from the assumed joint of rotation.

Table 1

Mean (SD) of the main anthropometric parameters for ABD and SCI volunteers

		Sagittal Plane				Coronal Plane			
	Age	Body Height, H (cm)	Body Mass (kg)	Trunk Mass, m (kg)	Trunk CoM, h (m)	Trunk Inertia (kg·m ²)	Trunk Mass, m (kg)	Trunk CoM, h (m)	Trunk Inertia (kg·m ²)
ABD	28.4 (10.8)	174.5 (10.3)	72.4 (8.8)	41.6 (5.3)	0.27 (0.01)	4.3 (0.9)	32.1 (4.2)	0.19 (0.03)	2.7 (0.6)
SCI	44.0 (11.3)	174.0 (7.5)	75.3 (16.9)	43.2 (10.1)	0.27 (0.01)	4.5 (1.3)	33.3 (8.0)	0.18 (0.01)	2.8 (0.8)

Abbreviations: ABD = able-bodied; SCI = spinal-cord-injured; CoM = center of mass.

Note. h is the location of the trunk CoM from the assumed rotation joint in the given plane.

Table 2

Relative average contributions of extrinsic and intrinsic torques T_p , T_d , T_i , and T_s , respectively, to the total output from the neural PID controller, T_n , as functions of injury status and perturbation direction

		Proportional	Derivative	Integral	Intrinsic
Able-bodied	Anterior-posterior	31 ± 3	65 ± 2	3 ± 1	27 ± 4
	Medial-lateral	39 ± 4	55 ± 7	5 ± 1	76 ± 3
Spinal cord injured	Anterior-posterior	42 ± 6	57 ± 7	3 ± 2	49 ± 5
	Medial-lateral	33 ± 8	66 ± 6	2 ± 1	95 ± 4

Abbreviations: PID = proportional-integral-derivative.

Note. Extrinsic: proportional K_p , derivative K_d , integral K_i ; intrinsic: K and B .